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Anna L. Waterbrook
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Editors

Sports-related Fractures, Dislocations and Trauma

Advanced On- and Off-field Management

Springer

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Editors
Morteza Khodaee
Department of Family Medicine and Orthopedics
University of Colorado School of Medicine
Denver, CO
USA

Anna L. Waterbrook
Department of Emergency and Sports Medicine
The University of Arizona
Tucson, AZ
USA

Matthew Gammons
Vermont Orthopedic Clinic
Rutland, VT
USA

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I dedicate this book to my son, Arshia, who is on a path to be an incredible young gentleman; my daughter, Kimia, who is a bright star of our lives; and my beautiful wife, Atousa, who has stood by me for over 15 years, as well as to my dad, Ebrahim, who showed me the value of working hard to reach my goals, and in memory of my mom, Parvin, who taught me how to be a caring person. Without their love and support, this would not have been possible.

Morteza Khodaee

I dedicate this book to my family – my husband who has always been my rock and didn't even flinch when I told him I would be working on another book while pregnant with twins and my four children who sustain me and always push me to be the best person I can be.

Anna L. Waterbrook

To my coeditors, Drs. Khodaee and Waterbrook, thank you for your monumental efforts that made this book possible. Thank you to my wife, Julie, and daughters, Kyra and Kelsey, who constantly inspire me to be a better person. Thanks to all those that dedicate their time to covering sports and ensuring health and safety of our athletes.

Matthew Gammons

Preface

This book was first envisioned during the 25th American Medical Society for Sports Medicine (AMSSM) Annual Meeting held in Dallas, Texas, in the spring of 2016. We discussed the need for a comprehensive book in the field of Sports Traumatology. Since there is no book that covers all aspects of sports-related trauma, we wanted to have one book specific to musculoskeletal and other types of trauma encountered in different sporting activities. After contacting Springer and receiving preliminary approval, we started on listing the important topics that needed to be enclosed. With this in mind, we prepared a table of contents that was primarily oriented to the practicing clinicians who care for athletic population on the sideline or in the office.

From the beginning, it was obvious that we had a huge task in front of us. There was no book to model this book after. On the one hand, we wanted to cover all aspects of sports traumatology, and, on the other hand, we wanted to focus on nonsurgical managements. Our focus was to provide as many visual aids (e.g., illustrations and radiographic images) as possible. For this, we would like to specifically thank Mr. Daniel Kolb and Mrs. Amy Brumenschenkel and other staff at Denver Health East Grand Community Clinic in Winter Park, Colorado.

Today, there are unparalleled opportunities to make substantial progress in managing athletes with acute trauma. Although the wealth of information and new, powerful imaging modalities have transformed the management of sports traumatology, there are still deficiencies in evidence-based recommendations. The goal is to manage these athletes in the most efficient ways to allow them to a swift and safe return to sports. This obviously presents new challenges, as there is no consensus in many of these mostly "expert opinion" recommendations. We have been mindful of this and have asked each author to consider the best available evidence in the management of patients in each of their respective fields. Therefore, we would recommend multicenter randomized control trials to evaluate specific managements (e.g., surgical and nonsurgical) for each injury in the future.

From the very beginning, a group of experts in the different areas were put together, all of them enthusiastically agreeing to participate. It is their collaboration that provides the added value to this book. It would have been impossible to complete this book without the invaluable help of Mr. Michael Griffin from Springer.

We believe that we have achieved our goals and that we have come up with a comprehensive and practical text that covers initial and non-operative management of a variety of trauma in Sports Medicine. This book is a unique collection of the topics that have not been previously covered in a single book. Some of the information is difficult to find in other sources. We hope this book will help clinicians manage their athletes with acute trauma, which in the end is our real goal. If so, all the efforts will have been completely worthy.

Denver, CO, USA — Morteza Khodaee
Tucson, AZ, USA — Anna L. Waterbrook
Rutland, VT, USA — Matthew Gammons

Contents

Contributors

Kylie Adamek, MD Department of Internal Medicine, University of Colorado School of Medicine, Denver, CO, USA

Drew Ashby, MD Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

Brenden J. Balcik, MD Department of Emergency Medicine, West Virginia University School of Medicine, Morgantown, WV, USA

Elana Bannerman, MD Department of Family Medicine, Primary Care Sports Medicine, Southern New Hampshire Medical Center, Hudson, NH, USA

Justin H. Bartley, MD Department of Orthopedic Surgery, Lovelace Hospital, Albuquerque, NM, USA

David M. Baxter, DO Department of Sports Medicine, Healthplex Sports Medicine, Springfield, PA, USA

Holly Beach, MD Department of Family and Community Medicine, Primary Care Sports Medicine, University of Arizona, Tucson, AZ, USA

Sagir Bera, DO, MPH, MS, ATC, CSC David Geffen School of Medicine at UCLA, Department of Family Medicine and Sports Medicine, Burbank, CA, USA

Jodi M. Blustin, MD Department of Family Medicine, North Memorial Health and University of Minnesota, Minneapolis, MN, USA

Richard Bortz, MD Winter Park Medical Center, Denver Health, Winter Park, CO, USA

Val Bratinov, MD UPMC Sports Medicine Fellowship Program, Pittsburgh, PA, USA

Jonathan T. Bravman, MD Department of Orthopedics, Division of Sports Medicine and Shoulder Surgery, University of Colorado School of Medicine, Denver, CO, USA

Bradley G. Changstrom, MD Department of Internal Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA

Sonia Chaudhry Department of Pediatric Orthopaedic and Hand Surgery, University of Connecticut School of Medicine, Connecticut Children's Medical Center, Hartford, CT, USA

Eric T. Chen, MD Department of Physical Medicine and Rehabilitation, University of Washington, Seattle, WA, USA

Yvonne Chow, MD, MS Department of Internal Medicine, Division of Sports Medicine, Albany Medical Center, Albany, NY, USA

Stephanie Chu, DO Department of Family Medicine and Orthopedics, Division of Sports Medicine, University of Colorado School of Medicine, Aurora, CO, USA

Jeremy D. Close, MD Department of Family and Community Medicine, Sports Medicine, Thomas Jefferson University, Philadelphia, PA, USA

David Cole, MD Department of Primary Care/Sports Medicine, One Medical, San Francisco, CA, USA

Douglas Comeau, DO, CAQSM, FAAFP Department of Family Medicine, Division of Sports Medicine, Ryan Center for Sports Medicine, Boston University, Boston, MA, USA

Adam M. Cooper, MD Department of Family and Community Medicine, Sports Medicine Section, Thomas Jefferson University Hospital, Philadelphia, PA, USA

Katherine S. Dahab, MD Department of Orthopedics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO, USA

Moira Davenport, MD Department of Emergency Medicine, Allegheny General Hospital Emergency Medicine Residency, Temple University School of Medicine, Allegheny General Hospital, Pittsburgh, PA, USA

William Denq, MD Departments of Emergency Medicine and Sports Medicine, University of Arizona University of Arizona, Tucson, AZ, USA

Rajwinder S. Deu, MD Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Jessica Devitt, BS, MD Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

Omar Dimachkieh, MD Department of Orthopaedic Surgery, UT Physicians, Houston, TX, USA

Sameer Dixit, MD Departments of Orthopaedics, Medicine, and Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Lutherville, MD, USA

Geoffrey M. Dreher, DO Department of Sports Medicine, Crozer-Keystone Health Network, Springfield, PA, USA

Robin H. Dunn, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Alexander Ebinger, MD Department of Emergency Medicine, University of Colorado School of Medicine, Aurora, CO, USA

Katherine M. Edenfield, MD Department of Community Health and Family Medicine, University of Florida College of Medicine, Gainesville, FL, USA

Jeffrey P. Feden, MD, FACEP Department of Emergency Medicine, Alpert Medical School of Brown University, Providence, RI, USA

Jonathan T. Finnoff, DO Department of Physical Medicine and Rehabilitation, Mayo Clinic Sports Medicine Center, Minneapolis, MN, USA

Matthew Gammons, MD Concussion Clinic, Vermont Orthopaedic Clinic, Killington Medical Clinic, Rutland Regional Medical Center, Rutland, VT, USA

Jordan S. Gold, MD Department of Radiology, MedStar Washington Hospital Center, Washington, DC, USA

Andrew T. Gomez, MD Swedish Providence Medical Group - Mill Creek Sports Medicine, Everett, WA, USA

Jocelyn R. Gravlee, MD Department of Community Health and Family Medicine, University of Florida College of Medicine, Gainesville, FL, USA

Andrew John Maxwell Gregory, MD Department of Orthopedics, Vanderbilt University Medical Center, Nashville, TN, USA

Rebecca Griffith, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Alicia Gustafson, DO Northwest Allied Physicians, Tucson, AZ, USA

Greg Gutierrez, MD Department of Family Medicine, University of Colorado School of Medicine, Denver Health, Winter Park Medical Center, Winter Park, CO, USA

Christopher Guyer, MD, FACEP Divisions of Emergency Medicine and of Sports Medicine, Henry Ford Health System, Detroit, MI, USA

Utsav Hanspal, MD, MPH Department of Orthopedics and Sports Medicine, Lourdes Hospital, Binghamton, NY, USA

Justin E. Hellwinkel, MD, MS Department of Orthopedic Surgery, New York Presbyterian/Columbia University Medical Center, New York, NY, USA

John C. Hill, DO Department of Family Medicine and Orthopedics, CU Sports Medicine, University of Colorado School of Medicine, Denver, CO, USA

Martin D. Hoffman, MD VA Northern California Health Care System, Mather, CA, USA

Department of Physical Medicine and Rehabilitation, University of California Davis, Sacramento, CA, USA

Christopher Hogrefe, MD, FACEP, CAQ-SM Department of Emergency Medicine, University of Iowa Hospitals and Clinics, Iowa City, IA, USA

Rachel Brakke Holman, MD Department of Physical Medicine and Rehabilitation, University of Colorado School of Medicine, Aurora, CO, USA

Stephen Huang, MD Department of Orthopaedic Surgery, John Peter Smith Hospital Sports Medicine, Fort Worth, TX, USA

Kenneth J. Hunt, MD Department of Orthopedics, Division of Foot and Ankle, University of Colorado School of Medicine, Aurora, CO, USA

Kyros Ipaktchi, MD, FACS Department of Orthopedic Surgery, Denver Health Medical Center, Denver, CO, USA

Bjorn C. Irion, MD Western Orthopedics & Sports Medicine, Grand Junction, CO, USA

Nathaniel S. Jones, MD, CAQ-SM Department of Orthopaedic Surgery & Rehabilitation, Health Sciences Campus, Loyola University Medical Center, Maywood, IL, USA

Christopher D. Joyce, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Morteza Khodaee, MD, MPH, FACSM, FAMSSM, FAAFP Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA

Robert Kiningham, MD Department of Family Medicine, University of Michigan, Ann Arbor, MI, USA

Yuka Kobayashi, DO Department of Orthopedic Surgery, Medical College of Wisconsin, Milwaukee, WI, USA

Brian J. Krabak, MD, MBA, FACSM Department of Rehabilitation, Orthopedics, and Sports Medicine, University of Washington and Seattle Children's Sports Medicine, Seattle, WA, USA

Matthew J. Kraeutler, MD Department of Orthopedics, St. Joseph's University Medical Center, Paterson, NJ, USA

Ellen Kroin, MD Department of Orthopaedic Surgery, Loyola University Medical Center, Maywood, IL, USA

Allison D. Lane, MD Department of Emergency Medicine, University of Arizona/Banner University Medical Center, Tucson, AZ, USA

Mark E. Lavallee, MD, CSCS, FACSM York Sports Medicine Fellowship Program, York, PA, USA

Pennsylvania State University College of Medicine, Hershey, PA, USA

USA Weightlifting, Sports Medicine Society, Colorado Springs, CO, USA

International Weightlifting Federation, Budapest, Hungary

York Hospital, York, PA, USA

Lior Laver Head of the Hip Preservation and Sports Hip Service, Sports Medicine Unit, Department of Orthopaedics, Hillel Yaffe Medical Center, Hod-Hasharon, Israel

Hannah P. Leahy, DO Department of Physical Medicine and Rehabilitation, Wellspan Health, Gettysburg, PA, USA

Matthew S. Leiszler, MD University Health Services, University of Notre Dame, Notre Dame, IN, USA

Christine A. Marschilok, MD Department of Family and Community Medicine, Thomas Jefferson University, Philadelphia, PA, USA

Benjamin Marshall, DO Department of Physical Medicine and Rehabilitation, University of Colorado School of Medicine, Aurora, CO, USA

Robert Masocol, MD Department of Family Medicine, Greenville Health System/University of South Carolina School of Medicine, Greenville, SC, USA

Seth A. Mathern, MD Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

Stephanie W. Mayer, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Andrew McBride, MD Department of Family Medicine, University of Colorado School of Medicine, Aurora, CO, USA

Joseph Medellin, MD, MPH, MBA Department of Sports Medicine, Henry Ford Allegiance Health, Jackson, MI, USA

Omer Mei-Dan, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

David Millward, MD, MS Primary Care Sports Medicine, Campus Health Services, The University of Arizona, Tucson, AZ, USA

Aaron J. Monseau, MD Department of Emergency Medicine, West Virginia University School of Medicine, Morgantown, WV, USA

William J. Moreau, DC, DACBSP, FACSM Sports Medicine Division, United States Olympic Committee, Colorado Springs, CO, USA

Rebecca Ann Myers, MD Longs Peak Family and Sports Medicine, Department of Family and Sports Medicine, University of Colorado School of Medicine, Longmont, CO, USA

Dustin Nabhan, DC, DACBSP, CSCS, RMSK Sports Medicine Division, United States Olympic Committee, Colorado Springs, CO, USA

Catherine Mygatt Naden, MD Ryan Center for Sports Medicine, Boston University, Boston, MA, USA

Kyle B. Nagle, MD, MPH Departments of Pediatrics, Orthopedics, and Rehabilitation, Seattle Children's and University of Washington School of Medicine, Seattle, WA, USA

Vicki Nelson, MD, PhD Department of Family Medicine, Greenville Health System, Greenville, SC, USA

Cory A. Newman, PT, DPT, OCS Proactive Physical Therapy, Tuscon, AZ, USA

Britain O'Connor, MD Family Medicine Residency, Rush Copley Medical Center, Aurora, IL, USA

Lauren Oberle, MD Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

Joshua V. Okon, MD Department of Family and Community Medicine, Sports Medicine Section, Thomas Jefferson University, Philadelphia, PA, USA

Andrew Pasternak, MD, MS Silver Sage Sports and Fitness Lab, University of Nevada Reno School of Medicine, Reno, NV, USA

Deepak S. Patel, MD, FAAFP, FACSM Rush Copley Sports Medicine, Rush Medical College, Yorkville, IL, USA

Stephen R. Paul, MD, MA Department of Family and Community Medicine, Campus Health Service, University of Arizona, Tucson, AZ, USA

Sourav K. Poddar, MD Department of Family Medicine and Orthopedics, CU Sports Medicine Center, University of Colorado School of Medicine, Denver, CO, USA

Brent C. Pottenger, MD, MHA Department of Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Ashwin L. Rao, MD Department of Family Medicine, University of Washington, Husky Stadium Sports Medicine Center, Seattle, WA, USA

Jeremiah W. Ray, MD, FACEP, CAQSM Department of Orthopedic Surgery, University of California at Davis Medical Center, Davis, CA, USA

Mark F. Riederer, MD Department of Orthopaedic Surgery, University of Michigan, C.S. Mott Children's Hospital, Ann Arbor, MI, USA

Jaron Santelli, MD Department of Emergency Medicine and Orthopedics, University of Maryland Medical Center, Baltimore, MD, USA

Volker Scheer, MD, MRCGP, MECOSEP Health Science Department, Universidad a Distancia de Madrid (UDIMA), Madrid, Spain

Ultra Sports Science Foundation, Pierre-Bénite, France

Adam Schiff, MD Department of Orthopaedic Surgery, Loyola University Medical Center, Maywood, IL, USA

Daphne Amanda Scott, MD Primary Sports Medicine, Hospital for Special Surgery, New York, NY, USA

Frank Scott, MD Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Adam Seidl, MD Department of Orthopedics, University of Colorado School of Medicine, UCHealth Steadman Hawkins Clinic Denver – Surgery Center, Englewood, CO, USA

Darcy Selenke, MD Department of Family Medicine, University of Colorado School of Medicine, Denver Health, Winter Park Medical Center, Winter Park, CO, USA

Babak Shadgan, MD, MSc Sports Med, PhD Department of Orthopaedics, University of British Columbia, Vancouver, BC, Canada

Medical, Prevention and Anti-Doping Commission, United World Wrestling (UWW), Corsier-sur-Vevey, Switzerland

Kimberly Sikule, MD Department of Family Medicine, University of Vermont, Burlington, VT, USA

Amy M. Singer, DO Department of Family Medicine, Banner UMC, Tucson, AZ, USA

Stephen M. Spadafore, MD Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

Kimberly M. Spahn, MD Department of Orthopaedics, Naval Medical Center San Diego, San Diego, CA, USA

Jack Spittler, MD, MS Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA

J. Herbert Stevenson, MD Department of Family Medicine, Division of Sports Medicine, University of Massachusetts Medical Center, Worcester, MA, USA

Jennifer D. Stromberg, MD Carolina Family Practice and Sports Medicine, Duke Community and Family Medicine, Cary, NC, USA

Emily A. Sweeney, MD Department of Orthopedics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO, USA

Sheila E. Taylor, DO Christiana Care in Wilmington, University of Delaware, Delaware, USA

James Thing, MBBS, BSc, MRCGP, FFSEM Sports and Exercise Medicine, Barts Health, London, UK

Jill N. Tirabassi, MD Department of Family Medicine and Community Health, University of Massachusetts Medical Center, Worcester, MA, USA

Spencer Tomberg, MD Department of Emergency Medicine and Orthopedics, Denver Health Medical Center, Denver, CO, USA

Karin VanBaak, MD Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

Armando F. Vidal, MD The Steadman Clinic, Steadman Philippon Research Institute, Vail, CO, USA

Bryant Walrod, MD Department of Family Medicine, The Ohio State University, Wexner Medical Center, Columbus, OH, USA

Abigail Y. Wang, BS Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Anna L. Waterbrook, MD Department of Emergency and Sports Medicine, The University of Arizona, Tucson, AZ, USA

K. Linnea Welton, MD Department of Orthopedic Surgery, MultiCare Health System, Auburn, WA, USA

Ryan C. Wennell, DO Sports Medicine, Wellspan York Hospital, York, PA, USA

Luke Widstrom, DO Department of Orthopedics, Essentia Health-Duluth Clinic, Duluth, MN, USA

Jessica J. Wingfield, MD Department of Orthopedics, Hand Surgery, University of Colorado School of Medicine, Denver, CO, USA

Michelle Wolcott, MD Department of Orthopedics, CU Sports Medicine, University of Colorado School of Medicine, Denver, CO, USA

Andrew M. Wood, MD Department of Family Medicine, Denver Health Medical Center, Denver, CO, USA

David Ziegler, MD Department of Orthopedics, Denver Health Medical center, Denver, CO, USA

Matthew G. Zmurko, MD Vermont Orthopaedic Clinic, Rutland Regional Medical Center, Rutland, VT, USA

Adam C. Zoga, MD, MBA Musculoskeletal Radiology, Sidney Kimmel Medical College, Thomas Jefferson University, Philadelphia, PA, USA

Part I

General Approach to Sports Trauma

Anna L. Waterbrook

1 Initial Evaluation, Resuscitation, and Acute Management

Anna L. Waterbrook and Moira Davenport

Key Points

- The initial sideline evaluation of an injured athlete involves rapid triage and assessment using primary and secondary survey to assess for life-threatening injuries.
- Sideline management of these injuries focuses on identifying and correcting immediate life threats rather than trying to determine the precise anatomic etiology.
- A high degree of suspicion is needed for many potentially life-threatening injuries: frequent re-evaluations are essential and any decompensation at any time or any clinical concern warrants immediate transport to the emergency department.

One of the primary roles of the sports medicine physician is the management of acute trauma from sports. Whether it is a benign or more serious life-threatening injury, the sports medicine physician needs to be able to rapidly evaluate and triage these athletic-related injuries. As the speed and intensity with which a variety of sports are played increases, there is concern that injury rates and severity will rise proportionally. The vast majority of serious, acute injuries that are seen by sports medicine providers will be due to blunt trauma that is most commonly seen in contact/collision sports such as football, ice hockey, snowboarding, skiing, bungee jumping, and other sports characterized by rapid deceleration and/or high-energy impact. Despite this, there is a paucity of available sports medicine literature on how to manage acute trauma in sports. As outlined in the Team Physician Consensus Statement [1], it is imperative that sports medicine physicians be aware of the many injuries that are associated with blunt trauma and the acute sideline management of these injuries.

Developing a generalized approach to the management of acute trauma in sports, whether evaluating patients on the sideline or in the office is critical. The most important element in the appropriate management of sideline trauma is early recognition and management of injuries. Advanced Trauma Life Support (ATLS) [2] is a course designed to teach physicians how to provide emergency care and stabilization for trauma patients. ATLS principles emphasize identifying and treating the greatest threat to life first and recognizing that lack of a definitive diagnosis should not impede treatment. During the primary survey, life-threatening injuries are identified in a prioritized sequence based upon the effects these injuries will have on a patient's physiology. Initially, it is frequently not possible to identify the specific anatomic injury. The primary survey is based upon the degree of life threat, with the most immediate threat addressed first. The mnemonic ABCDE (airway, breathing, circulation, disability, environment) defines the specific, ordered evaluations and interventions that should be followed for all patients and is based upon how quickly each injury could lead to death, e.g., loss of airway is the most immediate life threat that will lead to death before problems with breathing, circulation, or head injury. This is a very different approach than the performance of a detailed history and physical exam to develop a differential diagnosis before implementing treatment approach that is typically taught during medical training.

When approaching any athlete who is found down after sustaining trauma, it is important to first quickly assess the athlete's vital functions. This can be quickly accomplished by evaluating the athlete's Glasgow Coma Scale (GCS) (Table 1.1), systolic blood pressure, and respiratory rate.

A. L. Waterbrook (✉)
Department of Emergency and Sports Medicine, The University of Arizona, Tucson, AZ, USA
e-mail: awaterbrook@aemrc.arizona.edu

M. Davenport
Department of Emergency Medicine, Allegheny General Hospital Emergency Medicine Residency, Temple University School of Medicine, Allegheny General Hospital, Pittsburgh, PA, USA

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Table 1.1 Glasgow Coma Scale (GCS)

	1	2	3	4	5	6
Eye	Does not open eyes	Eyes open in response to painful stimuli	Eyes open in response to voice	Eyes open spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Inappropriate words	Confused, converses	Oriented, converses normally	N/A
Motor	Makes no movements	Decerebrate response (extensor posturing to painful stimuli)	Decorticate response (flexor posturing to painful stimuli)	Nonpurposeful movement to painful stimuli	Localizes to painful stimuli	Follows commands

A blood pressure cuff is not required to perform a rapid assessment of the patient's blood pressure. As a general rule of thumb, if only the patient's carotid pulse is palpable, then the systolic blood pressure is 60–70 mm Hg; if both the carotid and femoral pulses are palpable, then the systolic blood pressure is 70–80 mm Hg; and if the radial pulse is also palpable, then the systolic blood pressure is >80 mm Hg.

Once the athlete's vital functions have been assessed, then it is appropriate to move on to the primary survey. One quick way to do this is to ask the athlete his/her name, what happened/last play in game. Appropriate responses suggest normal function of vital parameters, e.g., ability to speak clearly (no airway compromise), breathing not severely compromised (air movement to produce speech), and no decreased level of consciousness (can describe what happened).

Fig. 1.1 Jaw thrust, chin lift technique

Airway

The first priority in any trauma patient is airway management to ensure adequate oxygenation and ventilation. It is essential to know the available equipment and how to gain airway access in case of an emergency. The airway should be assessed for patency and any evidence of an airway obstruction due to acute issues such as foreign body or tracheal/laryngeal fracture. In addition, any patient with a GCS of <8 should be assumed to not be protecting the airway. If airway compromise is suspected, there are some techniques that can be used to attempt to open up and maintain the airway. The airway should first be opened with the jaw thrust, chin lift technique (Fig. 1.1), maintaining cervical spine protection when appropriate. This can be effective in relieving an airway obstruction caused by a large tongue, especially in someone who is unconscious or semi-conscious. Bag valve mask ventilation (Fig. 1.2) can be used on the sideline to provide oxygenation and ventilation until more definitive help arrives. If oxygenation or ventilation is difficult, devices such as nasopharyngeal (Fig. 1.3) or oropharyngeal (Fig. 1.4) airways can be used to help open up the airway. Oropharyngeal airways should only be used in unconscious patients as they will likely stimulate the gag reflex in patients who are more awake. Other airway devices,

Fig. 1.2 Bag valve mask ventilation

such as supraglottic airways (e.g., laryngeal mask airway, King Airway) (Figs. 1.5 and 1.6), are easy to use and keep in a sideline medical bag. These devices can often maintain an airway until more help arrives and there is placement of a definitive airway (e.g., endotracheal tube). While the necessity of needing to perform an emergency cricothyrotomy on the sidelines in sports is exceedingly rare, it is a procedure that sideline physicians should be familiar with when all other rescue airway techniques have failed [3].

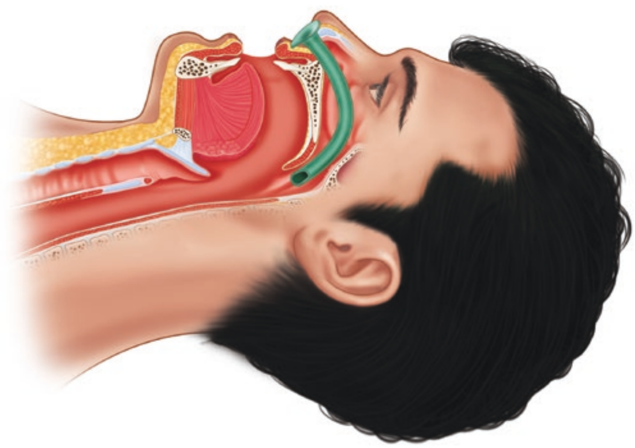


Fig. 1.3 Nasopharyngeal airway

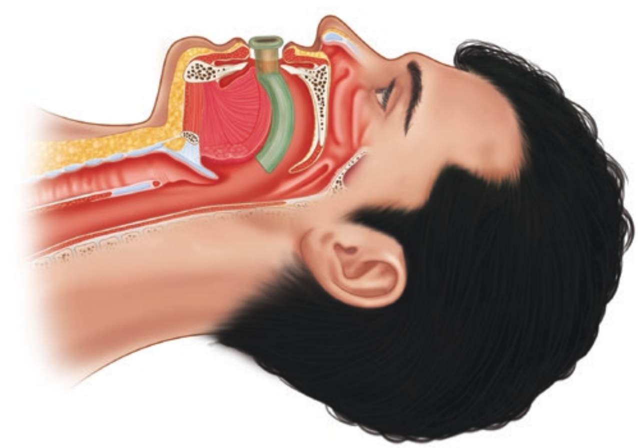


Fig. 1.4 Oropharyngeal airway

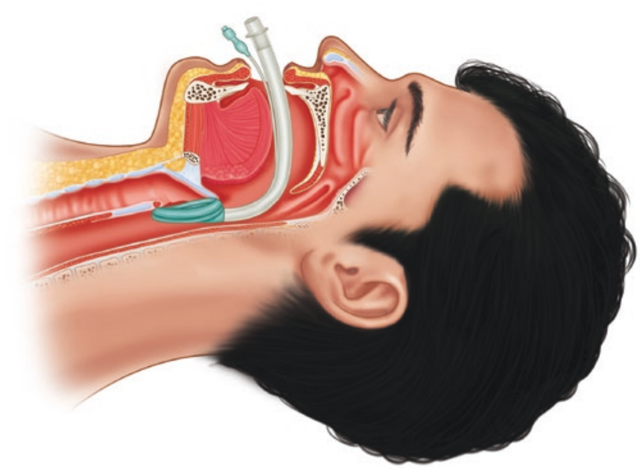


Fig. 1.5 Laryngeal mask airway (LMA)

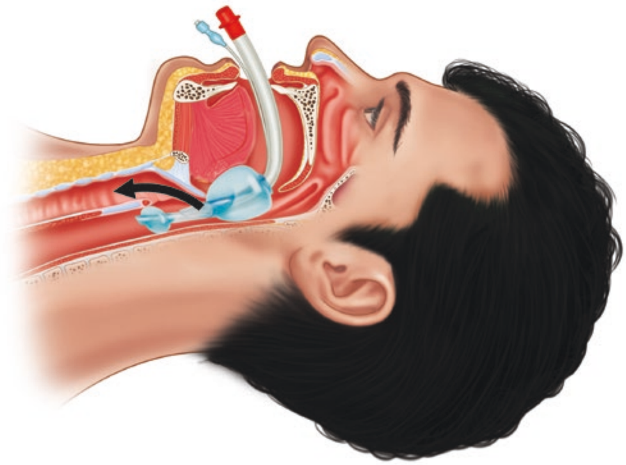


Fig. 1.6 King airway

Breathing

Once the airway is either found to be intact or has been appropriately stabilized, the adequacy of ventilation should be assessed. The evaluation of ventilation includes chest wall excursion, identification of abnormal chest wall segment motion, use of accessory muscles, presence of symmetric breath sounds, and palpation of the chest wall as well as evaluation for jugular venous distention. Injuries which may severely impair ventilation include tension pneumothorax, flail chest (paradoxical chest wall movement) with or without pulmonary contusion, massive hemothorax, and open pneumothorax; these conditions need to be stabilized immediately.

Although rare to see on the sidelines, tension pneumothorax is a life-threatening condition that presents with respiratory distress, absent breath sounds on one side, and/or jugular venous distention. This can be managed swiftly on the sidelines with emergent needle decompression (Fig. 1.7). This technique is easy to perform; the hardest part about performing this procedure is actually making the decision to do it. To perform a needle decompression, use a large gauge angiocatheter (12 or 14 gauge), and place it in the second intercostal space in the midclavicular line. Remove the needle and leave the catheter in place. Successful decompression is signaled by a rush of air and clinical improvement. Performing this procedure could save the athlete's life by allowing adequate oxygenation until a chest tube can be placed. In an unstable patient, tension pneumothorax is a clinical, not a radiographic, diagnosis. Performance of needle decompression of a suspected tension pneumothorax should never be delayed to obtain radiographic confirmation. An open pneumothorax or "sucking chest wound" can also greatly impair an athlete's ventilation. If an open chest wall defect is noted, the area should be covered with a dressing

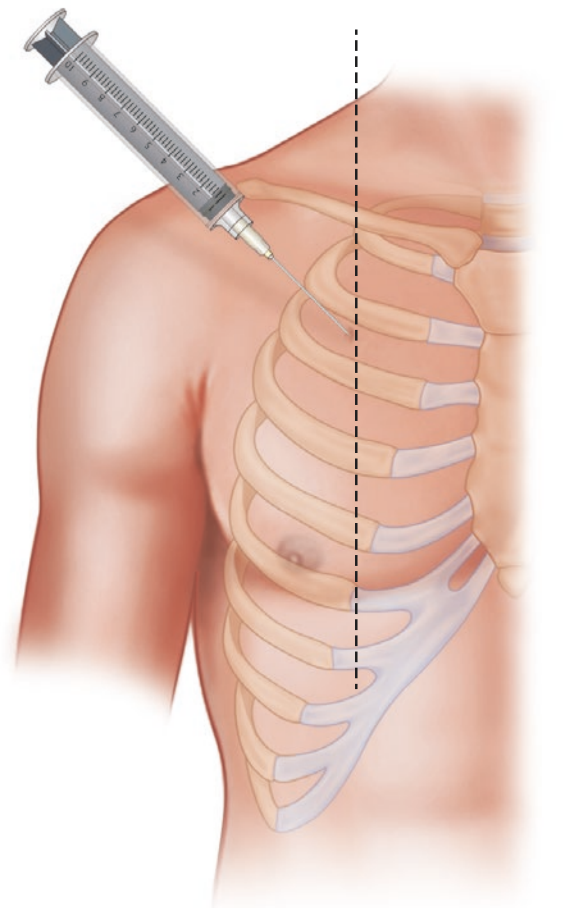


Fig. 1.7 Placement for angiocatheter in emergent needle decompression of pneumothorax

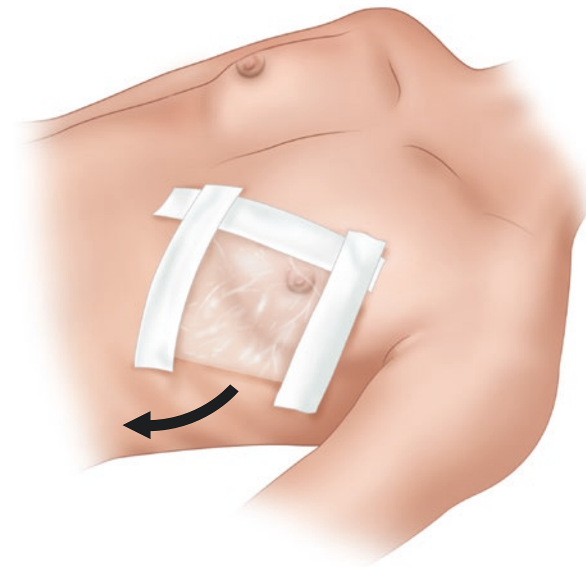


Fig. 1.8 Dressing covering three of four sides for open pneumothorax

that covers three of the four sides of the lesion to minimize breathing difficulty and allowing air to escape but not enter the wound (Fig. 1.8).

Sporting equipment worn in some sports can make assessment and management of airway and breathing more of a challenge. A couple of tricks to assess breathing in an athlete include putting the hand on the chest to look for chest rise or putting a mirror by the nose/mouth of the face down athlete to assess for condensation.

Circulation

Once the airway and breathing have been assessed and stabilized, the athlete's circulatory status should be assessed. After tension pneumothorax, bleeding is the most common cause of hemorrhage and shock and is the predominant cause of preventable deaths after trauma. Sideline assessment of circulation should include evaluation of the athlete's level of consciousness, skin color, and pulse. It is important to keep in mind that athletes may not have the usual level of tachycardia with blood loss due to their baseline bradycardia. The two most important management strategies if hemorrhage is suspected are to control any evidence of external bleeding with direct pressure and place two large bore IVs and begin the administration of fluids.

Disability

The next step in the acute assessment of trauma in sport is to evaluate the athlete for any of evidence of disability. This includes assessment of neurologic compromise due to either intracranial injury or spinal cord injury. Sideline assessment should include level of consciousness, pupillary size, and presence of any lateralizing signs. Cervical spine immobilization and spine-boarding may be necessary but will be covered in a different section of this book.

Environment

Finally, once airway, breathing, circulation, and disability have been assessed and stabilized appropriately, the next and final step in the primary survey is the evaluation of the environment. This refers to both fully exposing the patient to allow for a complete evaluation and to temperature control. On the sideline it may not always be appropriate or feasible to expose the entire patient; however, attention to temperature control is important (e.g., removal of wet clothing and placement of warm blankets or cooling towels). Possible contaminants should be removed as quickly as possible.

Differential Diagnosis

The emphasis on emergent sideline management of trauma should focus on identification and treatment of life-threatening injuries. While the specific diagnosis is not nearly as important as recognizing that a potentially serious injury exists, it is important to keep in mind your differential diagnosis including but not limited to intracranial hemorrhage, soft tissue neck injuries, spinal cord injury, airway obstruction, tension pneumothorax, open pneumothorax, flail chest with pulmonary contusion, massive hemothorax, cardiac tamponade, and severe intra-abdominal and/or external bleeding.

After primary survey and resuscitative efforts are completed and patient has normal vital functions, then the secondary survey begins.

Secondary Survey

Once life-threatening injuries have been identified and stabilized, a more extensive examination should be performed. The entire body should be examined and frequent reassessment of vital signs should be done to ensure that the patient has not had a significant change in overall condition. If the vital signs do worsen, the secondary survey should be stopped to allow adequate resuscitation to occur. It is also important to consider the overall situation when performing a secondary survey. If possible, it is best to get the injured athlete into the training room or an ambulance before performing the comprehensive examination.

The medical history and the history of the actual trauma should be included in the secondary survey. ATLS protocols recommend the use of the AMPLE mnemonic for obtaining the history: allergies, medications, past illness/pregnancy, last meal, and events/environmental concerns related to the traumatic episode [2].

While the whole body is included in the secondary survey, a particular focus should be given to the neurologic exam. A detailed examination can detect subtle injuries. Additionally, the GCS should be recalculated every 3–5 minutes.

Several conditions with the potential to cause hemodynamic instability can be identified during the secondary survey. Isolated rib fractures can lead to a pneumothorax, while fractures of consecutive ribs can significantly alter pulmonary mechanics. The presence of rib tenderness, abnormal chest wall segment motion, or subcutaneous air should be noted. The presence of abdominal bruising (or hematoma) should also be noted. Cullen's sign is a periumbilical hematoma/ecchymosis while Gray-Turner's sign is flank ecchymosis. These findings should raise suspicion for retroperitoneal bleeding and significant abdominal cavity injury.

The rapidly improving quality of portable ultrasound machines has led to the increasing use of these devices by field side physicians. This modality has the potential to significantly improve the diagnostic ability of the sports medicine physician. US can be used to assess for pneumothoraces, intra-abdominal free fluid, fractures, and ligamentous disruptions. Further applications of US will be discussed in the following section.

HEENT

The HEENT exam can reveal several critical findings. Once the airway has been deemed to be stable, the sports medicine physician should carefully search for potential threats to this stability, including loose teeth, lacerations with significant bleeding, and surrounding fractures (maxillary, mandibular, zygomatic arch). The orbits should be gently palpated, visual acuity checked, and extra-ocular motion documented to assess for fractures and associated nerve disruption. If nasal trauma is present, it is important to ensure that a septal hematoma is not seen. Raccoon eyes (periorbital ecchymosis) and/or battle sign (mastoid ecchymosis) (Fig. 1.9) should raise the suspicion for a concomitant skull fracture.

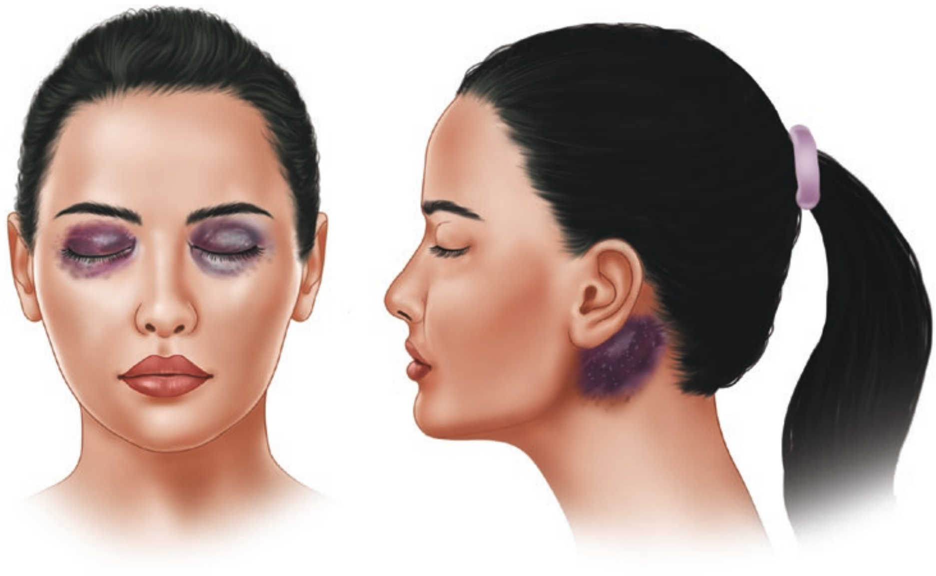
Thorax

Breath sounds should be reassessed as a part of the secondary survey. Palpation of the chest should be performed to evaluate for areas of tenderness, areas of crepitance, and abnormal chest wall motion. Additionally, the portable US can be used to assess for possible pneumothorax; the sliding lung sign can rule out most pneumothoraces. Shallow respirations with tenderness to palpation should raise the clinical suspicion for rib fracture with possible underlying pulmonary contusion. Administration of oxygen is recommended in cases of suspected pneumothorax or if the athlete has shallow breathing due to pain.

Abdomen

A thorough abdominal examination should be performed during the secondary survey. The presence of organomegaly should be assessed, although the presence of organomegaly would more likely be associated with a preexisting condition rather than an acute hematoma. Any localizing tenderness should raise concern for an underlying viscus injury. Furthermore, the finding of concurrent right shoulder pain and abdominal pain, particularly left upper quadrant and slight epigastric pain, should trigger further evaluation of a possible splenic injury. The US could be used to perform the FAST exam (Morrison's pouch, splenorenal window, subxiphoid view, and suprapubic view) to further evaluate for intra-abdominal trauma. The presence of Cullen's sign (periumbilical ecchymosis) (Fig. 1.10) and/or Gray-Turner

Fig. 1.9 Left: Racoon's eyes, Right: Battle sign



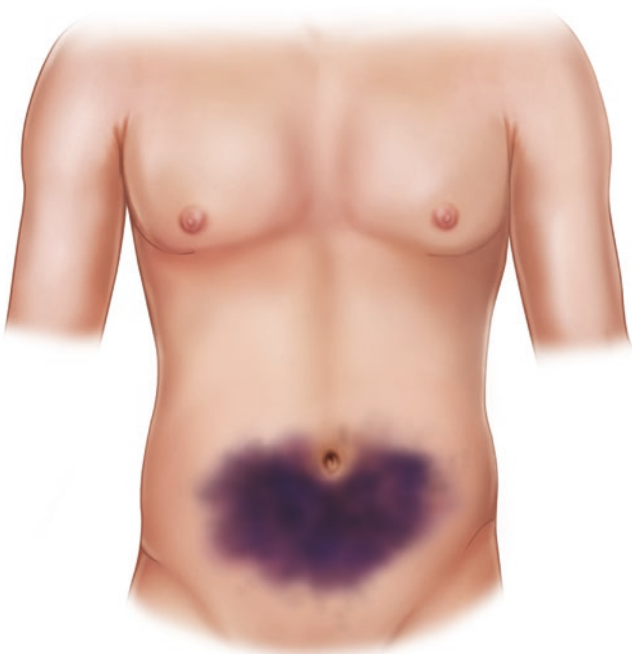


Fig. 1.10 Cullen's sign

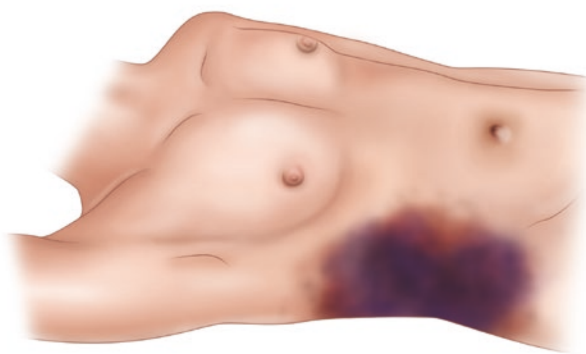


Fig. 1.11 Gray-Turner sign

sign (flank ecchymosis) (Fig. 1.11) should heighten suspicion for a retroperitoneal injury. While the US could be used to assess the structure of the kidneys, this would not allow full assessment of the retroperitoneal area. Any athlete with a suspected abdominal or retroperitoneal injury should have fluid resuscitation started as soon as possible. Continuous hemodynamic monitoring is also recommended for these patients.

Pelvis

The pelvis should be palpated to detect any obvious deformities and to assess tenderness to palpation. Opinions vary as to whether the stability of the pelvis should be assessed in the field. Proponents argue that early identification of instability (and thus likely fracture) better directs the resuscitation, while opponents argue that the assessment may dislodge blood clots at the site of a fracture and hasten clinical deterioration. The current consensus is that a pelvic binder should be applied in any case of suspected pelvic fracture but that "rocking the pelvis" should be avoided.

Spine

If possible, the entire spine should be palpated to assess for any tenderness, step-offs, or other deformities. This should be done while maintaining cervical spine stabilization.

Musculoskeletal

The presence of a fracture, dislocation, or significant ligamentous injury should be identified as soon as possible in the secondary survey. A thorough neurovascular exam is paramount to the assessment of the injured extremity. Any obvious long bone fracture or dislocation with neurovascular compromise should quickly be splinted in a position that best re-establishes normal anatomy. This may require administration of analgesics. Any open wounds should be gently debrided as quickly as possible to limit contamination. Hip and knee dislocations are considered true orthopedic emergencies due to the significant risk of vascular compromise (medial femoral circumflex and inferior gluteal arteries to the femoral neck and the popliteal artery with knee dislocations). Given the large muscle mass about the hip, it may not be possible to reduce a hip dislocation on the field. However, a high index of suspicion is needed to detect a knee dislocation. The knee capsule is typically disrupted during a dislocation; therefore, an effusion is not typically seen. Patients with a suspected knee dislocation should have the knee splinted in 15–20° of flexion for transport. Sideline US may be used to detect dislocations, bone fractures, and disruption of some of the more superficial soft tissue structures (i.e., ATFL, Achilles tendon, patellar tendon). Lastly, it is important to continuously monitor musculoskeletally injured patients for a change in neurovascular status and the potential development of compartment syndrome.

Pain Management

Sideline pain management is one of the more controversial topics in sports medicine. The American College of Sports Medicine's 2013 Team Physician Consensus Statement states that a team physician faces ethical challenges regarding "the use of local or systemic pain medications to allow participation" [1]; however, the Consensus Statement does not specify which medications the sideline physician should or should not carry. Most state high school athletic associations have set guidelines for medications that should be readily available on the sideline. The emergency physician covering sporting events should check with the respective state high school athletic associations and sports governing bodies for further guidance.

If the decision is made to provide analgesics, the overall status of the patient should be considered before any medication is administered. Sideline or responding EMS units may have more advanced pain medications available; these can be used following a discussion among the field side physician, the EMS crew and possibly the crew's medical command physician. If the athlete is a minor, the parent or guardian should be involved in the decision to administer analgesics, particularly narcotic agents. Any patient receiving narcotic pain medications should have continuous cardiovascular monitoring prior to receiving the medication. Lastly, patients with cervical spine precautions or altered mental status should not be given oral agents.

Referral to the ED

The decision to transfer a patient to the ED is multifactorial. Any patient that has had a loss of consciousness may need to be immediately transported. Transportation to a medical facility should be strongly considered in any pediatric patient-athlete requiring fluid resuscitation as well as should any athlete with suspected heat-related injury [4]. Hemodynamic stability, extent of injury, services available at the event site, subspecialty capabilities at local hospitals, and athlete/family concerns should all be considered when making the decision to transport the injured athlete.

The level of competition and the venue may allow imaging studies to be performed onsite; most professional sporting facilities have musculoskeletal ultrasound and standard radiographic capabilities. Additionally, many of these facilities have point-of-care blood testing available, allowing for immediate assessment of electrolyte status as well as hemoglobin/hematocrit.

The need for X-rays can be determined by using clinically validated decision-making tools. The Ottawa Foot/Ankle and Knee Rules have been extensively validated and have been shown to be applicable to adult sports-related trauma. The need for emergent elbow X-rays has been assessed via the elbow extension test. If a supine patient is not able to fully extend the elbow (a positive test), X-rays are recommended. This has been found to have 96–98% sensitivity for detecting a fracture [5, 6]. To date, no clinical decision tools have been validated for the wrist or the shoulder [6].

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Sports Coverage for Traveling Teams

2

William J. Moreau and Dustin Nabhan

Key Points

- Sports medicine teams should be prepared for travel-related health risks for athletes and staff.
- Sports medicine teams encounter many challenges due to international, national, and state laws and regulations in terms of licensure, scope of practice, and the ability to carry medicine and equipment.
- Appropriate documentation and later analysis of outcomes is crucial for ongoing audit of travel medicine program efficacy and team satisfaction with medical service provision.

Introduction

Traveling with athletic teams is like traveling with a large family; you never know what will happen, but it is best to prepare for anything and everything. Much like the practice of sports medicine, the more experience one has with traveling teams, the better prepared they will be to meet the next medical challenge. Using a broad selection of medical specialties and recognizing the benefits of having a diverse staff (i.e., age, gender, and cultural background) within sports medicine staffing plans will provide the best opportunity to meet every challenge encountered. Each member of the sports medicine team should be encouraged to use their expertise and past experiences to provide input on the medical plan.

Safety in athletics can be attained only by appropriate planning for and supervision of practice, competition and travel [1]. This process is time intensive. For example, the medical staff planning for Olympic and Paralympic Games typically starts a minimum of 4 years in advance of the competition. Reviewing experiences and data from prior events is a useful planning tool that can save time and money and help the sports medicine team avoid unexpected challenges.

W. J. Moreau · D. Nabhan (✉)
Sports Medicine Division, United States Olympic Committee, Colorado Springs, CO, USA
e-mail: Dustin.Nabhan@usoc.org

Planning

Each competition location presents its own challenges and requires a situationally accurate medical plan. An organized approach to preparation improves outcomes by reducing error [2]. In addition to the staffing and medical equipment that you travel with, consider the following:

- Pre-travel health status of the delegation
- Legal issues
- Travel formulary planning
- Local medical assets
- Emergency plan for the venue
- Management of blood-borne pathogens and infectious disease
- Language barriers
- Rules for importing medications and medical equipment
- Relationships with host medical staff
- Medical footprint
- Transportation (venues/emergent access/ hospitals)
- Rules concerning when the medical staff can enter the field of play
- International health insurance coverage

Preparation for major events should include a site visit by the lead clinician. A site visit allows for research of medical resources at the athletic venue and in the region of the interest, strategic planning for logistical challenges such as travel from venues to regional medical facilities, and the opportunity to develop professional relationships with event medical staff. Prior to the site visit, it is recommended to contact the local organizing committee's medial leadership and request guided tours of medical venues, anti-doping areas, and hospital facilities affiliated with the event. For international

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travel, engaging a medical translator may be helpful to facilitate accurate communication between parties.

Pre-travel Medical Evaluations

Reviewing each athlete's medical history and pre-participation examination prior to travel is a suggested best practice for the team physician. It is important to note any predisposing conditions, allergies, or current medications. If there is concern an athlete may not be able to participate due to a pre-existing health condition, the team physician should provide any additional tests or consultations required to make a clearance decision prior to travel. It is important for the sports medicine team to consider each individual and their specific needs. For example, when developing sports coverage plans for athletes with a disability, it is important to consider what additional equipment or medicine may be required for the successful management of their healthcare needs.

Medical Licensure and Travel to Treat

The traveling healthcare provider must be cognizant of laws and regulations regarding medical licensure, scope of practice, medical malpractice coverage, and policies on travel with medications and medical equipment. Travel medicine law is in a state of constant evolution, and a review of current practice acts should be made during preparation for travel. For example, in the United States, there currently are uniform federal policies allowing healthcare providers to treat their own athletes across state lines [3, 4]. In the European Union, there are variable regulations on travel to treat, some of which are difficult to understand [5]. Large international events such as the Olympic or Pan American Games often provide temporary limited licensure for clinicians traveling to treat their own team. The application process and scope of the temporary licensure can change from event to event. Malpractice carriers may not cover providers who are traveling outside of their medical jurisdiction. In a survey that explored insurance coverage and travel, 5 of 11 malpractice carriers stated they would not provide coverage to a provider who travels [5]. Medical leadership for major events should help educate and facilitate clinicians on the options for temporary medical licensure and the scope of practice associated with this licensure and discuss the availability of professional malpractice insurance for the event.

Traveling with Medicine

The traveling clinician must prepare to be compliant with customs and immigration regulations in the destination country before crossing international boundaries with controlled substances or medical equipment. These regulations are variable by state or country. The substances with greatest regulatory control are narcotics and psychotropic medications. An independent and quasi-judicial organization designed to provide international drug control (International Narcotics Control Board) does exist; however, each country most likely have their own regulations. Generally accepted principles in travel with protected substances include the following [6]:

- That travelers should be allowed to carry quantities of such substances for personal use, usually for use of up to 1 month.
- That travelers should also have a letter or prescription from their doctor if traveling with a narcotic substance (but not necessarily for psychotropic medications).
- These types of medications should be in the original container from the pharmacy.

However, due to the potential risks associated with travel and narcotics, it is generally safer to avoid carrying these substances over international boundaries. Partnering with local physicians to provide these prescriptions, if necessary, may be a preferred strategy for the traveling clinician. In the United States, the Drug Enforcement Agency (DEA) does not support team physicians traveling with narcotics. One should also be aware of unanticipated international challenges of traveling with medications. For example, erectile dysfunction medications may not be brought into South Korea. Generally speaking, most typical sports medications are not an issue.

Athletes who are currently taking medications should review their medication list prior to travel to ensure an adequate supply for the duration of the trip and check for compliance with their sport federation's anti-doping regulations. Resources for the clinician performing medication reviews for banned substances include the following:

- The World Anti-Doping Agency (WADA) prohibited list www.wada-ama.org
- The United States Anti-Doping Agency (USADA) athlete guide to the prohibited list ww.usada.org
- The Global Drug Reference Online www.globaldro.com

Medical Equipment

It is important to understand there may be restrictions and regulations regarding crossing international borders or shipping medical equipment and supplies. Many countries' customs and immigration laws are specific about what types of high-technology medical equipment can be brought into their country. For example, in Brazil high-technology equip-

ment cannot be moved through a city without accompanying documentation. Shipping medical equipment can require significant time to arrival because of the delay in clearing the equipment through customs. These delays (both into and out of the country) should be considered in advance of sending the shipment to distant locations. International shipping companies are a valuable resource because they are well versed in these processes.

Medical Emergency Response Planning

The medical emergency response plan (MERP) or emergency action plan (EAP) is a cornerstone to successful athletic event management. A MERP must take into consideration the medical care at the field of play, staging areas, and lodging locations. Each of these may require a referral to a different hospital for emergency services. During the site visit, face-to-face visits with emergency personnel and regional medical leaders in advance of the event are imperative in establishing relationships and lines of communication. During these meetings, objectives should include learning about regional emergency procedures, including how to activate emergency response number(s). There will be significant variations in different regions of the world. The standards of care, training requirements for the medical staff, quality of service provisions, and availability of resources for emergency services vary around the world. In the authors' experience, the clinician should avoid making assumptions regarding the quality of care in a region until those services have been evaluated.

Each sport venue should have its own emergency action plan. It is important to understand the emergency action plan for the venue that you are traveling to. There are inherent challenges in the management of an emergent situation in unfamiliar surroundings with unfamiliar people. The medical team should practice implementing the EAP prior to the event. This will allow the lead clinician to assess the emergency response and skills of the event medical team in order to build solutions for any perceived deficiencies. Deliberate practice provides opportunities for education of the affiliated providers and the further internal development of contingency plans. Each member of the medical staff must understand their specific role and be capable of locating emergency equipment and activating emergency services in a timely manner. For example, identifying the locations for AEDs provides for early access to the AED as a critical component to survival of sudden cardiac arrest the athletic setting [7].

The MERP/EAP should include specific protocols for clinical care, destination of transport, and communication pathways for specific medical situations. Examples of situations that should be included in a MERP/EAP comprise the following:

- Head injury
- Spine injury
- Unresponsive patient
- Cardiac arrest
- Impaled object
- Death and repatriation
- Terrorist event
- Motor vehicle accident
- Multiple victim accident
- Massive bleeding
- Musculoskeletal traumas (fracture/dislocation)
- Air ambulance access
- Designation of evacuation locations, locally and within the continent

Communication during and after an emergency is crucial to the outcome of the patient's health. Legal documents allowing for release of health information to key stakeholders, HIPPA compliant messaging protocols, and emergency contact information for each delegation member should be included in the administrative plans prior to travel.

To build a functional sports medicine team when traveling, the defined chain of command must be predetermined. Typically, the team physician will be the individual that takes the lead in providing direction regarding the management of injured individuals requiring urgent care. When traveling a staffing plan should be pre-identified regarding which level of injury will require a medical staff member to travel to assist in the management of the injured athlete. When key medical staff are displaced to accompany an athlete who is being transported, the chain of command at the venue will change.

A communication plan regarding significant injury or illness will require advanced planning. It is imperative the medical updates regarding the health status of an injured individual are clearly defined and followed. Consider who and how medical updates will be provided and to whom information will be shared. Be cognizant to ensure that the protection of personal health information does not change because the injury or illness occurred at an athletic event.

Preparing for Travel Health Risks

There are unique physiological and environmental stressors associated with travel that can be minimized with appropriate preparation. The health stressors of traveling include changing time zones, entering different altitude, climate, changes in diet, and cultural/societal health challenges. Risk management should include identifying and educating travelers on the specific health and safety threats, recommending precise preventative measures (vaccinations, medications, repellants), and providing the mechanisms necessary for safe travel [8].

Each geographic region has endemic infectious disease risk. Many infectious diseases can be prevented with appropriate immunization and chemoprophylaxis treatment. Infectious disease risks are constantly changing; therefore, it is important for the healthcare provider to seek current information regarding risk mitigation for the destination of interest. The CDC maintains an excellent online resource for the traveler with specific guidelines on risk and prevention, including routine and destination-specific vaccination schedules [9].

The most common types of illness in travelers include upper respiratory and gastrointestinal complaints [10]. Athletes traveling over 5 time zones have a 2–3 times greater risk of illness [10]. Athletes traveling to high altitude may experience sickle cell crisis, acute mountain sickness, or non-acute mountain sickness or gastrointestinal disturbance [11, 12]. Changes in food and water quality in a new destination can be risk factors for travelers' diarrhea [13]. One study of Americans traveling to developing countries found that 46 percent of the travelers experienced diarrhea [14].

Preventative strategies for travel illness include the following:

- Frequent hand washing and use of sanitary foams and gels [15].
- Prophylactic use of probiotics. In one study, lactobacillus GG was found to prevent 49% of travelers' diarrhea [16].
- Avoidance of unbottled water; ice cubes; uncooked vegetables; unpeeled fresh fruit; unpasteurized mild, raw, or undercooked meat and seafood; and food from street vendors [17].

Heat and humidity can be a significant concern for the traveling athlete. The CDC has declared heat illness to the leading cause of death in high school athletes [18]. An athlete may be at increased risk for heat illness when there is a significant increase in heat or humidity as compared to their home environment. Healthcare providers practicing in hot and humid environments require emergency action plans for prevention of heat illness with acclimation protocols, rapid recognition of heat illness through education of staff and coach, and an emergency action plan for rapid cooling in the event of heat illness occurrence [19].

Heat acclimatization is the most important intervention to reduce physiologic strain due to heat [20]. As a rule, acclimatization takes 7–14 days to increase plasma volume. Planning itineraries that allow for appropriate acclimatization may assist with heat illness prevention and athlete performance in competition.

Jet lag occurs when there is disordered sleep after travel over more than two time zones [21]. Jet lag can result in impaired physical and cognitive performance [22]. The symptoms of jet lag can be reduced with appropriate planning. Tips to reduce jet lag include the following [22]:

- Prior to departure, shift bedtime 1–2 hours closer to the destination of the time zone.
- Drink water and avoid caffeine and alcohol during travel.
- Use strategic naps to reduce sleep debt.
- Increase exposure to bright light in the morning in the arrival time zone.
- Consider melatonin and zolpidem to induce sleep if necessary.
- Consider caffeine to induce wakefulness if necessary.

Medical Documentation

The traveling healthcare team must have a process in place to allow for daily documentation of medical services to maintain standard of care. The medical team should have access to each member of the athletic team and entourage's medical history and pre-participation exam findings for medical planning and as a reference in the event of an emergency. Electronic health records (EHR) have provided an excellent solution for this issue. Secure, cloud-based EHR programs allow for access to medical records and documentation of medical care at any site with Internet access. Access to patients records in real-time facilitates integrated care from multidisciplinary medical teams. Medical teams utilizing electronic medical records have reported greater efficiency, quality of care, and patient satisfaction as opposed to those using paper records [23, 24]. At mass sporting events, EHRs provide for improved injury surveillance and risk factor identification, an important first step in the injury and illness prevention process [25].

The first step in injury prevention is injury surveillance [26]. Participation in injury surveillance programs protects the athletes from additional injury or illness [1]. The data from these types of studies will direct future planning efforts in regard to the effective and efficient utilization of limited resources to protect athlete health. As previously noted, the same standards for protecting health information within the EHR in a clinical setting are also applicable in the world of sports.

After Action Report

After action reports (AAR) generated by medical teams provide the most valuable information for planning of subsequent events. Post-event documentation of the logistical details (who, what, when, where, how) provide a resource for future medical teams on how to prepare. Plans for staffing, inventory management, and travel and preventative planning can all be improved with the use of prior data. Large sport organizations may consider utilizing a standard AAR template to develop consistency in documentation of previous events.

Seeking feedback from key stakeholders, both internal and external to the medical team, can be an effective tool for measuring outcomes. Performance audits in the form of interviews or surveys can provide insight into what went well and what areas of improvement can be made. Targeting individuals or populations who are openly critical of services to provide feedback to the medical team may be an effective method to increase satisfaction at future events. Openly sharing results of these audit tools with others provides a format for transparent troubleshooting in advance of future events.

Conclusion

In planning for sports coverage for traveling teams, the human aspect of the sports medicine team can be more important than the knowledge factor of the individual [27]. The leader of the traveling team should surround themselves with individuals who are motivated to serve the delegation with the appropriate skill sets to ensure positive outcomes. The time commitment for successful planning for sports medicine coverage for traveling teams is a key predictor to a successful outcome. It is important to build upon the past experiences of individuals who have already provided sports medicine services while traveling with teams in order to provide a framework for success.

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Mass Sporting Event Coverage

3

Andrew Pasternak and Brian J. Krabak

Key Points

- Endurance events such as marathons, ultramarathons, and Ironman distance triathlon provide unique medical issues because of the distance of the events and how spread out the competitors are during the race.
- The amount of medical coverage for an event depends on numerous factors including the number of participants, race distance, race location, and philosophy of the race.
- Clarifying the expectations for medical coverage with the race director and competitors prior to the event is essential.
- In general, athletes should be aware that the more remote the event, emergency medical response time will likely be longer. In such events, athletes may need to be more responsible for their medical care.
- The lines of communication for emergencies need to be clearly defined before the race to optimize response time for first responders and race medical staff.
- Medical directors should take into account specific issues to their race (altitude, heat, cold, etc.) and ensure the medical protocols, equipment, and athletes are prepared for those issues.

Introduction

Compared to "traditional sports" that take place in a stadium or single location, most endurance events take place over a large geographic area. As an example, marathon courses tend to be either point to point or a large loop. As a result, the medical team has to be vigilant for medical emergencies along the entire 42-km course. Distances for the medical team to cover only increase for ultramarathons and Ironman distance triathlons. During endurance races, athletes also spread out over a large area due to their different paces. In the latter portions of a marathon, there can be over 25 km from the leaders to back of the pack and the last athletes may finish 4–5 hours after the first athletes. The longer the race, the geographic spread and time gaps between the competitors become even larger. Finally, as endurance events get longer and more difficult, cutoff times for some races are now 100–150 hours. As a result, sleep deprivation becomes more of an issue not only for the athletes but for the medical team as well. Response time to athletes needing medical care in off-road races is typically longer as access to the athlete is more difficult. In triathlon, there is the additional concern of medical problems occurring during the swim or bike portions of the race, adding another level of complexity. In some instances, the medical team for the event may need to respond to the medical needs of not only the athletes but also for volunteers and spectators, especially at larger or remote events. Because of these logistical issues, medical coverage in endurance events requires significant advanced planning.

Race Day Preparation

Relationship with Race Director

The first step to prepare for medical coverage of an endurance event is to discuss expectations of the medical team with the race director. There is no set standard for coverage

A. Pasternak (✉)
Silver Sage Sports and Fitness Lab, University of Nevada Reno School of Medicine, Reno, NV, USA

B. J. Krabak
Department of Rehabilitation, Orthopedics, and Sports Medicine, University of Washington and Seattle Children's Sports Medicine, Seattle, WA, USA

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of ultra-endurance events [1]. Key factors to know include an estimate of the number of participants, potential weather conditions, and geographical issues that could affect an athlete's health. There also needs to be frank discussion about budget. Some races have the financial resources to pay for onsite EMS coverage, medical supplies, and/or compensation to medical providers for coverage. Other race directors offer sponsorship opportunities or other acknowledgments for in-kind donations of medical supplies and staff. Pay or other compensation issues may affect the provider's legal coverage, which will be covered later.

The role of the medical team varies from race to race. In many races, the medical team's goal is to stabilize and triage athletes until EMS can arrive. In larger races with more resources, the medical team may play more of a role in treating acute medical issues helping the athlete complete their goal. In races where treatment is possible, it is wise to decide ahead of time if any specific treatments would disqualify an athlete from continuing (e.g., giving IV fluids or oxygen during the race, which in most races disqualifies the athlete) [1] [2].

While somewhat uncommon, the race director and medical director need to agree on a protocol if the medical staff feels an athlete is unable to safely continue but wants to continue. In most situations, the race director should defer this difficult decision to the medical staff. The medical staff needs to be very thoughtful about this decision and should understand the culture and mindset of ultra-endurance athletes [3]. In all races, there needs to be an agreement that the safety of the athletes is paramount, recognizing there are inherent risks involved with endurance racing.

For an inaugural event, the medical director should help the race director decide the best locations for both aid stations and medical stations. Road marathons will typically have aid stations for hydration and nutrition generally anywhere from every 3 to 5 km [4]. For off-road or wilderness events, the distances between aid stations are usually greater and are based on where volunteers can easily access the course. At most races, there may not be enough medical personnel to have a medical station next to every aid station along the course. Medical stations should be near aid stations but separate enough to ensure the medical team has space to work without interference. In general, because athletes have more medical issues later in the race, it's preferable to have more medical personnel and medical stations concentrated in the later portions of the race if possible [5, 6].

When athletes drop out of the race due to non-emergent medical conditions or fatigue, they often need transportation back to the start or finish line. The logistics for transporting athletes should be planned ahead with the race director.

Legal Issues

While the likelihood of a lawsuit arising from coverage at an endurance event is low, it could still potentially occur. Between the race director and the medical director, it is important to make sure the event and the medical personnel have appropriate liability insurance in case of a medical lawsuit [7].

All events should have general event liability insurance. General liability insurance, however, may not include coverage for medical care provided by volunteer medical staff. Providers should check with their individual malpractice coverage to determine if their plan will cover the care they provide at these events.

While most states have enacted Good Samaritan laws, these may not apply to medical care within the context of an endurance race. Most Good Samaritan laws generally apply to providers who provide care for a life-threatening condition at the scene of an emergency; thus, much of the care provided at endurance races may not fall under those provisions. The Good Samaritan laws, however, vary from state to state. If a provider is receiving compensation (e.g., race merchandise) from the race to provide coverage, Good Samaritan laws would likely not apply to that provider.

It is beneficial for the event organizers and medical staff to have athletes sign an event waiver. While these may not prevent a lawsuit, they may help the event defend themselves in a lawsuit as they document the athlete was aware of the risks involved with the event.

Medical care provided at races falls out of the definition of a "covered entity" by the 1996 Health Insurance Portability and Accountability Act (HIPAA) privacy rule [8]. It is prudent, however, for medical staff and race directors to treat personal medical information as confidential, realizing some information will be needed to be transmitted over race communications in the case of an emergency. Medical staff should also be careful about posting information or photos on social media without athlete consent.

The medical director should be aware of individual state regulations pertaining to visiting medical staff providing care at events without a valid license in the state where the event is taking place [7]. For events and races occurring in other countries, it is advisable to discuss the planning and preparation with local EMS and medical personnel familiar with the area.

Athlete Preparation

Prior to the race, athletes should be aware of what medical services will be available and where they can access the medical services on the course. Again, there is no set standard for the amount of medical coverage needed for endur-

ance races. Depending on the size, location, race logistics, and cost of registering for an event, some events may be able to have fairly robust medical teams throughout the course, while other races may only be able to have medical personnel at the finish line, if that. Especially for athletes who have competed in large road races and are now competing in smaller trail races, the expectations of the medical care may be greater than the race can provide. Some events, such as the Badwater 135 running race, have no specific medical team and require each athlete to provide their own medical team [9]. The infamous Barkley Marathons have no helpers at aid stations or medical stations during each 20-mile loop [10]. Most importantly, race personnel need to communicate with the participants prior to the race so that the athletes are aware of how self-reliant they need to be.

Depending on the challenges of the race, location, and the number of entrants, some races will ask for the athlete's medical history and/or have the athlete get medical clearance from their regular physician. Again, there is no set standard for when to do this and the best method to do this. If the medical team decides to collect pre-race medical history, the medical director should review these records prior to the race and contact the athlete if there are any unusual conditions requiring additional information. It is helpful to remind athletes with chronic medical conditions, such as diabetes, seizure disorders, or severe allergic reactions, to wear a medical ID bracelet or have a medical ID tag visible. This small item can immensely help the medical responders provide prompt, appropriate care.

Finally, for races in remote conditions, athletes may want to consider purchasing supplemental insurance in the unlikely cost of a helicopter evacuation. For example, the Air Med Care Network (http://www.airmedcarenetwork.com) offers memberships that cover the cost of a helicopter evacuation for less than the cost of many race registrations.

Contingency Planning

Endurance events are typically outdoor events where the athletes are subjected to the elements for anywhere from 2 hours to 2 days or more. Athletes are also typically exercising at a fairly high level of exertion. Because of this, the race director and medical director should discuss plans for extreme or dangerous environmental conditions. These plans make up a critical portion of the race protocol. Some races are infamous for their environmental extremes and athletes train and plan specifically for those conditions. In other cases, when unexpected environmental conditions occur, the race director and medical director need to collaborate on making a decision that is not only safe for the athletes but also safe for the volunteers covering the race. This can include unexpected storms, flooding, and fire. If EMS responders are already busy helping the surrounding community due to the underlying concern (especially in the case of fire and flooding), it is prudent to not stress the EMS system further and cancel the event.

Organizing Your Medical Team

Organizing your medical team starts with identifying an experienced medical director. The medical director is the individual responsible for overseeing all aspects planning and operations of the medical team on race day.

In addition to other appropriately trained physicians, the medical team may include athletic trainers, emergency medical technicians, podiatrists, paramedics, physical therapists, massage therapists, nurses, and students. Having a blend of professionals with complementary skills is optimal.

Staffing numbers will depend on the size of the event and distance of the event and location. Generally, one can plan for 1–2 physicians per 1000 runners at a road marathon along with other medical professionals [4]. Knowledge of participant demographics and historical data of medical needs at past races will be helpful in planning the size and make-up of the medical team [11].

Novice medical team members may not be familiar with the conditions they may encounter on race day. Some communication, either in a pre-race meeting or electronically, about the recognition and care of commonly seen issues including blisters, gastrointestinal distress, appropriate use of intravenous (IV) fluids, thermal-related disorders, and exercise-associated hyponatremia, is advisable.

For triathlons, the medical team needs to be prepared for medical issues arising from the swimming and bicycle portion of the races [12–14].

Because of the amount of time some aid stations may need to be providing care, especially in longer ultra-endurance events, it is helpful to organize your medical staff into shifts to help combat sleep deprivation and fatigue.

Coordination with EMS

No matter the size, distance, or location of the race, it is critical for the race director and medical director to communicate with local EMS responders prior to the race. The EMS leaders can answer questions about the appropriate jurisdictions to contact when races go through different cities, counties, states, or countries. It is useful to review the course with the EMS team and identify the most accessible points for responders if an athlete needs to be evacuated, which could include helicopter-landing areas. EMS can make sure that

they have appropriate staffing for not only your event but also for any other large-scale events in the community. Recent data from the Boston Marathon has also shown that EMS response times and patient outcomes are adversely affected on race day [15]. Finally, in light of the 2013 Boston Marathon bombing, for large events taking place in cities, some advanced planning for terrorism attacks and possible mass casualty is now warranted.

Race Protocol

Part of the responsibility of the medical director is to create and update a medical race protocol. The race protocol should be a practical and frequently referenced document. The race protocol is an opportunity for the medical team to put into writing the action plans needed for various scenarios. Generally, this affords the medical director a chance to review and contemplate the logistical issues that the medical team may encounter. In addition, it gives the race medical staff a single resource to review before the event and refer to during the event.

While the race protocol needs to be complete, it also needs to be concise enough to make it useful. Writing sections in brief "bullet" style and simple language are helpful for the medical staff. Keep in mind the intended audience for the race protocol as the medical team may consist of physical therapists, nurses, podiatrists, and other non-physician medical staff.

General sections of the race protocol include the following:

1. Background of the event including the number of participants/spectators and geographical and climate issues
2. Communications protocols
3. Emergency contact numbers of race personnel and EMS
4. Review of presentation and treatment for commonly seen and critical care issues
5. Response plans in case of catastrophic events (flooding, fires, lightning, terrorism, etc.)
6. Medication and IV protocols
7. Medical kit inventory

Medical Documentation

The medical staff should document medical encounters that occur during the event. This is particularly beneficial when an athlete requires medical evaluation and will attempt to continue in the event or if the athlete needs to be transported for additional care. It is highly advisable to document any prescription medications given to athletes, including medical contraindications to the medications, possible side effects, and treatment response. Generally, minor issues such as blister care or minor abrasions do not warrant medical documentation. In general, a one-page piece of paper with check boxes allows providers to record the interaction quickly and simply (Fig. 3.1).

Medical Kit (Table 3.1)

- In addition to basic first aid and medical supplies, there are several items that may be particularly useful in the endurance event setting.
- Adequate blister care supplies including elastic tape, moleskin, and blister pads are needed given the high frequency of skin issues during endurance events [16].
- Automatic external defibrillators (AEDs) should ideally be available throughout the race course in the event of sudden cardiac arrest. These are obviously more helpful in a road race than in a wilderness race as response time to the downed athlete would likely be shorter in a road event.
- Point-of-care blood chemistry analyzers (i.e., i-STAT) are useful when hyponatremia, hypoglycemia, or other metabolic abnormalities are suspected. They can be difficult to operate, however, in temperature extremes.
- Rectal thermometers are essential for measuring accurate core temperature when heat or cold illness is suspected. The thermometers should have the capacity to measure low core body temperatures (which not all thermometers have).
- The medical kit should be organized in clearly marked boxes, clear bags, or compartments. The medical team should familiarize themselves with the kits when they arrive on site.
- Prescription medications in the medical kits should only be given out if a provider with a valid pharmacy license is caring for the athlete.
- The medical kit can be simplified if the focus of the medical team is triage and stabilization of athletes and awaiting EMS response. In scenarios where EMS response times could be longer due the remoteness of the event, the medical kit may need to be appropriately expanded.
- Because of the risk of exercise-associated hyponatremia and the availability of a point-of-care blood chemistry analyzers, the medical director may want to include hypertonic saline with dosing recommendations as part of the medical kit, assuming the medical team has the appropriate staffing to administer an IV [17]. Most EMS responders do not routinely carry hypertonic saline.

Medical Team/Evaluator: ______________________________
Date: July 16th 2016 July 17th 2016

Arrival Time:____________ **Departure Time:** ______________

Location (circle one): Start/Finish Tunnel Creek Diamond Peak Hobart

Athlete Name: ____________________ **Age:**______
Gender (circle one): M / F
Race Number: ____________________________

Distance (circle one): 55K 50 mi 100 mi

PMH:

Allergies:

Meds:

Signs/symptoms:

☐ Fatigue	☐ Abdominal Pain	☐ Confusion	☐ Blisters
☐ Lightheaded	☐ Nausea/Vomiting	☐ Wheezing	☐ Chafing
☐ Blurred Vision	☐ Diarrhea	☐ Muscle Cramps	☐ Skin Abrasion
☐ Chest Pain	☐ Feeling hot		☐ Skin Laceration
☐ Shortness of Breath	☐ Feeling cold	Other__________________	

Vital Signs	Time:			
Temp				
BP Sitting				
BP Lying				
Heart Rate				
Resp Rate				
Pulse Oximeter				
Weight				

HPI:

Exam:

Labs/Other findings:

Diagnosis:

☐ Exercise assoc collapse	☐ Blisters	☐ Muscle Cramps
☐ Dehydration	☐ Skin Abrasion	☐ Muscle Strain
☐ Hypothermia	☐ Skin Laceration	☐ GI Distress
☐ Heat Exhaustion	☐ Ankle Sprain R L	☐ Asthma Exacerbation
☐ Hyponatremia	☐ Knee Sprain R L	☐ Anaphylaxis

Other____
____

Treatment:

☐ Supine with legs elevated	☐ Blister care	☐ Albuterol Inhaler
☐ Oral fluids	☐ Wound Dressed	☐ Ice Pack
☐ 3% Hypertonic Saline _____cc infused	☐ Ace Wrap	☐ Zofran 4 mg SL

Other____
____

Final Disposition Plan (check all that apply):

[] Continue participation	[] Disqualified from further participation	
[] Patient able to ambulate independently.	[] Patient able to tolerate po	[] Patient urinating normally
[] Discharged to self	[] Discharged with friend/family member	[] Ambulance evacuation

Provider Signature: ________________________ **Date:** __________

Fig. 3.1 Medical documentation form

Table 3.1 Medical kit inventory

Emergency drugs	Oral airways
Adrenalin	Pulse oximeter
Benadryl	Glucometer
Zyrtec or other antihistamine	I stat
Nitroglycerin tabs (cardiac emergency)	AED
	Mylar Space heat blankets
Aspirin tabs (cardiac emergency)	SAM splints
Albuterol inhaler/nebulizer	Oxygen
Tylenol	Ambu bag
Loperamide	Mega Mover
Tums	Thermometer (hypothermic)
PPI (Nexium or other)	Tampons
IV Solutions	**Podiatry supplies**
Normal Saline	Moleskin
3% Hypertonic Saline	#11 blades
IV starts	Spenco 2nd Skin, 1 × 1
Needles, 20 gauge, 18 gauge	Baby powder
Syringes, 5 or 10 cc	Assorted gloves
IV tubing	Scissors
IV starter kits	Vaseline
Tape and bandages	Neosporin
Tape, 2″, 3″	4 × 4's
2″ or 4″ Coban	2 × 2's
Ace Bandages, 2″, 4″	Duct tape
Steri-strips	Alcohol wipes
Band-Aids	Hydrogen peroxide
Gauze	Cotton balls
4 × 4	Ziploc bags
2 × 2	KT tape
Tube gauze #2, #4	Zinc oxide ointment
Others	
Eye lubricant drops (BSS)	
Alcohol preps	
Betadine preps	
Benzoin	
Cotton applicators	

Logistics

- The main medical tent should be clearly visible near the finish line.
- For larger races, organizing the finishing line tent into sections including a triage area, general medical area, and intensive medical area may help expedite care. A physician with training in sports medicine or emergency medicine should be designated to perform triage duties for athletes presenting to the medical tent.
- In general, medical aid stations should be established approximately every 3–5 km on the race course for a major road marathon. For events on trails or smaller races, the distance between medical aid stations is usually dependent on access issues and is generally much further apart.
- Aid stations can be set up alongside medical stations but should remain physically separate to respect space and avoid congestion in and around the medical stations.
- If race volunteers are given t-shirts, it can be helpful to give medical personnel a distinctly colored t-shirt so they can be easily identified.
- Medical tents should be protected from the elements as much as possible and have the ability to both warm hypothermic athletes and cool hyperthermic athletes.

Communication

- Two-way radio or cellular communications can be used to maintain open lines of communication between the main medical tent, on-course medical aid stations, and mobile medical team members on patrol.
- All communications should be tested prior to the event as communication can often drop in remote areas. Backup systems for communication may need to be considered for larger races, especially if unexpected conditions affect communications (e.g., fire or power outages affecting cell phone coverage).
- The chain of command and protocol for communication on race day should be distributed to all medical team members prior to race day.
- Prior to the event, it should be determined which medical personal should be responsible for activating the EMS system. Many races cross through multiple counties, states, and sometimes countries. The medical director and medical team should have a clear understanding of which EMS system to activate in the case of an emergency.

Transportation

- Endurance events can involve numbers of participants and spectators on the order of thousands to hundreds of thousands. Races of this magnitude often require closing streets and shutting down of thoroughfares to accommodate for the massive number of runners and spectators during the event. During ultra-endurance events, many athletes now have a "team" that helps them at aid stations with nutrition, personal care, and motivation. While the team needs to have access to their athletes, it may not be advisable to allow the athlete's team access to parts of the aid station and/or the medical station without permission.
- Medics on bike patrols or on all-terrain vehicles (ATV) can be used to monitor for down or distressed athletes in between aid stations for both road and trail race courses.
- Designated road cars and mobile medical vans can be used for transit of supplies and non-critically ill or injured runners to and from the race course and main medical tent.

Post Race Wrap-Up

The medical director and race director should always specifically thank the medical team for their efforts after the race. It is also important to query the medical team about medical

and logistical issues that came up during the races and how they were handled. Specific areas to look for suggestions and improvement include supplies, adequate staffing, and communications.

What Do the Physicians Need to Know While Covering an Endurance Event?

- The size and physical area that endurance events traverse presents a unique logistical challenge in allocating medical resources on race day.
- There are typically fewer athletes requiring medical assistance early on in the race than in the later stages, and medical team members should be distributed appropriately.
- Cooperation with local fire, police departments, and EMS teams is essential in the event of ill or injured athletes requiring transfer to a higher level of care.

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4

Stabilization, Immobilization, and Transportation

Brenden J. Balcik and Aaron J. Monseau

Key Points

- The term stabilization may apply to the athlete as a whole or simply an injured extremity.
- On-field injuries are inevitable and as such may need immediate care. Splints should be used to provide basic stabilization, pain control, and maintenance of neurovascular status.
- Splints placed on the field or sideline are not meant to provide definitive management.
- In the event of a life-threatening injury, the initial evaluation should always begin with Airway, Breathing, Circulation, Disability, and Exposure (ABCDEs).
- Equipment removal may be necessary for the evaluation of the injured athlete, and it is important to be familiar with different types of equipment and techniques for removal.
- On the field spinal immobilization may be necessary.
- It is imperative the provider know indications for spine immobilization of the injured athlete.
- Fractures and dislocations may occur and the on-field provider should be familiar with various reduction techniques for effective injury management.

Introduction

In the United States, both the Physical Activity Council and the Bureau of Labor Statistics report overall increasing trends in sports participation over the last 5–10 years [1, 2]. As such, sports-related injuries can be expected to rise as well. Sports-related traumatic injuries may occur anytime or anywhere. Whether it is on the field, on the court, in training, or in competition, these injuries require prompt evaluation, diagnosis, and management. More often than not, diagnostic modalities are not readily available, and this poses a unique challenge to sports medicine physicians. A solid understanding of injury mechanisms, presentation, and management is paramount for those in the role of sports medicine physician. In this chapter, we review the recognition and assessment of sports injuries and how to stabilize, immobilize, and transfer the athletes to the nearest medical facility.

Stabilization

In sports-related trauma, the term stabilization may apply to an injured extremity or to the athlete as a whole. In some cases, this means simply splinting a grossly deformed extremity with what is available such as air casts, malleable splints, or athletic equipment. In other more severe and potentially catastrophic cases, this refers to stabilizing the athlete in the face of a life-threatening injury or condition. In this section, we review basic stabilization concepts of injured extremity before transportation to a medical facility. We also review the steps required for the initial assessment and stabilization of the severely injured athlete.

Splinting

For sideline, splints should be used to provide basic stabilization, pain control, and maintenance of neurovascular status after compromise. Splints placed on the field or sideline are not meant to provide definitive management [3, 4]. In addition to fractures, they can be useful in the acute management of sprains, joint infections, tenosynovitis, acute arthritis, gout, and lacerations over joints [5, 6]. Specific diagnoses are covered throughout this book, but here, we will cover

B. J. Balcik (✉) · A. J. Monseau
Department of Emergency Medicine, West Virginia University School of Medicine, Morgantown, WV, USA
e-mail: bbalcik@hsc.wvu.edu

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basic splinting techniques, types of splints, and some pearls and pitfalls. There are many splinting videos on the Internet that may be helpful. As opposed to casts, which have largely fallen out of favor for the management of nearly all acute injuries, splints allow for swelling as they should not be circumferential on an injured extremity [7].

Historically, and even now when needed, any rigid material (or even a pillow) can be used to fashion a splint when they can be adequately secured to the patient. Splinting materials vary greatly in price and utility. Malleable aluminum splints are widely marketed for pre-hospital and sideline use. Bean-bag splints that utilize a vacuum to achieve rigidity are popular as well. Classic splinting materials such as plaster and fiberglass, while not necessarily providing definitive management, can provide a few weeks of immobilization in some cases [6–8].

In addition to splints, there are several other types of products and devices used to treat injuries in the acute setting. Both the shoulder sling and shoulder immobilizer can be used to treat humerus fractures and shoulder injuries (Chap. 15, Figs. 15.9, 15.10, and 15.18). Ankle devices, such as a stirrup splint, lace-up ankle brace, or other ankle braces, provide stabilization for minor ankle sprains. A knee immobilizer can be used for a severely unstable knee but should be avoided if possible as it will slow recovery in most cases. There are braces marketed for each joint that can be useful for sprains or strains but are too numerous to describe here. Finally, crutches are an essential supply item to avoid weight bearing on an affected leg [6, 7].

Splinting Technique

After thorough inspection for lacerations and examination including neurovascular exam, padding is required to prevent skin breakdown. Cotton rolls or sleeves that can be cut to length are preferred. Cotton roll gives the benefit of easily modifying the amount of padding by simply increasing or decreasing the overlap. Take special care to make sure bony prominences are well padded. When fashioning a make-shift splint from things such as tree branches or even malleable aluminum, padding may not be necessary but, if available, will still provide additional comfort [6–8].

Plaster was the splint material of choice until relatively recently when fiberglass splints have shortened the time for the splint to harden. Even the fastest plaster splints still take several times as long to harden than fiberglass splints. When plaster is layered through to 15 or more layers, it is much stronger than fiberglass so plaster is still preferred for some lower extremity splints [4, 6, 7]. Some products will come with a cotton covering which makes application even faster.

When wetting the plaster or fiberglass, one should only use cool or tepid water. While this will slow the hardening of the splint, the exothermic reaction required for the splint to harden can actually cause a burn of the underlying skin if warm or hot water is used [6, 7]. Once the splint material is applied, an elastic bandage should be used to hold the splint in place. This elastic bandage should never be stretched tight as this will increase the risk of neurovascular compromise and possibly even compartment syndrome. The role of a splint is to immobilize not to provide compression or reduce swelling [4, 6–8].

After the splint is secured with the elastic bandage, the splint should be molded to provide adequate immobilization. This will almost always require three points of pressure with one near the center in the opposite direction as one proximal and one distal. This is especially important in fractures requiring reduction where the center point of pressure should be directly over the fracture line [4, 7, 8].

Once the splint has hardened, the neurovascular status should be rechecked distal to the splint. Other complications, such as burns, pressure sores, and infections, should be monitored. If any such complications are suspected or if the patient has ongoing, significant pain, the entire splint should be removed and the extremity re-examined. For those patients only complaining of increasing pain, a short trial of loosening of the elastic bandage may be attempted, and a full removal is warranted if this does not resolve the pain within minutes [3–8]. See Table 4.1 for more details about common splints used in sports injuries.

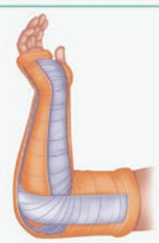
Caveats

- Bony prominences should be well padded.
- "Measure twice and cut once" with splinting material.
- Use cool or tepid water on splinting material (*NEVER HOT WATER*).
- Avoid getting padding wet.
- To avoid neurovascular compromise, elastic bandage should not be stretched tight.
- Splint material must be molded to provide adequate support.
- Always recheck and document neurovascular status after applying splint.
- When complications are expected, take the splint off and examine the area.

Casting

Casting should not be used in sideline management; in fact, casting should be used for very few emergency department diagnoses and only when the physician who will be providing follow-up agrees. An acute injury will typically swell for at least a few days, and a cast may precipitate neurovascular compromise and even compartment syndrome. If a cast is applied at a later time, the same general principles of splinting as above should be followed. Specific casting types will not be covered in this chapter [4–7].

Table 4.1 Common splints used in sports injuries

Picture	Type	Uses	Tips
	Thumb spica	Scaphoid fracture seen or suspected, De Quervain tenosynovitis	Wine glass position
	Ulnar gutter	Ulnar fractures, phalangeal/ metacarpal fractures of 4th and 5th fingers	For metacarpal neck fractures, MCPJ at 90° with PIPJ and DIPJ fully extended (intrinsic plus position) to avoid contractures.This does not immobilize the radius due to supination/pronation
	Sugar tong	Distal radius and ulna fractures	Prevents supination/ pronation
	Double sugar tong or reverse sugar tong	Elbow, forearm, distal humerus fractures	Limits flexion/extension and supination/pronation. Good for unstable fractures.
	Long arm posterior	Elbow/forearm injuries, distal humerus fracture, both-bone forearm fracture	Doesn't completely eliminate supination/pronation (use sugar tong for this)

Stabilization of Athletes with Potential Life-Threatening Injuries

The initial evaluation should always begin with Airway, Breathing, and Circulation (ABCDEs) [9].

- *Airway:* Assess the airway. It is important to maintain in-line cervical stabilization.
- *Breathing:* Assess quality of breathing. Also, evaluated for airway obstruction, tracheal deviation, subcutaneous emphysema, or other evidence of airway emergency.

Table 4.1 (continued)

Picture	Type	Uses	Tips
	Coaptation	Humerus fracture	Most humerus fractures treated adequately with sling only
	Cock-up	Soft tissue hand/wrist injuries, wrist fracture, 2nd-5th metacarpal fractures	May choose to add dorsal splint for added stability. Can still supinate/pronate so not good choice for distal radius/ulna fracture
	Long leg posterior	Knee injuries, femoral condyle fractures, tibial plateau fractures or proximal tibia-fibula fractures	Very seldom used but can provide temporary stabilization for transport or pain control
	Ankle posterior	Distal tib/fib fracture, reduced dislocations, severe sprain,tarsal/ metatarsal fractures	About 12-15 layers of plaster needed. Adding stirrup reduces inversion/ eversion and useful if unstable
	Ankle stirrup	Similar to posterior splint	Reduces inversion/ eversion so great for sprains. About 12-15 layers of 4-6 inch plaster. Adding posterior increases strength and reduces plantar flexion

- *Circulation:* Assess for any active bleeding. Also, check pulses.
- *Disability:* Assess level of consciousness. Evaluate for any neurologic deficits.
- *Exposure:* This is not typically done on the sideline but involves undressing the athlete to evaluate for additional injuries [9].

Equipment will vary by sport. It is extremely important for the provider to be familiar with the types of equipment worn by the athletes and be well-versed and comfortable with removal techniques of the equipment. Historically, it had been recommended that protective equipment be left in place prior to transport. However, the most recent National Athletic Trainer Association (NATA) guidelines recommend

removal of protective equipment prior to transport [10]. The reason for this is to allow easy access to the airway and chest should the athlete's condition deteriorate during transport. Additionally, this allows the providers most familiar with the equipment (athletic trainers, on-field physicians, etc.) to remove the equipment and minimize any complication(s). While equipment removal technique varies by manufacturer, a general approach can be undertaken at times when equipment removal is necessary. The most trained provider should be at the head of the athlete, maintaining in-line stabilization [11, 12]. If in a prone or side-lying position, the patient should be log-rolled to a supine position (Fig. 4.1). This should be done with the help of multiple providers if available. If airway compromise is a concern, the athlete with helmet and face mask should have the face mask removed immediately using technique appropriate for the model of helmet and face mask [11–14]. If access to the chest is required, the jersey and any undergarment should be removed [15]. Helmet and pads should be removed by the most experienced providers. Care should be taken to maintain cervical stabilization during this process. Once a helmet is removed, there is a tendency for the head to "drop" forcing the cervical spine into extension. Every effort should be made to maintain the neck in a neutral position (Fig. 4.2). Again, removal technique will vary by manufacturer and is important for providers to be familiar with equipment and removal techniques [16, 17].

Immobilization

Spine Immobilization

Multiple societies including the National Athletic Trainer's Association (NATA), The American College of Emergency Physicians (ACEP), and national EMS organizations have guidelines regarding spine immobilization [10, 18–20]. It is important for the provider to be familiar with these guidelines prior to competition. The provider should also be aware of any local EMS guidelines prior to competition. The decision to initiate spinal immobilization can be a difficult one. However, it should first be noted that the term spine immobilization is somewhat misleading as multiple studies have shown that true spine immobilization is difficult to achieve. Instead, the concept of spine motion restriction has been introduced. Similar to spine immobilization, spine motion restriction is intended to prevent additional spinal cord injury. If the decision to initiate spinal motion restriction is made, a cervical collar and long spine board and vacuum stretcher (or equivalent) must be readily available [20].

Fig. 4.1 Head and neck immobilization. (Courtesy of Jenny Jule Van Meter, ATC)

Fig. 4.2 Transferring the athlete to a spine board using a lift or lift and slide technique

Indications for spinal motion restriction [11]:

- Blunt trauma with altered level of consciousness.
  - Spinal pain or tenderness
  - Anatomic deformity of the spine
  - Neurologic complaint or deficits (weakness, numbness, or paresthesias)
  - High energy mechanism of injury and any of the following:
    - Intoxication
    - Inability to communicate
    - Distracting injury

When performing spinal motion restriction, extreme care should be taken to avoid any traction or distraction of the spine. Manual in-line cervical stabilization should be applied until a rigid cervical collar can be applied. The manual stabilization should be maintained even after the collar is applied (until patient is secured to spine board or vacuum stretcher) as some studies have shown that there is still some degree of cervical spine movement despite the use of a cervical collar [12, 13, 20].

When transferring the athlete to a spine board (or equivalent device), the preferred method is using a lift or lift and slide technique (Fig. 4.3). This technique limits the amount of spine motion as compared to log-rolling techniques (Fig. 4.1). Cadaveric studies have shown log rolling to cause excessive motion of the spine and therefore increase the chance of worsening a spinal cord injury [16]. It should be noted that the technique used to transfer an athlete to an immobilization device is largely dependent on the number of providers present and the familiarity and comfort of these providers with each technique [11, 14, 16, 21].

Once transferred to the immobilization device, the use of foam blocks, towel rolls, and straps should be used to secure the athlete to the device. The decision on clearance

Fig. 4.3 Log roll from a prone position to a supine position

of the cervical spine can be difficult at times. Due to this difficulty and the potential catastrophic repercussions of improper cervical spine clearance, clinical decision rules have been developed to aid in the decision of clearance versus the need for cervical spine imaging [22]. Two of the most widely known guidelines are the National Emergency X-Radiography Utilization Study (NEXUS) Low-Risk Criteria (NLC) and the Canadian C-Spine Rule (CCR) [22]. These rules were developed to rapidly rule out significant cervical spine injury and allow for more selective ordering of imaging.

The NEXUS criteria state cervical spine imaging is indicated for any athlete with trauma unless they meet all of the following criteria [22]:

- No posterior midline cervical spine tenderness
- No evidence of intoxication
- A normal level of alertness
- No focal neurologic deficits
- No painful distracting injuries

The CCR recommends radiography for any athlete that has any high risk factor such as age ≥65 and dangerous mechanism (e.g., fall from ≥3 feet, axial load to head, motor vehicle collision at high speed) [22]. If the above criteria are not met, the CCR recommends radiography if there are no low risk factor to allow safe assessment of range of motion such as athlete in sitting position or ambulatory at time or delayed onset of neck pain or absence of midline neck tenderness. If low-risk criteria are met, then the athlete cervical spine range of motion can be tested. This involves rotation of the neck 45° to the left and right. If the athlete is able to do this without pain, radiography can be withheld.

If either the NEXUS or CCR criteria can be satisfied, then the athlete's cervical spine can be cleared without the use of imaging. The provider should be familiar with both sets of

criteria and be able to apply it in these situations, specifically in the younger population to avoid any unnecessary radiation exposure. For those athletes in which imaging is indicated, the type of imaging should be determined on a case-by-case basis. Please note that there is no consensus on the treatment algorithm for cervical spine trauma [23].

Transportation

Injured athletes should be transported to a medical facility as soon as possible. Modes of transportation are dictated by the severity of injury and locale. For example, air transport via helicopter may be required for the severe head or cervical spine injury or any time-sensitive injury such as an open fracture or dislocation. For austere environments, transport by all-terrain vehicle (ATV) may be required. Additionally, if the injury occurs in a rural area without timely access to a medical facility, air transport may be required. In the event of a severe traumatic injury, transport via ambulance may be required if the patient requires any type of fluid resuscitation or blood products (e.g., for hypotension) or if the patient requires any type of pain control with intravenous medications. Local EMS agency protocols should be referenced and followed should this be needed. Ultimately, the decision for mode of transport should be made by the on-site sports medicine provider.

Injured athletes often need to be transported to a hospital that can deliver immediate, definitive care. If not readily available (due to weather or distance), the athlete should be transported to the nearest medical facility for stabilization and subsequent transfer to the nearest trauma center for definitive treatment. In case of mountain sports injuries (e.g., skiing), the patrol usually transfers the injured athletes to the closest medical facility. The medical facility may have plain radiography available. Oftentimes, these facilities can consult specialist at larger trauma center by phone or telemedicine. The destination hospital should have diagnostic capabilities such as CT and/or MRI as well as specialist availability in the cases of intracranial, spinal, or other traumatic injuries. The transportation protocol as well as hospital destination should be defined in the Emergency Action Plan (EAP) [11].

Dislocations and Closed Reduction

Subluxations and dislocations are conditions in which articular surfaces of a joint are no longer concentric. A subluxation is the milder form of these conditions, whereas a dislocation is the most severe form. In a complete dislocation, the articular surfaces of a joint are no longer in contact. These injuries are extremely important to the sports medicine physician as they represent 3.6% of all high school athletic injuries [24]. The underlying principle in treatment of dislocations is to reduce as soon as possible. The reason for this is twofold. The first is to avoid or prevent any neurovascular compromise. Some neurovascular deficits that may be present after a dislocation may resolve after reduction. However, the longer a joint is dislocated, the higher the likelihood the deficit may be irreversible. The second reason for prompt reduction is the longer a joint remains dislocated, the more difficult the reduction may be. The on-field provider should be familiar with common dislocations and various reduction techniques. The provider should have an excellent grasp of joint anatomy and be aware of the potential for concomitant injuries. The decision to perform a reduction on the sideline ultimately comes down to the provider's comfort level and the length of time to definitive care [25]. Specific techniques for reduction will be discussed in other sections of the book.

Summary

Injuries secondary to sports-related trauma are common. It is imperative that the sports medicine physician be aware of injury mechanisms and patterns, immobilization, and transport protocols as well as reduction and stabilization techniques for these injuries. While the sports medicine physician may not be providing definitive treatment, prompt recognition and management of these on-field injuries will certainly prevent aid in the prevention of long-term complications.

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5 Anesthesia and Acute Pain

Alexander Ebinger and Spencer Tomberg

Key Points

- Pain management is multimodal and comprised of oral, intramuscular, intranasal, and parenteral medications as well as local and regional anesthesia.
- Different medications exist for oral pain management and should be selected based on individual patient factors.
- Non-opiate and opiate analgesic options should be chosen based on patient tolerance, allergies, injuries, and desired effect.
- Regional anesthesia can provide effective pain control and reduce the need for oral or parenteral pain medications.
- Providers should be aware of the signs and symptoms of local anesthetic systemic toxicity (LAST).
- Hematoma blocks and intra-articular injections can be beneficial for facilitation of painful orthopedic procedures.
- In the appropriate setting with adequate resources, procedural sedation can facilitate painful procedures.

Introduction

Pain management is a frequent requirement for sports medicine physicians, particularly for acute traumatic injuries. Musculoskeletal pain is the most common presenting symptom, but in the acute setting, pain from other etiologies such as splenic rupture, pneumothorax, dental trauma, or infection also must be considered. The pathophysiology of acute pain is multifaceted. In the peripheral nervous system, pain is mediated by nociceptive fibers (A-δ and C) that transmit painful stimuli (chemical, mechanical, thermal) via afferent fibers to the dorsal horn of the spinal cord. These signals progress up the spinal cord (mainly spinothalamic tract) and are processed in the somatosensory cerebral cortex [1, 2].

The experience of pain is variable, constituted of previous episodes of painful stimuli, physical and emotional maturity, social background, and family environment [3]. There are no objective tests for pain; therefore, the evaluation of pain is based on the patient's subjective description of their symptoms. The initial assessment should include details of the history including when the pain began and the quality, severity, timing, and exacerbating and alleviating factors. Pain is dynamic and needs to be continually reassessed. Tools for helping evaluate pain include but are not limited to visual analog scales, numeric rating scales, and adjective rating scales though these are likely more beneficial from a research standpoint than clinical care [4–6]. Eliciting whether or not a patient is experiencing pain and utilizing their response to guide analgesia is appropriate, though it may undertreat or overtreat pain. Careful assessment of certain patient populations at risk for oligoanalgesia such as children, elderly individuals, and those with communication barriers must be undertaken and continually reevaluated so as not to overlook appropriate pain management [7].

Management of pain is multifactorial. It includes emotional reinforcement, physical support, and pharmacologic treatment. From a pharmacologic perspective, medication therapy should be selected to match analgesia appropriate to the patient's pain. One must consider the etiology of the pain, such as nerve-mediated discomfort or inflammation from acute trauma. Adequate control of pain is important, not only for patient relief and satisfaction but also to facilitate further patient care and reduce the associated stress

A. Ebinger (✉)
Department of Emergency Medicine, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: alexander.ebinger@ucdenver.edu

S. Tomberg
Department of Emergency Medicine and Orthopedics, Denver Health Medical Center, Denver, CO, USA

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response [8]. Poorly treated pain may have adverse physiologic effects such as increased sympathetic drive, resulting in increased peripheral vascular tone, increased respiratory rate, and increased myocardial oxygen demand. Further, poor pain management during an initial event may increase the duration and need for pain control in subsequent events.

Pharmacologic Pain Control

There are numerous oral and parenteral medications available to achieve pain control. Nonsteroidal anti-inflammatories, acetaminophen, and opiates are common. This section provides a generalized overview of methods of pharmacologic pain control though it is not meant to be exhaustive nor comprehensive in the overview of each drug discussed.

Non-opiate Analgesia

Nonsteroidal anti-inflammatory drugs (NSAIDs) are effective analgesic and anti-inflammatory medications. Pain control is achieved through inhibition of the cyclooxygenase-2 (COX-2) enzyme, which diminishes pain response by the dorsal horn neurons. Nonselective nonsteroidal anti-inflammatories inhibit both the COX-2 enzyme and the COX-1 enzyme, found throughout the body. Nonselective NSAIDs include ibuprofen, naproxen, indomethacin, diclofenac, and meloxicam. Benefits of NSAID medications include lack of respiratory and CNS depression. This medication class can be used safely and concurrently with opioid medications and may reduce the amount of narcotic pain medications required to treat acute pain. Ketorolac is currently available for IM or IV use. Despite the ability to quickly administer this drug, there is no data to suggest a faster time to onset of the medication [9–11]. Risks of long-term NSAID usage include GI bleeding, renal impairment, platelet dysfunction, and allergic reaction [12]. NSAIDs must be used with caution in patients with baseline renal impairment, peptic ulcer disease, or baseline platelet dysfunction or increased bleeding risk. NSAIDs should not be routinely prescribed to patients who are taking oral anticoagulants, ACE inhibitors and diuretics, glucocorticoids, and methotrexate.

Another type of nonsteroidal prescription pain relievers is selective COX-2 inhibitors. As the name implies, these medications do not act on the COX-1 enzyme, thereby reducing the incidence of gastric complications. COX-2 inhibitors have not been demonstrated to have any significant pain relief advantage over nonselective NSAIDs. Currently, only celecoxib is available. Previously available drugs such as rofecoxib and valdecoxib have been pulled from the US market due to concerns regarding increased risk of cardiac death.

One common concern regarding NSAID usage is the question of whether it impedes fracture healing and increases the risk of non-union. Theoretically, NSAIDs may inhibit prostaglandin formation, which leads to inflammation, essential to bony healing [13]. To date there are no good randomized control studies that have studied this topic. NSAIDs are likely safe when administered in short courses following a fracture until further information can be gathered to answer this question [14].

For mild pain, acetaminophen can provide pain control in individuals without significant hepatic impairment. Unlike the NSAID class, acetaminophen does not have any antiplatelet effect and may be used cautiously in patients with renal impairment. Acetaminophen is well tolerated at therapeutic doses, and in the absence of conditions that overwhelm the glutathione pathway, hepatic toxicity is rare. Alcohol dependence and liver disease increase the risk of hepatotoxicity.

Cyclic antidepressants and anticonvulsants can be utilized to treat acute onset neuropathic pain. Medications include amitriptyline, carbamazepine, gabapentin, and pregabalin. These medications require close initial monitoring for side effects and often require dosing up-titration. These medications can be helpful in treating painful conditions such as sciatic nerve pain, post-herpetic neuralgia, and trigeminal neuralgia [15, 16].

Muscle relaxants such as cyclobenzaprine and diazepam have been used to treat musculoskeletal pain, mainly back pain. The degree of effect these medications have is approximately the same as ibuprofen though they present a different set of side effects, mainly CNS depression, drowsiness, and dizziness. Caution should be exercised when prescribing opiate pain medications in addition to these medications.

Topical medications can be a helpful treatment for mild to moderate localized pain. These typically have a lower systemic side effect profile than oral medications and fewer central acting complications. Medications such as topical lidocaine, diclofenac, and capsaicin are available for application directly at the site of pain. These medications may be a useful adjunct for patients who cannot otherwise tolerate oral versions of the medication class due to conditions such as baseline platelet dysfunction, anticoagulation, or peptic ulcer disease.

Opioid Analgesia

Opiates (referring to drugs that are structurally similar to the natural alkaloids found in opium) are commonly used to treat moderate to severe pain. The term opioid refers to any structure with similar biologic activity to an opiate. There are many types of opioid pain medications with different rates of onset, methods of administration, and side effects (see Table 5.1) Advantages to opioid medication administration include relatively rapid onset of pain relief, ability to titrate to effect, and the ability to concurrently treat pain with

Table 5.1 Opiate anesthesia and dosing

Drug	Opiate-naïve adult dose	Pharmacokinetics	Comments
Morphine	0.1 mg/kg IV 10 mg IM/SC 0.3 mg/kg PO	Onset: 1–2 min IV, 10–15 min PO Peak effect: 3–5 min IV, 15–30 min IM Duration 1–2 h IV, 3–4 h IM/SC	
Hydromorphone	0.01 mg/kg IV 1–2 mg IM	Onset: 3–5 min IV Peak effect: 7–10 min IV Duration: 2–4 h	
Fentanyl	1.0 mcg/kg IV 100 mcg intranasal	Onset: 1 min IV Peak effect 2–5 min IV Duration 30–60 min	
Oxycodone	5–10 mg PO	Onset: 15–30 min Duration: 3–6 h	Metabolized to oxymorphone
Hydrocodone	5–10 mg PO	Onset: 30–60 min Duration: 4–6 h	Metabolized to hydromorphone
Codeine	30–60 mg PO	Onset: 30–60 min Duration: 4–6 h	Metabolized to morphine
Tramadol	50–100 mg PO	Onset: 10–15 min Duration: 4–6 h	Synthetic, weak mu agonist Does not cause physiologic dependence Not a scheduled drug Side effects increasingly present with higher doses

Modified from Ref. [21]

non-opioid analgesics. Pitfalls to opioid medications include respiratory depression, hypotension, and, over time, medication tolerance, hyperalgesia, and addiction.

Opiate mechanism of action is modulated by binding to specific opiate receptors: delta, kappa, and mu. Additionally, nociception is a structurally similar hormone. Opiates bind to these receptors in variable fashion. The mu-1 receptor predominately produces supra-spinal analgesia, whereas the mu-2 receptor results in euphoria, respiratory depression, and miosis and reduces GI motility. The delta receptor produces a lesser degree of analgesia compared to the mu receptor. The kappa receptor predominantly produces dysphoria, along with dissociation and delirium. It also inhibits antidiuretic hormone release resulting in diuresis [17–20].

The most commonly used opioid pain medications include morphine, hydromorphone, and fentanyl. Morphine is a natural alkaloid, whereas hydromorphone is a semisynthetic alkaloid, and fentanyl is a synthetic piperidine. Meperidine was once commonly used for pain control but has fallen out of favor given the superior alternative options [22]. Codeine is a prodrug that is converted to morphine though patients who are CYP2D6 deficient or are rapid metabolizers do not have a strong analgesic effect. Further, there are many adverse effects of codeine and it has fallen out of favor as an agent for pain control. True opioid allergies are rare. Opioid receptors exist on mast cells and histamine is released in a dose-dependent fashion, causing pruritus, flushing, and dizziness, rather than being a true IG-E-mediated allergy. If there is a reaction to a particular opioid, switching opioid classes is advised. There is little data to suggest a cross-sensitivity between classes. In addition to the histamine response, the most common side effects of opiates are constipation, nausea, and vomiting. In increasing doses, CNS and respiratory depression are also potential deleterious consequences.

Opioids can be delivered in parenteral, oral, intramuscular, subcutaneous, intranasal, and transdermal fashion. IV opioids have a more readily predictable absorption than do IM injections, and repeated dosages are less painful. Individuals can vary greatly in their response to a single dose of opioid analgesia, dependent on factors including age, initial pain severity, and chronic exposure to opioids. Body mass and gender have no effect. To safely administer opioid pain medications, providers must consider renal and hepatic function, baseline respiratory insufficiency, age, and history of drug addiction or dependence. Most active opioid metabolites are renally excreted; thus patients with renal impairment are at risk of metabolite accumulation and increased risk of respiratory and CNS depression. Hydromorphone and fentanyl are preferred in renal failure patients, though in reduced initial doses. Similarly, elderly patients are at higher risk for opioid-related adverse events due to the higher probability of multi-organ dysfunction and drug-disease interaction. The starting dose for elderly patients is typically one-half that of younger patients, though frequent pain checks along with assessment of neurologic impairment and vital signs are required as this may not achieve adequate analgesia [17, 23].

Naloxone is available as a reversal agent for opioid toxicity and overdose. This is available in IV, IM, and intranasal (IN) forms. The half-life of naloxone is approximately 45 min, so care must be taken to monitor a patient after recovery to ensure they do not require further doses of naloxone.

In addition to pure opioid agonists, tramadol weakly binds to the mu receptor and also weakly inhibits norepinephrine and serotonin reuptake. In comparison to oxycodone and hydrocodone, it has a more favorable side effect

profile and may have less risk of addiction. When given with acetaminophen 325 mg, it has been shown to be as effective as hydrocodone and acetaminophen together [24].

Medications, such as buprenorphine, nalbuphine, and butorphanol, have an opioid agonist-antagonist effect and can be utilized for pain control with less risk of respiratory depression. These appear to have a ceiling effect of their efficacy, reducing utility, though they still have similar abuse rates to oxycodone and hydrocodone. These are not currently used as first-line pain medications.

There has been substantial discussion around the potential risks and benefits of opiate analgesia and the optimal timeframe for duration of treatment. There is no established consensus on this topic though the CDC has issued recommendations for opiate use for chronic pain [25]. This area of medicine is under great scrutiny and is currently a frequent news item. Current recommendations include limiting the number of medications prescribed with frequent rechecks to assess ongoing opiate need and utilization of multimodal analgesia with other non-opioid medications and methods of pain control such as local and regional anesthesia.

Peripheral Nerve Blocks and Hematoma Blocks

While systemic medications can provide adequate pain relief, there is a practicality to treating pain by targeting the nerves upstream of the site of the injury. Pain signals are modulated via peripheral nerves activated by sodium channels. Local anesthetics block these sodium channels, effectively intercepting the pain signal from the injury before it makes it to the spinal cord and central nervous system.

Local Anesthetics

There are a number of choices for sodium channel-blocking anesthetic. Lidocaine has an onset of action of 45–90 s and a duration of 15–60 min [26]. Bupivacaine has an onset of action of 2–10 min and the duration of action is 3–7 h. Local infiltration can provide pain control to the area being injected. Additionally, when injected around a nerve sheath, these medications can provide regional anesthesia distal to the site of infiltration. The addition of epinephrine to the anesthetic can increase the impact and duration of the anesthetic effect.

There are two classes of local anesthetics: amides and esters. Most allergies are to esters, as they are broken down to para-aminobenzoic acid. There are few allergic reactions to amides or their metabolites, and it is reasonable to switch classes if a patient has an allergy to one class or the other. In reality, most reported allergies to local anesthetics are due to preservatives in multi-use bottles and not to the anesthetic itself. Therefore, the use of single-use bottles without preservatives will usually remove the hypersensitivity.

Table 5.2 Local anesthetic dosing and characteristics

Anesthetic	Maximum dose (mg/kg)	Maximum dose with epinephrine (mg/kg)	Onset of action	Duration of action	Class
Lidocaine	4	7	45–90 s	15–60 min	Amide
Prilocaine	6	9	2 min	30–90 min	Amide
Bupivacaine	2	2.5	2–10 min	180–420 min	Amide
Ropivacaine	3	4	5 min	120–360 min	Amide

Modified from Refs. [27–32]

Esters are rapidly broken down and have a shorter half-life than amides. This quick breakdown makes esters safer to give to pregnant patients because most of the drug is metabolized before it reaches fetal circulation. Amides, on the other hand, have a longer half-lives and cross into fetal circulation in greater amounts. While this is usually a negligible amount for procedures like spinal anesthesia or a small laceration repair, it can be significant if large amounts are used for large laceration repairs (Table 5.2).

Anesthetic dosing is variable, based on the medication available and its concentration, as well as the size, number, and accessibility of the targeted nerve. For instance, it is reasonable to inject 5 mL of 1% lidocaine without epinephrine (10 mg per mL) for an ulnar nerve block as the total dose of anesthetic is only 50 mg (assuming the patient weighs at least 17 kg). However, if one plans to anesthetize the femoral nerve, it may take up to 20–30 mL of injectate to adequately bathe the nerve fully. Thus, one must understand the maximum dosing of the medication planned for use and dilute it appropriately to achieve the volume required for each nerve suggested below. For example, if you have a 50 kg patient and only 2% lidocaine without epinephrine (20 mg/mL), you can only use 10 mL of 2% lidocaine without epinephrine (50 kg∗4 mg/kg = 200 mg/[20 mg/ml] = 10 mL). Thus, to block the femoral nerve without exceeding the maximum dose of lidocaine, one needs to plan to add 10–20 mL of normal saline for the procedure. If the patient weighs 100 kg and 1% lidocaine with epinephrine is available; to block the femoral nerve, you can use up to (100 kg∗7 mg/kg = 700 mg/[10 mg/mL] = 70 mL) before reaching the maximum dose, thus not having to dilute the injectate at all.

The same calculations can be performed with any of the previously mentioned anesthetics by knowing the maximum dosing (understanding how adding epinephrine changes this) and the concentration of the medication, with a target injectate volume as mentioned in the forthcoming descriptions.

Local anesthesia is generally safe, particularly in small doses, but practitioners need to be aware of the signs of local anesthetic systemic toxicity (LAST) and the treatment of any toxic effects. The central nervous system (CNS) is the first system to show the effects of local anesthetic toxicity. Patients can develop muscle twitching and tremors and other symptoms that are excitatory in nature. They may start to feel dizziness, lightheadedness, and circumoral paresthesia. The toxicity can progress to visual and auditory hallucinations and disorientation. In cases of severe toxicity, patients will develop tonic-clonic seizures, CNS depression, and coma. Respiratory compromise can also occur, and the resulting acidosis and hypercarbia can exacerbate the CNS symptoms.

The cardiovascular system can also be affected in LAST, but these symptoms typically present after CNS symptoms. (This is less true with highly lipophilic drugs like bupivacaine, where CNS and cardiac toxicity are more likely to present simultaneously.) Local anesthetics work on both peripheral blood vessels and the myocardium. If concentrations are high enough, the local anesthetics can block spontaneous sodium channel pacemaker activity at the SA node leading to sinus bradycardia and cardiac arrest.

In the case of systemic toxicity, it is essential to recognize the signs and symptoms early. The initial focus for the treatment of LAST should be placed on managing Airway, Breathing, and Circulation (ABCs). Acidosis and hypoxia amplify the effects of the toxidrome; therefore, the airway should be aggressively managed. Patients with hemodynamic instability may require vasopressor support; therefore, when evaluating "C's," it is important to ensure that the patient has adequate vascular access.

LAST can also cause seizures. These seizures can worsen acidosis, which amplifies the toxicity of the anesthetic. Therefore, seizure suppression is paramount. The first-line medications for seizure suppression are benzodiazepines. In one study propofol was shown to attenuate some of the cardiotoxic effects of bupivacaine; however, there is concern that propofol can cause myocardial depression which could amplify the effects of LAST on the heart. Given these concerns, recommendations are that propofol can be used in small doses for seizure suppression if benzodiazepines are not effective [33]. Finally, neuromuscular blockade with succinylcholine will not stop seizure activity, but it can be considered to address the metabolic acidosis that is generated from muscle contraction during tonic-clonic seizure activity.

If there is a high suspicion of LAST, intralipid should be given early. Intralipid works both as a sink to lower toxic free anesthetic and it helps to correct acidosis. Intralipid has been shown to increase survival in laboratory studies and has been used successfully in practice to treat anesthetic toxicity.

If there continues to be considerable signs of toxicity after these treatments, cardiopulmonary bypass is an option for rescue management [34].

Regional/Peripheral Nerve Blocks

To effectively accomplish regional nerve blocks, a provider needs be skilled in identifying the nerve bundles that are carrying pain signals and accurate in delivering targeted anesthetic medication. Nerve blocks are powerful tools for pain management.

Upper Extremity Anesthesia: Interscalene, Radial, Median, and Ulnar Nerve Blocks

Interscalene Nerve Block

The interscalene block is a great tool for pain control of a dislocated shoulder or proximal humerus fracture. The trunks of the brachial plexus can be located in the interscalene groove, between the anterior and middle scalene muscles, lateral to the sternocleidomastoid muscle. Blockade of these nerves provides anesthesia from the shoulder to the elbow. The block can often spare the forearm and hand, particularly the C8 and T1 distributions [35]. The trunks are identified on ultrasound in the lateral and inferior neck and typically are stacked on top of each other, which gives them the appearance of a "traffic light." While this is the classic appearance, there are many anatomic variations; 61% of people have left/right anatomic variation and some roots will travel within the anterior scalene muscle.

Initially the trunks of the brachial plexus are identified in the lateral neck using the linear ultrasound probe. The approximate location where the needle will be introduced is marked on the skin and the site of the procedure cleaned and a sterile cover is placed on the ultrasound probe. A sterile ultrasound gel is placed on the probe or shoulder/neck and the nerve roots are again identified. Next, a sterile needle is introduced from the lateral direction and is advanced toward the nerve roots under direct visualization. The needle is advanced until it is adjacent to the nerve roots. Small amounts of anesthetic (up to 15–20 mL in 5–10 mL aliquots) are injected adjacent to the nerve bundle. This can be visualized as hypoechoic fluid and it will start to separate the nerve from the surrounding tissue. The goal is to bathe the nerve in anesthetic creating a hypoechoic "doughnut" around the nerve when viewed on ultrasound, and anesthetic should not be injected directly into the nerve itself. If the needle happens to hit the nerve fascicles, there is less chance for damage if the bevel of the needle is placed in parallel with the directions of the fascicles; therefore, the needle should be placed with the bevel running in a cephalad-caudad orientation. Anesthetic is intermittently injected around each of the three nerve roots, with a goal of surrounding each nerve root with hypoechoic anesthetic fluid.

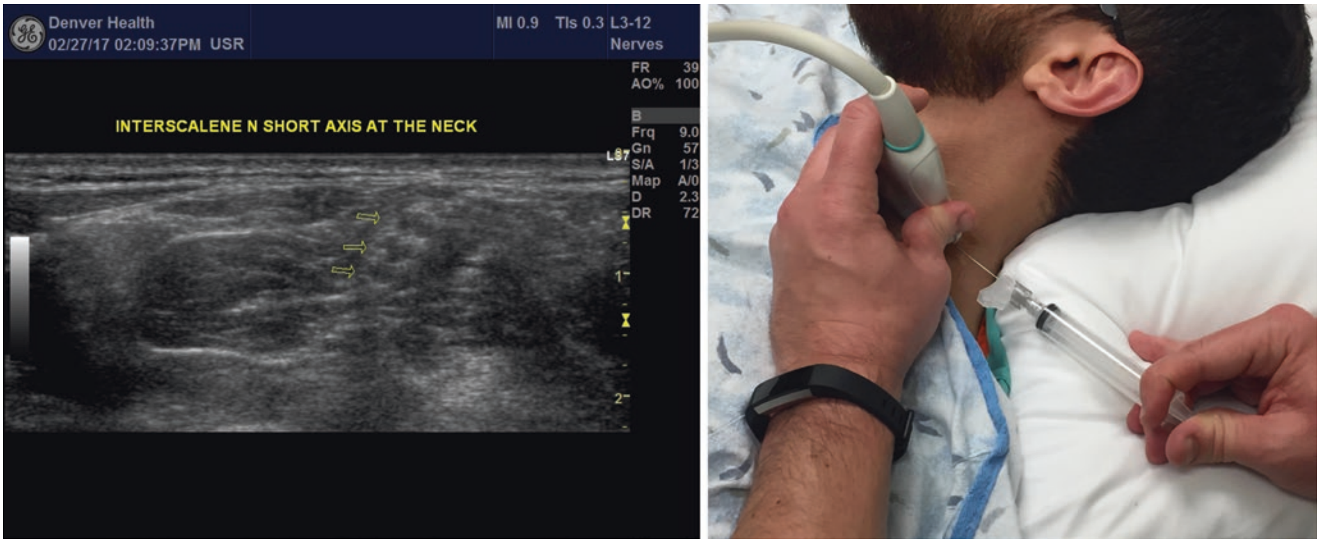

Fig. 5.1 Distribution of interscalene block anesthesia. Appearance of the interscalene nerve on ultrasound. Appropriate provider positioning for performing interscalene nerve block

When performing an interscalene block, it is important to recognize that the phrenic nerve commonly is located superior and medial to the anterior scalene muscle (Fig. 5.1). A lateral approach allows the needle to reach the plexus through the middle scalene muscle while skirting under the phrenic nerve. If the phrenic nerve is anesthetized, it can result in paralysis of the hemidiaphragm. It is likely that a small amount of anesthetic from an interscalene block will “leak” around the phrenic nerve, so this particular block should be avoided in patients with borderline pulmonary function.

Other risks associated with the interscalene block are pneumothorax, anesthesia of the superior cervical chain, or the recurrent laryngeal nerve, which can cause transient Horner’s syndrome or a hoarse voice, respectively, and long-term nerve injury. Symptoms of a pneumothorax will typically present 6–12 h after the procedure. Discharge instructions must be clear that patients should return to an emergency department or call 911 if they start to develop shortness of breath. The rate of long-term injury to nerves is between 0.02% and 0.4%. These injuries typically occur early because of intraneural edema, extra- or intra-neural hematoma, or direct damage to multiple axons. Occasionally patients will present with neuropathy weeks after the nerve block, and this is thought to be secondary either to scarring or possibly to prolonged immobility [36].

Overall, studies using ultrasound guidance for nerve blocks have demonstrated that anesthesia success rates are higher than anatomical landmark techniques and similar to techniques that use nerve stimulators, and complications are lower [37–39]. Given the lack of sideline access to nerve stimulators, and increasing access to point-of-care ultrasound, ultrasound-guided interscalene blocks can be an option for pain control of a dislocated shoulder or proximal humerus fracture that can be performed on the sideline or in a training room.

Forearm Anesthesia: Radial, Median, and Ulnar Blocks

Sideline providers can also target specific nerve distributions of the radial, ulnar, and median nerves for forearm injuries, including fractures, dislocations, as well as soft tissue injuries such as lacerations.

Radial Nerve

The radial nerve splits into sensory and motor branches at the elbow, so complete anesthesia is best achieved when the nerve is anesthetized above the elbow. The nerve is visualized using a linear ultrasound transducer in the transverse plane 3–4 cm proximal to the elbow crease on the lateral aspect of the upper arm or in the radial groove of the humerus. Anesthesia of the radial nerve at this position will cover the radial aspect of the dorsum of the hand and the radial aspect of the dorsum of the forearm [40]. The radial nerve can also be localized in the forearm by finding the distal radial artery and tracing it proximally until the nerve bundle becomes apparent on the radial side of the artery. For a more distal sensory block, a “line” of anesthetic can be drawn across the dorsum of the wrist starting just proximal to the radial styloid and extending toward the ulnar styloid. 5–10 mL of anesthetic should be instilled to bathe the nerve (Fig. 5.2).

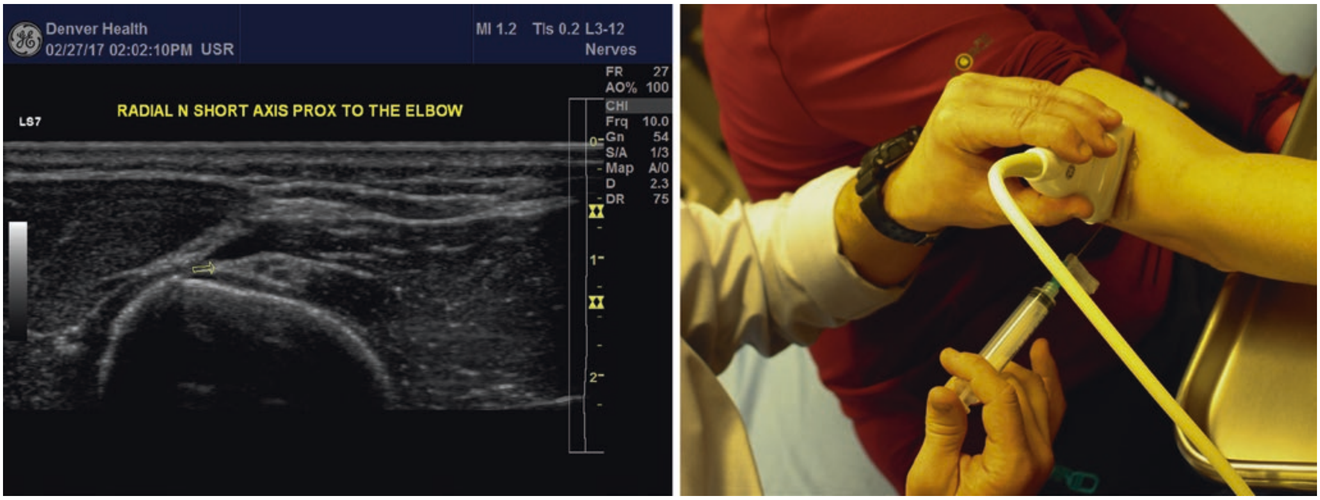

Fig. 5.2 Distribution of radial nerve block anesthesia. Appearance of the radial nerve on ultrasound. Appropriate provider positioning for performing radial nerve block

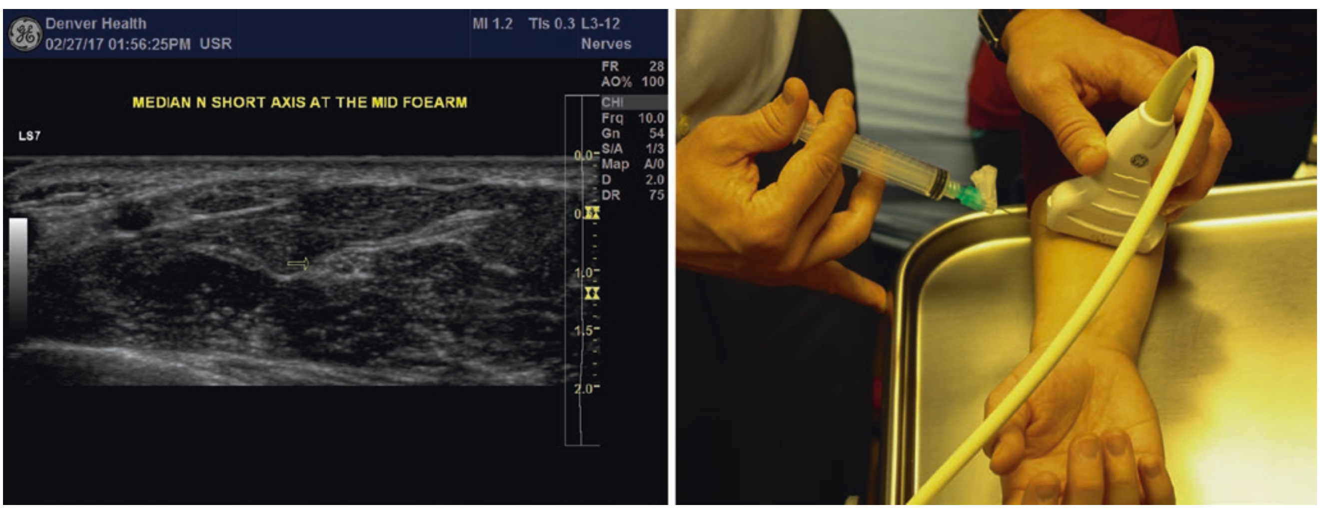

Fig. 5.3 Distribution of median nerve block anesthesia. Appearance of the median nerve on ultrasound. Appropriate provider positioning for performing median nerve block

Median Nerve

The median nerve can be visualized in the midline of the mid-forearm. The nerve can also be located at the wrist, but at this level it runs between the flexor digitorum profundus and superficialis. This makes it more challenging to differentiate the nerve bundle from surrounding tendons at the wrist; therefore, the mid-forearm is the preferred location for a block. Anesthesia of the median nerve will cover the radial aspect of the palm of the hand and can be useful for large lacerations [41]. 5–10 mL of anesthetic should be instilled to bathe the nerve (Fig. 5.3).

Ulnar Nerve

The ulnar nerve can be localized in the ulnar aspect of the distal forearm. The nerve is on the ulnar side of both the ulna and the ulnar artery. It can be found at the wrist or traced proximally up the forearm. At a more proximal location, the nerve and artery tend to separate, which lowers the risk of intra-arterial injection [42]. The nerve and the artery run in close proximity to each other and the nerve should be approached from the ulnar side to best avoid arterial injury [41]. 5–10 mL of anesthetic should be instilled to bathe the nerve (Fig. 5.4).

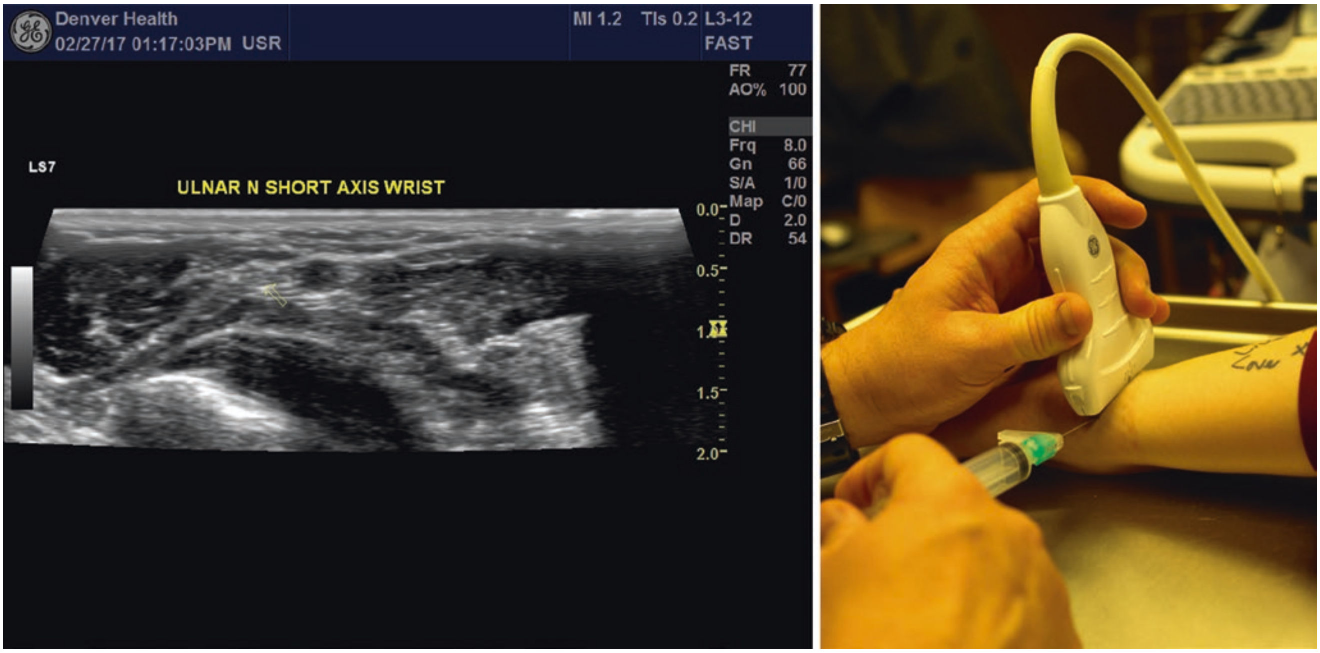

Fig. 5.4 Distribution of ulnar nerve block anesthesia. Appearance of the ulnar nerve on ultrasound. Appropriate provider positioning for performing ulnar nerve block

A study looking at patients who present to the emergency department with injuries amenable to nerve block of the forearm showed that it took an average of 9 min to complete each patient's nerve blocks and each patient required two blocks to achieve adequate pain control [41, 43]. This makes forearm blocks practical for the emergent and sideline treatment of distal forearm and hand injuries.

Facial Injuries

Ear, Supraorbital, Infraorbital, and Mental Nerve Blocks

Sideline providers should be able to treat most lacerations to the face, and there are a variety of nerve blocks that can be used to replace or complement direct injection of anesthetic into lacerations.

Ear Anesthesia

For trauma to the pinna of the ear, a field block can be used for anesthesia. By placing anesthetic in a diamond-shaped pattern (2–3 mL fluid in each direction) around the ear, complete anesthesia of the pinna can be obtained. The facial artery runs anterior to the ear and care should be taken to not inject directly into the artery (Fig. 5.5).

Nerve blocks are useful for repairing facial and lip lacerations, especially when the vermilion border is involved because they do not distort the anatomy while they provide excellent pain control.

Supraorbital and Infraorbital Nerve

For lacerations of the forehead and scalp, a supraorbital nerve block can be utilized. The supraorbital nerve is a branch of cranial nerve V and supplies innervation to the forehead and scalp, to the level of the lamboid suture. The supraorbital nerve exits the skull at the supraorbital foramen. To locate the foramen, a line is drawn to the inferior supraorbital ridge from the mid-pupil as the patient looks straight ahead. The inferior ridge is then palpated for a small notch, which is the exit point of the nerve. To anesthetize this nerve, local anesthetic is injected just superior to the ridge. If the patient is experiencing severe pain or paresthesias, the needle must be retracted 1–2 mm. Then, 2–3 cc of anesthetic can be deposited into the soft tissue.

For lacerations of the upper lip, an infraorbital block can be placed. The infraorbital nerve exits the skull from the infraorbital foramen. The approximate location of the foramen can be found by palpating the infraorbital rim (bony rim inferior to the orbit) to locate the small notch in the bone formed by the infraorbital suture. The foramen is approximately 2 cm below this notch. Drawing a line from the mid pupillary line, through the notch, and to the lateral aspect of the lip margin will give an approximation of the medial/lateral location of the foramen.

Anesthetic can be placed in a small bolus inferior to the foramen by either direct injection through the skin or by

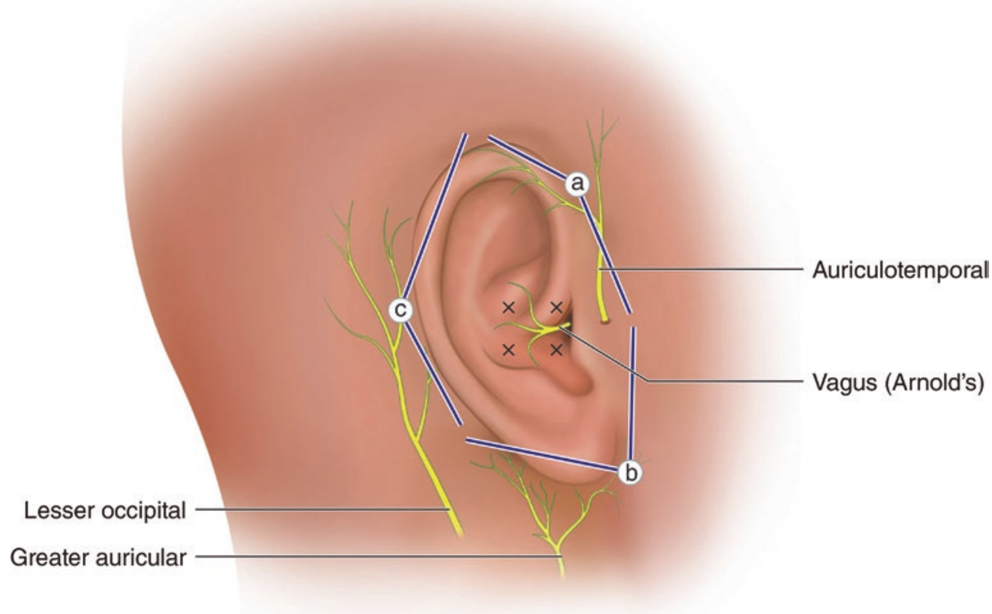

Fig. 5.5 Ring block for ear anesthesia

advancing a needle vertically from inside the mouth to a location just inferior to the foramen. The advantage of the intraoral approach is that it provides protection from injecting into the foramen itself and has been shown to be less painful.

For the intraoral approach, the needle should enter the upper gum line directly above the canine. The needle is then advanced to a location 1 cm below the foramen. This location is reached when the needle is advanced 2.5 cm deep into the gum. Care must be taken to direct the needle parallel to the long axis of the canine, if the needle is placed posterior and deep it can enter the orbit. If the provider is not sure of the location of the needle tip, they should stop the procedure.

When the needle tip is in the correct location, 2–3 mL of local anesthetic is injected to create a field block around the nerve. The intraoral and percutaneous approaches have been shown to be equivalent for both pain control and ability to achieve anesthesia (with a trend toward the intraoral approach being more effective) [44]. In either case the anesthetic should not be directly injected into the foramen; anesthesia is obtained from a field block when the anesthetic is placed inferior to the foramen. For this block use anesthetic without epinephrine, as vasoconstriction of the adjacent facial artery can cause local ischemia (Fig. 5.6).

Mental Nerve

For lacerations to the chin and lower lip, a mental block can be performed. The mental foramen is located on the lateral aspect of the inferior chin. If the foramen can be palpated, anesthetic can be injected superior to the foramen. The other option is an intraoral approach. To do this the needle is placed into the gum line just lateral/posterior to the canine and then is advanced to a position just superior to the foramen [45]. A small study demonstrated less pain and more success with achieving anesthesia when the intraoral approach is used [46]. As above, anesthetic should not be directly injected into the foramen; anesthesia is obtained from a field block by placing anesthetic superior to the foramen.

Lower Extremity Anesthesia: Fascia Iliaca, 3-in-1/Femoral, Sciatic, and Posterior Tibial Blocks

There are two main nerve blocks, the fascia iliaca and 3-in-1/femoral nerve block, that can be used to treat severe traumatic pain in the hip, including pain from femoral fractures, hip dislocations, and other soft tissue injuries. These blocks can also be used to treat pain from soft tissue injuries; however, if there is a concern for potential development of compartment syndrome from a soft tissue injury, these blocks are contraindicated. These blocks, if used appropriately, seem to confer similar pain control [47]. Ultrasound guidance improves the likelihood of success.

Femur fractures can be devastating injuries and they can present a pain management conundrum because of their frequent association with hypovolemic shock. Opiates can be suboptimal options for pain control if the patient is hypotensive or has altered mentation. Nerve blocks can be used to treat pain while preserving patient's sometimes precarious hemodynamic status.

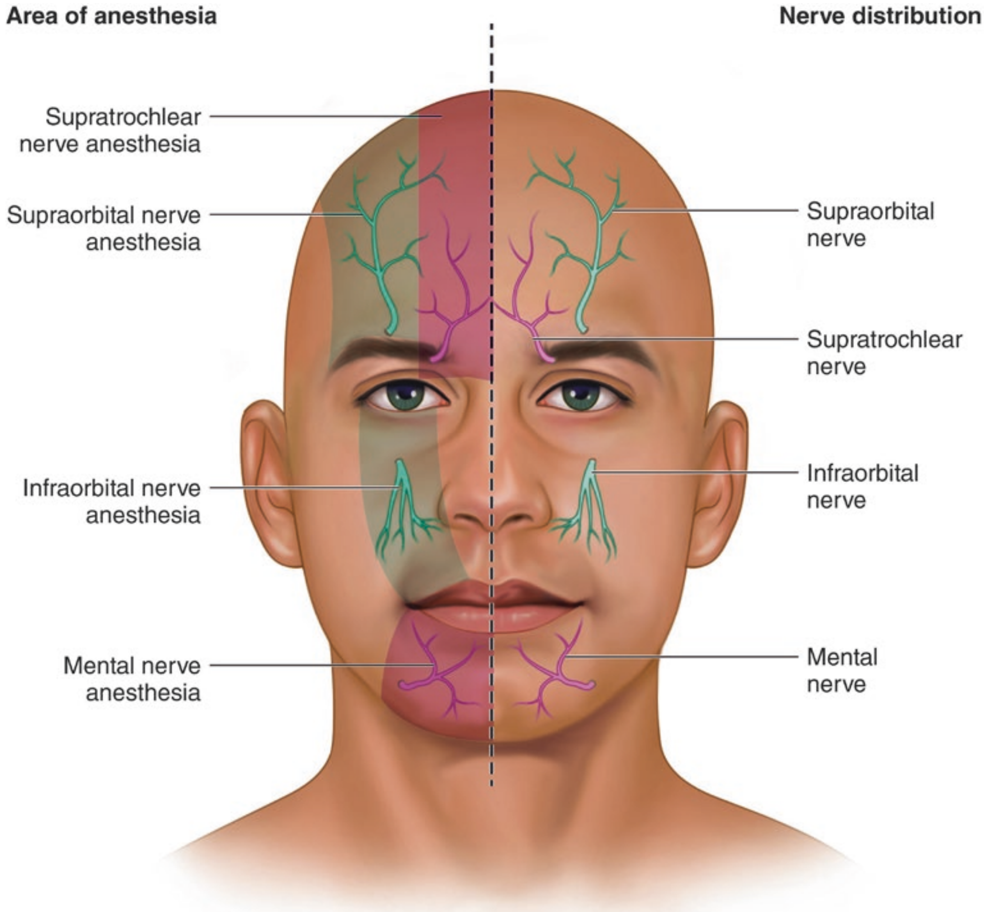

Fig. 5.6 Supraorbital, supratrochlear, infraorbital, and mental nerve location and anesthesia

Fascia Iliaca Block

The fascia iliaca block will block the obturator, femoral, and lateral cutaneous nerves. This block can be accomplished using anatomical landmarks or ultrasound guidance.

To begin, a linear probe is placed perpendicular to the inguinal ligament and will point toward the umbilicus. The middle of the probe should be at the confluence of the lateral and middle one-thirds of the inguinal ligament, and this is lateral to the femoral artery and nerve. The fascia iliaca will be a hyperechoic linear structure that lies over the iliacus muscle and the sartorius muscle lies lateral and superior to the facial iliacus. Under direct guidance a 22-gauge needle is advanced through the sartorius in plane with the ultrasound probe to the fascia in a caudad to cephalad direction. The fascia will tent and then give way as it is pierced, aspirate to ensure that the needle is not in a vascular structure. Next, 1–3 mL of anesthetic is injected and should track medially and separate the fascia from the iliac muscle. If the hypoechoic fluid spreads superior to the fascia, the needle should be advanced. If the hypoechoic fluid spreads within the iliacus muscle, the needle should be pulled back. When the proper location is confirmed, 20–30 mL of dilute anesthetic is injected [48].

The ultrasound-guided technique has been shown to be more effective at providing complete anesthesia [49].

A meta-analysis of the fascia iliaca blocks showed similar pain control to parenteral NSAIDs and opiates, with the nerve block providing a longer duration of action. These studies were done for proximal femur fractures. Systematic reviews of the fascia iliaca block show that the blocks are effective at reducing pain and can reduce opiate use in patients with hip and femoral neck fractures [50].

3-in-1/Femoral Nerve Block: Ultrasound Guided (Fig. 5.7)

The alternative method is to anesthetize the femoral nerve under direct ultrasound visualization. This is called a 3-in-1 block because it accomplishes anesthesia of the femoral, obturator, and lateral cutaneous nerves.

The 3-in-1 block has been shown to give patients fast resolution of pain and multiple studies have shown that the procedure reduces the total amount of opiates that are required for pain control [51]. While the 3-in-1 block was originally used for femoral neck fractures, it has also been shown to

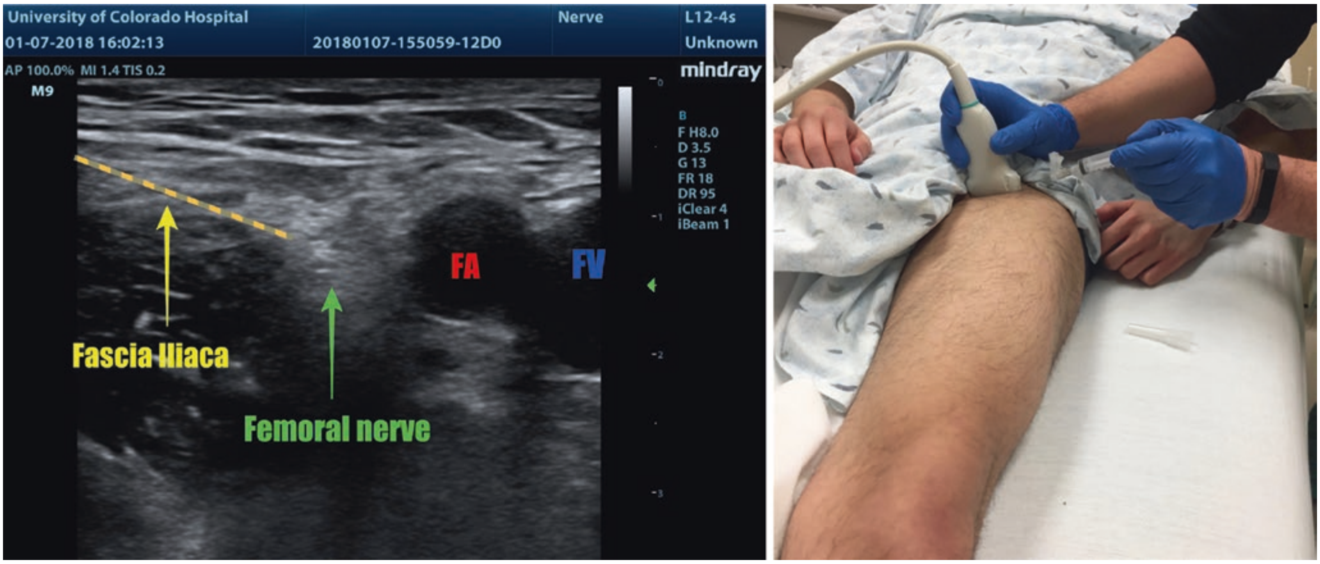

Fig. 5.7 Distribution of fascia iliaca, femoral nerve, femoral artery (FA) and femoral vein (FV). Appearance on ultrasound. Appropriate provider positioning for performing fascia iliaca block

be effective for pain control for knee surgeries and for distal femoral shaft fractures.

The major concern that has been about using nerve blocks for pain control is that the block can mask the discomfort that is typically used to monitor for compartment syndrome. Discussion should be had with the orthopedic consultant prior to performing this block.

To perform this block, position the patient supine with slight abduction of the patient's legs. Prep the groin in a sterile fashion and sterile drape the region. Sterile gloves should be worn and a sterile ultrasound cover used. The ultrasound is placed on the patient's groin with the indicator pointing to the patient's right. The probe should be slid cephalad and caudad until the following structures are identified. First, identify the inguinal ligament – a hyperechoic shallow structure. Once the inguinal ligament is identified, the probe should be slid caudad to identify the femoral vein and artery. The femoral vein is typically the most medial vascular structure and it is compressible, while the femoral artery generally lies lateral to the femoral vein and is non-compressible and is pulsatile. The femoral nerve sits lateral to the major vascular structures and is a hyperechoic triangle-shaped structure. The injection is made with a 21- or 22-gauge spine needle, advanced from the lateral side at a 30-degree angle. The needle should be advanced in plane with the ultrasound transducer so that the needle tip can be visualized as it approaches the nerve bundle. When the needle tip is adjacent to the nerve bundle (not in the nerve bundle), the provider should aspirate prior to injection to ensure that the needle is not within a vascular structure. After aspiration, 20–30 ml is injected, and this will appear as an expanding hypoechoic structure. Placing pressure 3-4 cm distal to the injection site will "push" anesthetic proximally, which will cause greater anesthesia of the obturator and lateral cutaneous nerves. The goal is to surround the nerve sheath with anesthetic; therefore, small adjustments to the needle may be necessary if the anesthetic is not tracking completely around the nerve [52, 53].

Sciatic Nerve Block/Popliteal Nerve Block

A popliteal/sciatic nerve block will provide anesthesia to the distal tibia, fibula, and the majority of the foot and is useful for soft tissue injuries, lacerations, and fractures. The sciatic nerve divides into the common peroneal and tibial nerve, variably, but typically approximately 5–12 cm proximal to the popliteal crease. When combined with a femoral block, complete anesthesia below the knee can be achieved.

To begin, the patient is positioned supine with the knee in 40–60 degrees of flexion and is supported with blankets under the proximal thigh. Prep the popliteal fossa and lateral knee in a sterile fashion and place a sterile drape around the region. Sterile gloves should be worn and a sterile ultrasound cover is used. The proceduralist positions themselves on the lateral aspect of the knee and holds a linear transducer in the popliteal fossa. First identify the popliteal artery and vein. Next, find the tibial nerve which lies superficial and lateral to the popliteal artery. Once the tibial nerve is identified, trace it proximally to where it is joined by the common peroneal nerve. This is the distal end of the sciatic nerve. Insert a 22-gauge needle toward the nerve using an in-line approach starting from the lateral side of the leg. Use the depth of the nerve on the screen as a measure of how deep

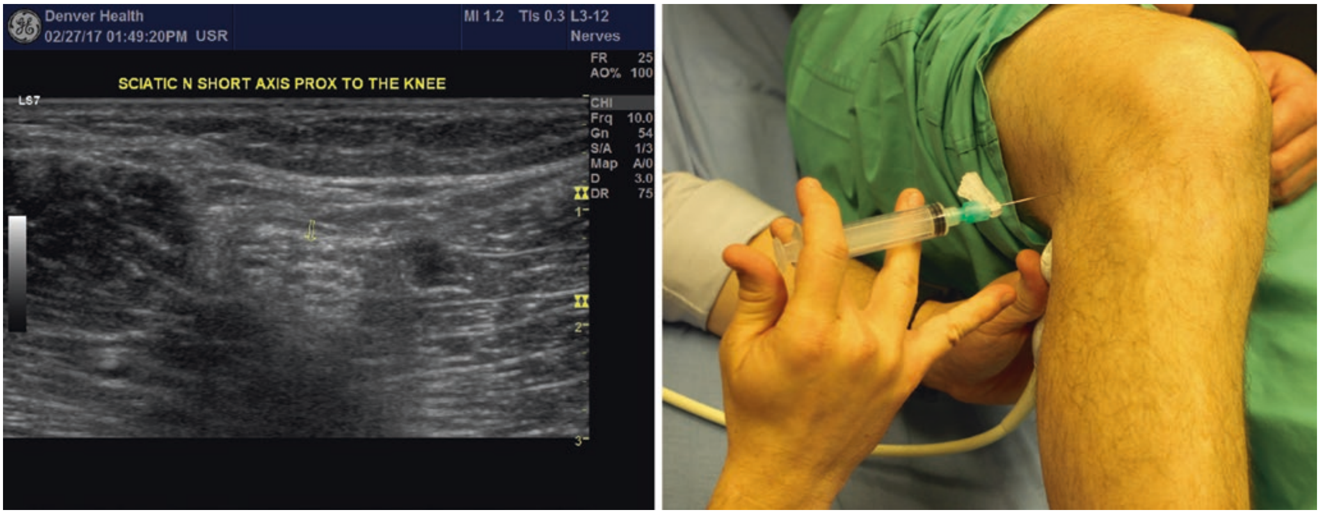

Fig. 5.8 Distribution of sciatic nerve block anesthesia. Appearance of the sciatic nerve on ultrasound. Appropriate provider positioning for performing sciatic nerve block

the needle should be inserted into the lateral posterior knee. The needle should be visualized for the entire procedure. When the needle tip is adjacent to the nerve bundle (not in the nerve bundle), the provider should aspirate prior to injection to ensure that the needle is not within a vascular structure. A total of 10–20 mL of anesthetic is injected. This is done in aliquots of 3–5 mL in regions around the nerve to bathe the entire nerve in anesthetic creating a “doughnut” of anesthesia [53] (Fig. 5.8).

Posterior Tibial Block [54]

Injuries and laceration to the sole of the foot are difficult to anesthetize with local injection of anesthetic. An ultrasound-guided posterior tibial nerve block will provide anesthesia to the skin of the sole of the foot and to the deep structures within the foot, including the calcaneus.

The posterior tibial nerve lies posterior to the medial malleolus and is usually posterior to the tibial artery.

To begin, the patient can be positioned with the knee in flexion and the hip externally rotated, so that the injured foot rests on top of the other leg. The patient can also be positioned in the lateral decubitus position with the injured extremity down and the knee flexed so the medial malleolus is presented facing up. The site is prepped in a sterile fashion and the ultrasound probe can be either covered with a sterile adhesive dressing or a sterile probe cover. The ultrasound probe should be placed in an anterior-posterior orientation just posterior and proximal to the medial malleolus. Next, the posterior tibial nerve is the hyperechoic structure adjacent to the hypoechoic/pulsatile tibial artery. The probe can be rotated to get a cross-sectional view of the nerve. A 27-gauge needle is used for the procedure. The needle can be advanced in-plane from the lateral side (which may be challenging given the length of the probe relative to the anatomy of the ankle) or may be advanced out-of-plane (coming in perpendicular to the probe). For an in-plane approach, the needle can be advanced from the medial or lateral side. The needle can be directly visualized as it approaches the nerve bundle. For an out-of-plane approach, the needle should be advanced at a steep angle (60–90 degrees) and tissue deformation can be monitored to locate the trajectory of the needle tip. For either approach the needle tip should be adjacent to the nerve sheath, not within in the nerve fascicle. Prior to injection aspiration should be performed to ensure that the needle tip is not in a vascular structure. Inject 5–10 mL of anesthetic under ultrasound guidance. The anesthetic should surround the nerve sheath. Adjustment of needle position or adjusting the bevel orientation of the needle may be needed to place anesthetic around the entire nerve [54] (Fig. 5.9).

The ankle joint has multiple innervations and the posterior tibial block will not be sufficient for ankle dislocation or fracture reductions (for complete ankle anesthesia, popliteal sciatic and saphenous blocks are needed).

Hematoma Blocks

At the site of a displaced fracture, a hematoma typically forms between the fracture fragments. This hematoma presents an opportunity to deliver anesthetic to the fracture site. This can provide significant pain control for fracture reductions.

Hematoma blocks have been shown to be equivalent to procedural sedation in both children and adults, and hematoma blocks are recommended first-line analgesia in forearm fractures in children [55]. The Cochrane review on the effec-

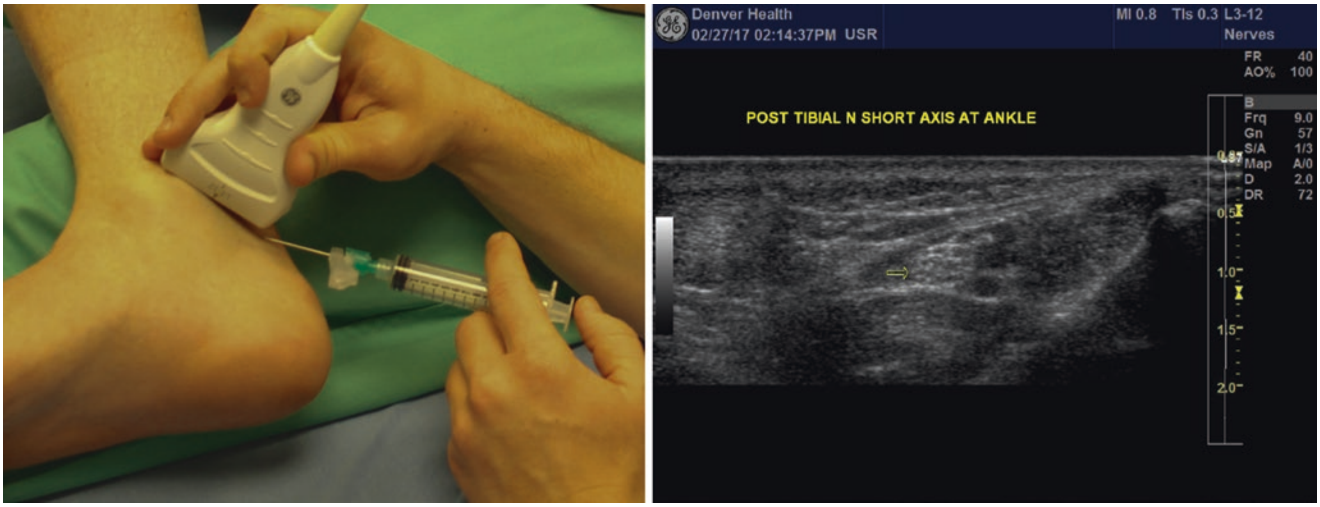

Fig. 5.9 Distribution of posterior tibial nerve block anesthesia. Appearance of the posterior tibial nerve on ultrasound. Appropriate provider positioning for performing posterior tibial nerve block

tiveness of analgesia for forearm fractures did not show any clear benefit of Bier block compared to hematoma block in adult patients for pain control, but the hematoma block was less resource intensive, and there was a trend toward more successful reductions in the Bier block group [55].

The majority of the neurovascular structures in the forearm are on the volar surface; therefore, the hematoma block should be attempted on the dorsal or lateral side. On the thigh the major neurovascular structures are located medially and proximal. In the mid to distal thigh and in the calf, these important neurovascular structures are posterior, which make either an anterior or lateral approach to the hematoma block most reasonable for the calf or distal thigh.

To perform a hematoma block, the first step is to locate the fracture. In some cases the fracture line may be visualized by the obvious deformity and may be easily palpated. In other cases (e.g., comminuted and impacted fracture with little displacement), ultrasound can be used to locate the fracture. To do this, the linear transducer is placed longitudinally along the bone and it is moved proximally and distally until the fracture is noted by an irregularity of the cortex. Once the fracture is located, the fracture site can be marked for injection. The fracture site is then prepped in a sterile fashion.

The hematoma block is performed with a 20–22-gauge needle. A needle is advanced into the fracture, and it should progress/“slide” into the fracture with little resistance. If the needle is within the hematoma at the fracture site, blood should be aspirated. Using a sterile technique, a needle can be introduced into the fracture location under direct ultrasound guidance. Once the correct location is confirmed, 10 mL can be injected into the fracture site [56].

Distal radius fractures are often accompanied by concurrent ulnar styloid fractures. The ulnar styloid fracture can also be anesthetized under direct visualization with ultrasound. This allows for more complete pain control during fracture reduction [57].

Intra-articular Shoulder Injection

There have been multiple studies investigating lidocaine and/or opiate injection directly into the glenohumeral joint for shoulder dislocations. These studies have generally shown that intra-articular lidocaine was superior to procedural sedation in the rate of complications and length of emergency department stay. There are no differences in success of reductions or patient satisfaction between the treatment modalities. The procedural sedation groups were shown to experience higher rates of respiratory depression, vomiting, and thrombophlebitis [58, 59]. There are two studies that contradict these findings, one had a rate of successful reduction that was a significantly lower than other studies and the other study compared intra-articular injections to demerol/diazepam, a drug combination that is not frequently used in emergency departments in the United States (this study also used traction/counter-traction as a reduction method and it could be argued that this particular method of shoulder reduction is better suited for procedural sedation). When all of these studies are combined, there are reasonable recommendations that intra-articular lidocaine injection is a reasonable first step for pain control for shoulder reductions prior to advancing to procedural sedation [60, 61]. Studies looking at postoperative pain control have shown that intra-articular opiates can significantly improve postoperative pain and that there are likely mu receptors in the joint space that can be targeted with opiates [62, 63]. Given this information, one may consider adding an intra-articular opiate to the injectate.

The steps for intra-articular lidocaine injection for shoulder reduction begin with locating the point 2 cm distal to the mid-acromion on the lateral aspect of the dislocated shoulder. This point can be marked with suction from an empty syringe or pressure from a fingernail. The site of the procedure is prepared in a sterile fashion. The provider puts on a mask and sterile gloves. A sterile needle is inserted into the shoulder at the point previously marked and directed toward the midline. Prior to injection, confirmation that the needle is in the joint space is accomplished when aspiration reveals a dilute bloody fluid, as trauma from the dislocation creates a diffuse hemarthrosis. Finally, 10–20 mL of anesthetic is injected into the joint space.

Procedural Sedation

On occasion, situations will arise where the injury incurred requires additional pain control beyond oral, parental, or regional anesthesia. Procedural sedation is the process of administering medications to a patient with the intent to depress their level of consciousness and control their pain in order to facilitate the performance of a painful procedure with little or no memory of the event while keeping the cardiac and respiratory systems fully functioning. Patients receiving procedural sedation should not routinely require airway protection or manipulation, in distinction from general anesthesia [64].

There are multiple levels of procedural sedation based on the patient's responsiveness, airway, breathing, and circulation. These are not defined by the drug, rather the patient's response to the drug, and therefore can be considered a continuum. Patients respond variably to medications and any practitioner providing procedural sedation must understand and be prepared to deal with all levels of sedation.

Minimal sedation is categorized as anxiolysis with normal, though sometimes delayed, response to verbal stimuli. Cardiovascular and respiratory function are typically unaffected and the risk of hypotension and hypoxia are very low [65]. Procedures commonly benefiting from minimal sedation include abscess drainage, lumbar punctures, and laceration repairs.

Moderate sedation is characterized by a depressed consciousness where the patient is still responsive to commands either verbally or with physical stimulation. Patients typically maintain their respiratory drive, though they occasionally require respiratory assistance due to hypoxia or hypoventilation [65]. Procedures commonly benefiting from moderate sedation include dislocation reduction, simple fracture reduction, chest tube placement, and electrical cardioversion.

Deep sedation is characterized by a depressed level of consciousness not responsive to verbal stimuli and with a motor response elicited by repeated or painful physical stimulation. Certainly, deep sedation poses the highest risk of cardiorespiratory compromise and may require interventions to combat hypoxia or hypoventilation [65]. Procedures requiring deep sedation include fracture-dislocation reduction and burn management.

Evaluating Patient Risk and Safety

Procedural sedation is a potentially high-risk endeavor, hinging on the provider's capacity, experience, and understanding of procedural sedation and its hypothetical complications, as well as the patient's baseline level of health and risk. In deciding when to perform procedural sedation for any given injury, one must appreciate the patient's underlying chronic illnesses, age, potential airway, and cardiovascular comorbidities and analyze these in the context of the procedure and depth of sedation required.

The American Society of Anesthesiologists classification table (see Table 5.3) is commonly utilized to describe a patient's overall health status. In ASA class I and II patients, there is less risk to procedural sedation than patients with an ASA score 3 and greater. For these higher-risk patients, another consideration must be whether the patient will be better served in the operating room.

A comprehensive history of medical issues such as cardiac or pulmonary disease, previous experiences with anesthesia and intubation, current medications, and allergies should be elicited. Additionally, environmental factors such as last meal and ingestion of drugs or alcohol should be determined.

The fasting status of a patient must be taken into consideration if the procedure is potentially elective and can be delayed. Historically, debate has been centered on whether or not prolonged fasting increases safety of procedural sedation. The American College of Emergency Physicians clinical policy states that in patients requiring emergent procedural sedation, fasting status is less relevant to the decision of whether or not to perform procedural sedation [66].

Table 5.3 ASA classification of risk

ASA Class	
I	A patient is a completely healthy, fit patient
II	A patient with mild systemic disease
III	A patient with severe systemic disease
IV	A patient with severe systemic disease that is a constant threat to life
V	A moribund patient who is not expected to survive without the operation

ASA physical status classification system is reprinted with permission of the American Society of Anesthesiologists, 1061 American Lane, Schaumburg, IL 60173–4973 [71]

However, a lighter target level of sedation for the shortest amount of time reduces vomiting and aspiration risk.

Evaluating the patient's difficulty of intubation is also extremely important. Potential difficult airways should be considered if a patient has a large tongue, short neck, micrognathia, morbid obesity, previous surgeries or radiation to the neck or airway, and a history of difficult intubations. Should these be present, careful deliberation of whether procedural sedation is appropriate should be undertaken. There are many predictors of difficult intubation, including the Mallampati score, thyromental distance, and cervical mobility. Likely a combination of these measurements and assessments most accurately predicts a difficult airway [67–72].

Preparation

Multiple steps are required to perform successful procedural sedation. It takes at least two staff members to complete the requisite tasks during a conscious sedation: administering medications, monitoring vital signs, managing the airway and it may take multiple sets of hands for complicated fracture reductions. Monitoring equipment including telemetry and capnography, supplemental oxygen, suction, airway adjuncts such as LMAs and nasal airways, a bag valve mask, and intubation equipment should be all available. Additionally, in the event of profound cardiac or respiratory depression, reversal agents and resuscitation medications should be available, as should equipment for vascular access.

Medications

Numerous medications are available for procedural sedation and analgesia (see Table 5.4). Providers must be familiar with the medication profile, target level of sedation desired, and risks for each medication provided. Commonly, medications such as midazolam, fentanyl, ketamine, etomidate, and propofol are used either in isolation or combination.

Midazolam is a short-acting benzodiazepine commonly used in isolation for minimal procedural sedation or in combination with fentanyl for moderate sedation. The peak effect is seen within 2–3 min. When used in combination with fentanyl (or other opioids), the risk of respiratory depression is heightened. Midazolam causes mild cardiovascular depression and hypotension is more common in dehydrated or volume-depleted patients. Paradoxical agitation has been reported with midazolam. Flumazenil is the reversal agent for midazolam.

Fentanyl is a short-acting opiate (see section "Pharmacologic Pain Control" for additional information). It can be used in isolation for minimal sedation or combination with midazolam for moderate sedation. A rare complication of fentanyl is rigid chest syndrome caused by spasm of the respiratory muscles, leading to hypoventilation, apnea, and hypoxia. This is seen in high doses of fentanyl administration. While naloxone may reverse effects of fentanyl, it does *not* reverse rigid chest syndrome.

Ketamine is a commonly utilized drug for moderate to deep procedural sedation. It is typically used in isolation. Ketamine produces a state of dissociation, accompanied by sedation and amnesia. Additionally, ketamine has analgesic properties in addition to some anxiolysis. The biggest advantage of ketamine is the preservation of respiratory drive with minimal effect on cardiac output and blood pressure. Ketamine can be administered either by IV or IM routes. One common side effect of ketamine is an emergence reaction, where patients experience hallucinations and nightmares as the medication wears off. Some providers will give a dose of midazolam to stave off the side effects, but the utility of this is unknown and it is not routinely recommended. It is reasonable to treat emergency reactions if they are identified. Laryngospasm has been reported with ketamine administration though it is typically transient. The provider can typically bag the patient through this process. If they are unable to successfully oxygenate the patient with BVM, the next step is to deepen the sedation and prepare for potential intubation.

Table 5.4 Medications for procedural sedation

Medication	Class	Action(s)		Dose	Adverse effects
Propofol	Alkylphenol derivative	Sedation Amnesia Anti-emetic	IV	0.5–1 mg/kg	Respiratory depression Hypotension
Ketamine	Phencyclidine derivative	Dissociation Analgesia Sedation Amnesia	IV IM	1–2 mg/kg 4–5 mg/kg	Emergence reaction Laryngospasm Emesis
Etomidate	Imidazole derivative	Sedation Amnesia	IV	0.1 mg/kg	Respiratory depression Myoclonus
Midazolam	Benzodiazepine	Sedation Amnesia	IV	0.05–0.1 mg/kg	Respiratory depression
Fentanyl	Opioid	Analgesia	IV	1 mcg/kg	Respiratory depression Rigid chest syndrome

Propofol is commonly used for moderate to deep sedation. One of the main advantages of this medication is the short duration of action and the ability to titrate this medication to effect. The effect of propofol lasts about 5–10 min. Propofol can produce both hypotension, particularly in volume-depleted patients, and sudden respiratory depression and apnea. Volume resuscitation should be performed prior to sedation. Additionally, the provider should be ready to perform airway maneuvers given the potential apneic effect. Propofol is contraindicated in patients with an egg or soy allergy. It is common for patients to experience a local burning effect at the IV site as the propofol is administered.

Often, ketamine and propofol are combined together for moderate to deep procedural sedation. The combination has an excellent safety profile and is intended to reduce risks of using either agent alone. Using ketamine in addition to propofol may help mitigate the respiratory and cardiovascular depression of propofol alone and lessen the occurrence of emergence reactions from ketamine alone. Additionally, propofol has no intrinsic analgesic properties so adding ketamine to this equation diminishes the need for additional medications such as opioids. There are multiple combinations of mixing, though most recommend starting with a dose of 0.5 mg/kg for each ketamine and propofol given separately from separate syringes and continuing to titrate to effect with additional propofol as needed.

Regardless of medication regimen selected, patients should be monitored for respiratory depression, laryngospasm, cardiovascular and hemodynamic instability, dysrhythmias, along with less serious complications such as transient hypoxia, emesis, and inadequate sedation.

Procedural and Post-procedural Monitoring and Recovery

During the procedure the patient should be continuously monitored and assessed. Depending on the desired depth of sedation, a patient's ability to follow commands and respond to stimuli should be measured. Heart rate, respiratory rate, blood pressure, hypoxia, and end-tidal carbon dioxide should be continuously assessed until the patient has recovered from sedation. The procedure should be aborted if patients experience substantial complications from the sedation medications.

Patients should be monitored until they have returned to their baseline mental status and do not require any additional cardiovascular or respiratory support. They should be able to speak, follow commands, and ambulate (if able) at baseline. Once patients have achieved this level of consciousness, it is unlikely there will be additional complications. Few adverse events have been demonstrated beyond 25 min after the final medication dose [73]. Patients should not drive for 24 h following procedural sedation.

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6 Imaging

Jordan S. Gold, Adam C. Zoga, and John C. Hill

Key Points

- Radiography is the first-line modality for sports-related trauma to evaluate for fracture.
- Computed tomography (CT) imaging is useful for evaluating the extent of osseous injury and preoperative treatment planning.
- Musculoskeletal ultrasound is useful in assessing ligament and tendon injuries and dynamic stress imaging can be used to grade ligament tears more efficiently and without the need for additional radiation.
- Magnetic resonance imaging (MRI) is the best modality to evaluate most soft tissue injuries; however, ultrasound can provide an alternative modality in certain situations.
- MRI should be performed for suspected radiographically occult fractures or stress fractures.
- Sports-related head and spine injuries should be imaged with CT and MRI; radiographs have limited utility.

Introduction

Sports-related injuries are encountered across a wide range of clinical settings and presentations including both acute traumatic events and more chronic overuse injuries. Radiological imaging plays a crucial role in the management of these individuals and can help determine a diagnosis and subsequently guide treatment. The imaging study of choice often depends on the type of activity, the athletic level or specific population involved, and the availability of resources. In the acute setting, radiography is often the first appropriate step in management given that the modality is easily accessible and allows for quick evaluation. However, often radiographs will not provide the degree of information needed to understand the extent of a particular injury, specifically those involving soft tissue pathology rather than the osseous structures. Computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound can provide the practitioner with important information that can aid in the appropriate next step in a patient's management. Advantage and disadvantage of these modalities are summarized in Table 6.1.

Sports-related trauma occurs in a variety of environments and across all athletic competitive levels, each of

Table 6.1 Imaging modalities commonly used in acute traumatic sports injuries

	Pros	Cons
Radiographs (X-rays)	Easily accessible and fast Osseous detail	Limited evaluation of soft tissue Radiation – low
CT	Extent of fracture injury Subtle fractures Faster than MRI Vascular injury	Limited evaluation of soft tissue compared to MRI Radiation – high
MRI	Soft tissue evaluation Osseous injury – contusions and stress injury No radiation	Limited availability Length of exam Patient discomfort Other contraindications
Bone scintigraphy	Sensitive for osseous stress (overuse) injuries	Length of exam Limited availability Radiation – medium
Ultrasound	Dynamic imaging Physical exam Rapid assessment No radiation	Limited utility for osseous structures Operator dependent

J. S. Gold (✉)
Department of Radiology, MedStar Washington Hospital Center, Washington, DC, USA
e-mail: Jordan.S.Gold@medstar.net

A. C. Zoga
Musculoskeletal Radiology, Sidney Kimmel Medical College, Thomas Jefferson University, Philadelphia, PA, USA

J. C. Hill
Department of Family Medicine and Orthopedics, CU Sports Medicine, University of Colorado School of Medicine, Denver, CO, USA

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which present with a different set of obstacles in terms of imaging. Starting with the top at the professional level, most high-end sports venues have access to radiographic equipment on-site for immediate imaging after an injury. Additionally, with advances in imaging system technology, an image can be transmitted digitally to a picture archiving and communication system (PACS) to be interpreted remotely by an expert musculoskeletal (MSK) radiologist if one is not available at the game or event. A *PACS* consists of the imaging modality equipment, a secure network for transmission, workstations for viewing and interpretation, and an archiving system for storage. The universal file format for transfer is DICOM (Digital Imaging and Communications in Medicine), which allows for preservation of image quality during the transmission process and is capable of including additional necessary patient information. Since professional sports teams, trainers, and team physicians tend to work closely with MSK radiologists, it is likely that they feel comfortable with the interpretation provided by those radiologists whom they have already established working relationships [1–4].

Obviously, the vast majority of sports-related injuries occur in settings without immediate access to imaging. Whether it be the young little leaguer with an awkward slide or middle-aged weekend warrior who pushed his marathon training too far, these patients often experience trauma that does not prompt a visit to the emergency room, but eventually warrants a trip to their primary care physician. Radiographs typically serve as the initial imaging modality of choice in these sports-related injuries, although when and how to image appropriately can be a difficult point of care in management. Fortunately over the years, many guidelines and algorithms have been developed to aid practitioners in the decision of whether radiographic imaging is necessary. The importance of appropriate imaging is multifaceted but at the forefront is limiting radiation exposure when possible to avoid any potential future adverse health effects. Albeit radiographs impart a relatively low dose of ionizing radiation, radiation exposure is not benign and the unnecessary use of radiographic imaging often propagates to other modalities including CT capable of transmitting significantly higher radiation doses. Additionally, given the growing interest in mitigating healthcare costs, avoiding unnecessary imaging would certainly help alleviate the cost burden on the healthcare system, especially when considering the sheer volume of sports-related injuries.

In 2014, Congress passed a bill, known colloquially as the Protecting Access to Medicare Act of 2014 (H.R. 4032) to address the utilization of imaging services. The bill mandated that the Centers for Medicare and Medicaid to specify a program requiring clinicians to consult with a qualified clinical decision support mechanism (CDSM) that relies on appropriate use criteria (AUC) when ordering advanced diagnostic imaging services. Although the incentive was targeted toward advanced imaging utilization, namely, CT, MRI, and nuclear medicine and excluded radiography, fluoroscopy, and ultrasound, the initiative did provide a further push to develop criteria for appropriate radiological imaging. One such resource was developed by the American College of Radiology (ACR), known as the ACR Appropriateness Criteria®. Collectively, this represents a large set of evidence-based guidelines to assist referring physicians and other primary care providers with selecting the most appropriate imaging modality or treatment, which are tailored to specific clinical scenarios. Each set of guidelines for a particular clinical scenario is developed by a panel of experts in that subspecialty of radiology. The appropriateness of particular imaging tests is subsequently given a rating on a 1–9 ordinal scale divided into three categories: “Usually not appropriate,” 1–3; “May be appropriate,” 4–6; and “Usually appropriate,” 7–9. These guidelines are searchable and readily available to referring physicians and practitioners in the field.

Radiography

Radiography is an imaging modality, which uses ionizing electromagnetic radiation, X-rays, to obtain a representative image of an object based on density characteristics. The resulting “radiograph” or “X-ray” has been the cornerstone of sports-related traumatic imaging since their initial discovery. The first documented example of a diagnostic radiograph was performed on February 3, 1896, at Dartmouth University by Dr. Gilman Frost on a young boy who had been treated for a wrist fracture. Often the initial imaging modality of choice, the radiograph evaluates the integrity of osseous structures (e.g., fractures). However, significant additional information can be obtained with careful review of the regional soft tissues.

Radiographic technique is extremely important to allow for adequate interpretation by radiologists and practitioners. Obtaining multiple views or projections when performing radiographs is critical. It is not uncommon for nondisplaced fractures to only be visible in a single projection due to in-plane fracture orientation with the inherent 2-D nature of radiography and other overlapping osseous structures (Fig. 6.1). Additionally, fracture displacement or angulation cannot be adequately evaluated, unless the abnormality is viewed in multiple projections. Furthermore, many radiographic signs rely on accurate patient positioning; consider the elbow joint effusion, which may be the only indication of a “radiographically occult” fracture. For example, when an elbow joint effusion is present, it can be seen on a true lateral radiographic projection as displacement or elevation of the posterior fat pad with crescentic lucency posterior to the dis-

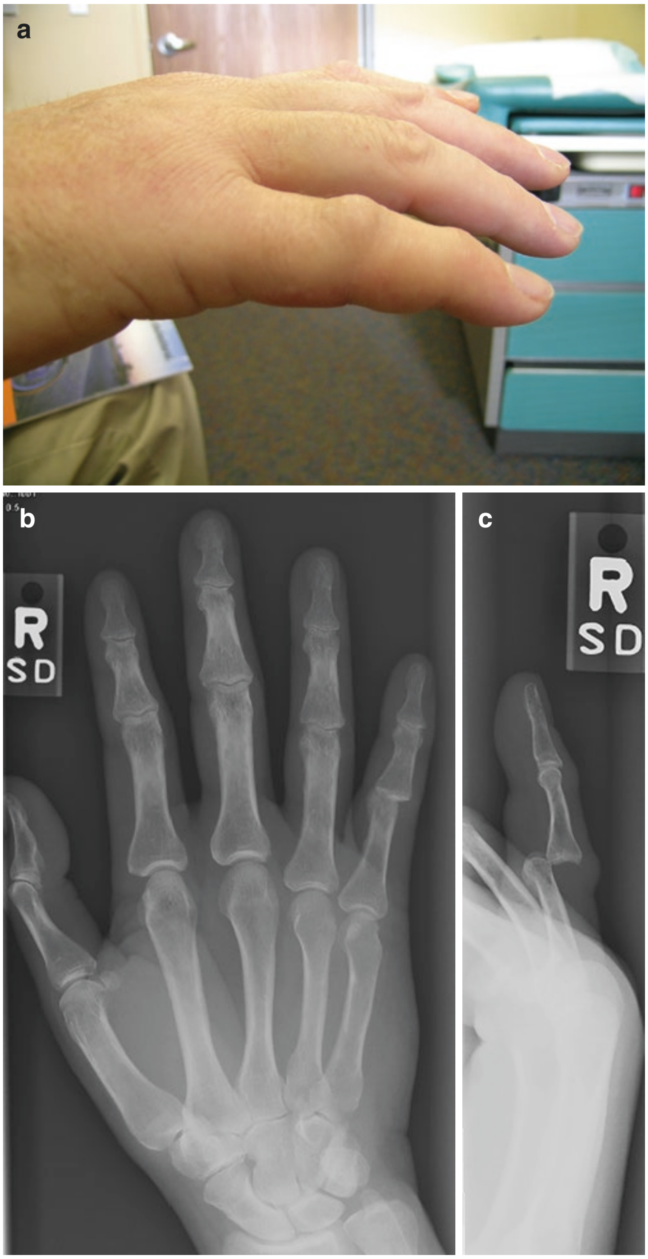


Fig. 6.1 Right 5th posterior PIPJ dislocation in a 51-year-old male as a result of his finger being jammed with the basketball (**a**). The dislocation could have been missed if only the AP (**b**) view was performed and not the lateral (**c**) view

tal humerus; however, if the fat pad is viewed out of plane, attenuation related to the surrounding soft tissue will obscure the finding and thus make the diagnosis more difficult to appreciate. Oftentimes, patients have limited mobility or pain with certain positions after an injury; however, it is imperative for technologists to be well trained in proper radiographic technique. In young patients before the closure of the epiphyseal plate, it may be necessary to obtain the contralateral images to identify subtle abnormalities from normal epiphyseal patterns.

A fundamental consideration throughout all of radiology but of vital importance to radiography is providing an accurate and specific history when possible. Radiographic techniques can be employed to obtain projectional views to evaluate certain osseous structures. Additionally, radiographic stress views can be helpful to extenuate various findings or demonstrate instability. For example, "Flamingo stress views" demonstrate step-off at the pubic symphysis suggestive of pelvic instability (Fig. 6.2). Several additional examples of particular radiographic technique will be discussed in the following sections.

Computed Tomography (CT)

CT imaging is a modality that uses ionizing electromagnetic radiation in a similar fashion to radiographs. However, X-rays are projected at multiple differing angles to produce a cross-sectional representation of anatomy based on tissue densities after computer processing of the data. Advantages of CT imaging include the removal of superimposition of structures in the area of interest and improved contrast resolution relative to radiographs allowing for small differences in tissue attenuation to be appreciated. Furthermore, the data obtained from a CT scan can be manipulated to view images in various planes, routinely axial, coronal and sagittal planes, which has essentially become the standard of care when it comes to CT imaging. Additionally, software allow for 3-D reconstructed images (surface or volume rendering) to be created, providing the radiologist and other physicians with valuable information, which may be difficult to appreciate on cross-sectional viewing. This can be helpful in surgical and treatment planning (Fig. 6.3). CT imaging is still relatively fast to perform with the vast majority of MSK applications scanned in under 5 minutes.

Certainly, the increased information obtained from CT compared to radiography is not entirely free. As briefly discussed earlier, the radiation dose delivered by CT imaging is substantially higher than routine radiography and is influenced by a number of different factors. However, as ordering providers, it is important to be aware of the potential adverse health risks related to excessive radiation exposure when choosing imaging studies. In sports-related trauma, this is even more critical, given that the individuals participating in these activities are typically younger than the general population and therefore at a greater risk to the potential adverse effects of radiation.

In regard to CT imaging after an acute sports-related injury, the vast majority of indications are related to determining the extent of injury and to guide surgical planning. Occasionally, persistent pain with negative radiographs will prompt a CT study; however, in most of those circumstances,

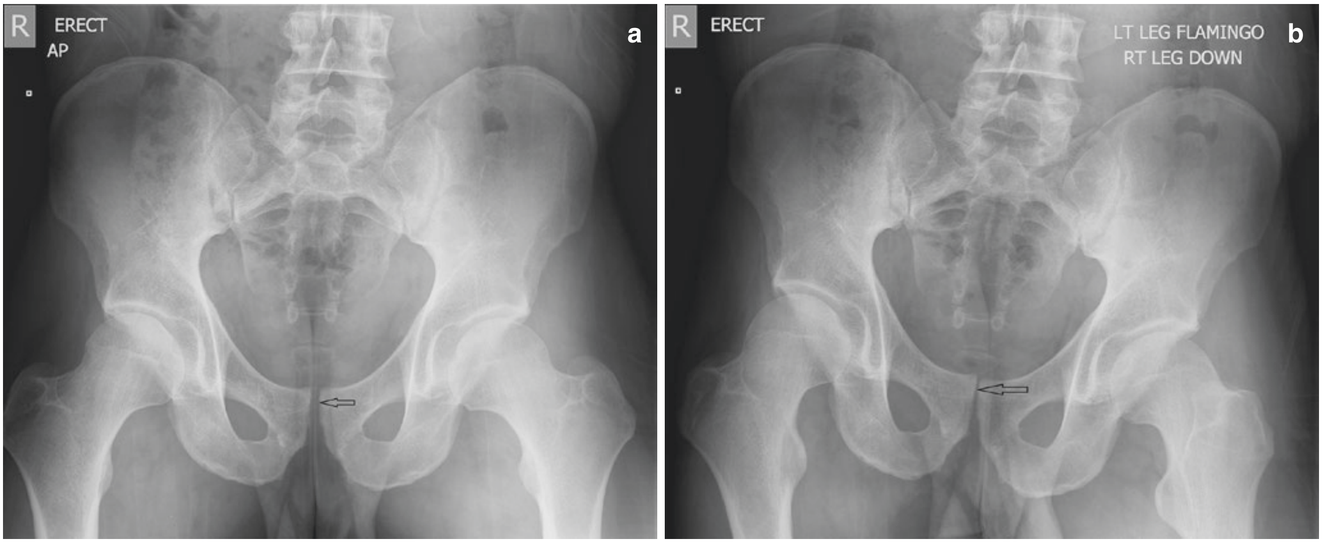
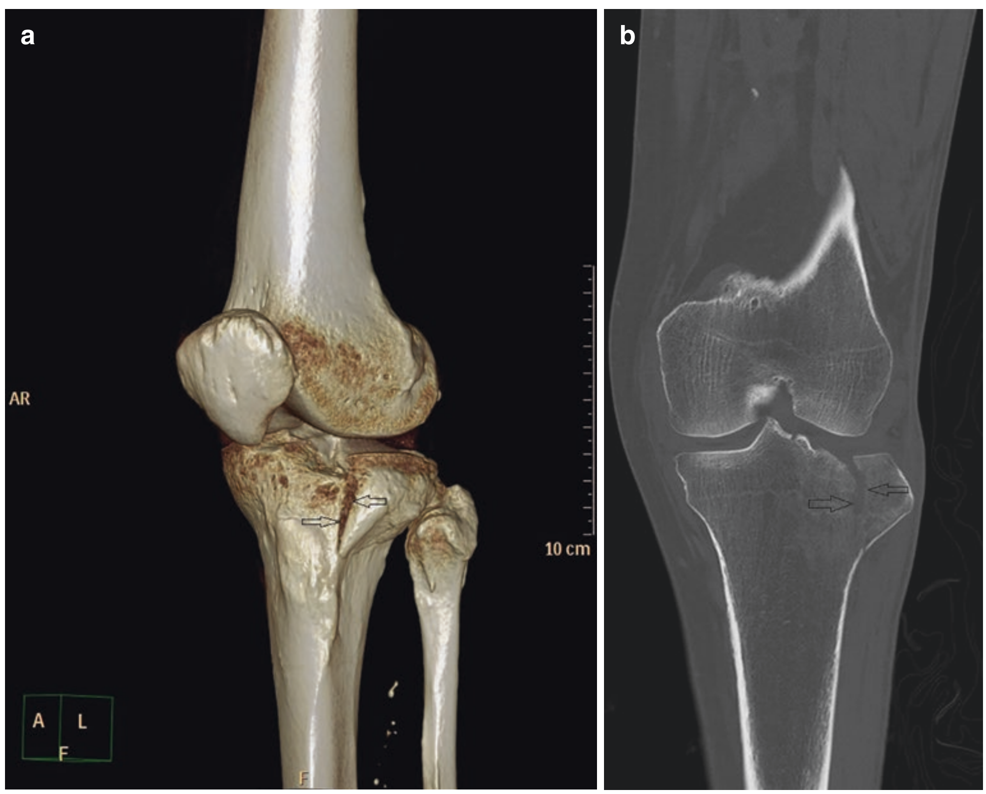


Fig. 6.2 Two erect AP radiographs of the pelvis demonstrate neutral (**a**) and stress (**b**) flamingo views, respectively, in a professional hockey player. Pubic symphysis offset (arrows) is only identified on the stress views suggestive of pelvic instability

Fig. 6.3 Reconstructed 3-D (**a**) and coronal CT (**b**) images demonstrating a proximal tibial fracture involving the lateral plateau. Reformatted and reconstructed images are useful for surgical planning



an MRI study will likely be higher yield given improved contrast resolution and better soft tissue evaluation. Fortunately, with MSK imaging in the acute setting, intravenous iodinated contrast is rarely indicated, which alleviates any of the potential complications related to its administration. Arthrography is a technique during which a contrast solution is injected into the joint space allowing for distention and improved visualization of various structures. CT and MR arthrography is routinely performed sports-related injuries and will increase the diagnostic yield, although typically MR arthrography is the preferred modality. Specific indications for CT arthrography include patients whom are unable to tolerate MRI and those with orthopedic hardware limiting the diagnostic utility of MRI due to regional artifact.

Magnetic Resonance Imaging (MRI)

MRI is a technique using powerful magnetic fields, radiofrequency pulses, and complicated computer processing to create representative images of the internal anatomy primarily based on hydrogen protons due to their abundance in both water and fat; noting however this is simplified explanation. MRI does not use ionizing radiation and those potential adverse health effects are negated. Additionally, MRI has improved contrast resolution compared to both radiography and CT imaging, which allows for anatomic differentiation based on molecular structure rather than simply density. MR imaging uses various parameter changes to distinguish between tissue components or emphasize particular anatomic structures. For example, several common MRI sequences use a technique known as fat suppression to remove the inherent “fat-related” signal in an image, which essentially creates a fluid-sensitive image. Many pathologic processes cause localized swelling or edema involving both the osseous structures and the surrounding soft tissues, which helps identify injuries. MR arthrography, as briefly described earlier, involves the instillation of a gadolinium-based contrast solution into the joint space allowing for improved evaluation of several intra-articular structures. For example, the glenoid labrum at the shoulder and acetabular labrum at the hip, although arthrography at multiple joints, can be helpful depending on the clinical scenario.

MRI has several disadvantages as an imaging modality. First and foremost is *time*; many MR imaging studies can take upwards of 30–60 minutes to achieve an adequate diagnostic study. Since imaging acquisition times are longer, there is an increased likelihood of motion-related artifact, which can obscure the images. Many patients will experience anxiety or claustrophobia related to MR scanning because of their positioning inside the imaging bore and the significant noise created by the system. Moreover, pain after an acute injury may pose difficulty in maintaining appropriate positioning within the scanner for an extended period of time. Recent advancements with MRI technology and software have shortened scan time and the MR imaging bores have become larger and more comfortable; however, many individuals still experience apprehension and emotional distress. In regard to imaging, spatial resolution is compromised with MRI relative to the previously described modalities; however, for MSK imaging, this does not typically impact interpretation or diagnostic ability (Fig. 6.4).

Ultrasound

Ultrasound is defined as sound having a frequency higher than that which is audible by humans (15,000–20,000 Hz). MSK imaging uses frequencies ranging from 4 to 18 megahertz (MHz). Higher frequency transducers give better resolution but lower penetration which is ideal for imaging superficial structures. Lower frequency transducers allow for better penetration of the tissues but have lower resolution and are recommended for imaging deep structures. Sound waves are the form of energy used with ultrasound, and therefore no ionizing electromagnetic radiation is required. Electric voltage is converted to sound waves by the reverse piezoelectric effect. Sound waves are reflected at tissue boundaries producing echoes. Sound wave echoes return to the transducer and are converted into volts by the piezoelectric effect; these volts are assigned a gray-scale color and location on the screen to produce an image.

Diagnostic ultrasound provides detailed evaluation of MSK structures and offers several advantages compared to other imaging modalities. There is the ability to image in real

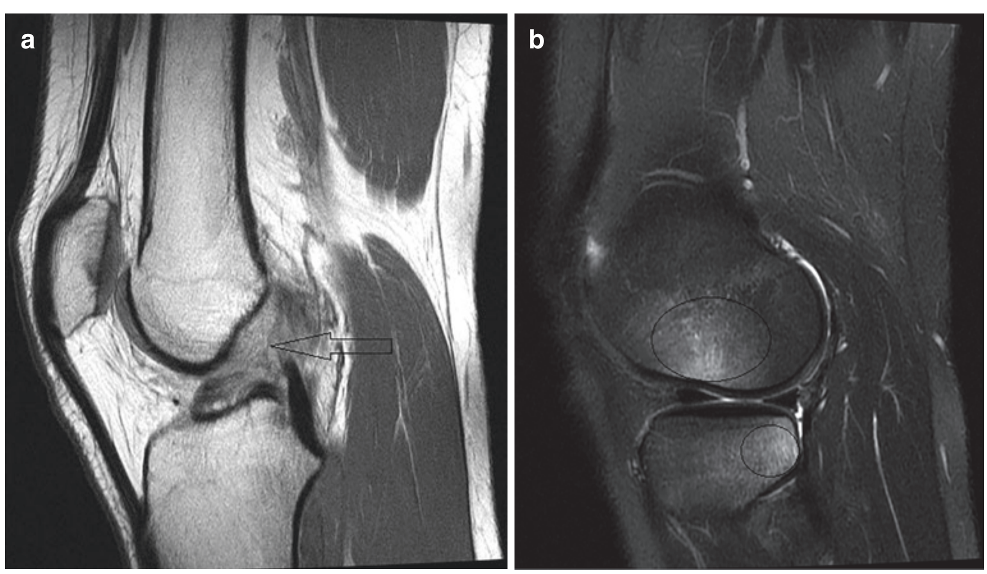


Fig. 6.4 Sagittal proton density (**a**) and fat-saturated T2-weighted (**b**) sequences of the knee, respectively. MRI allows for evaluation of both soft tissue and bony structures, clearly demonstrating a complete tear of the ACL (arrow) and associated pivot-shift osseous contusions (circles) at the sulcus terminalis along the lateral femoral condyle and posterior aspect of the lateral tibial plateau

time and interact with the patient during the examination, sometimes dynamically imaging the structures which cannot be done with MRI. High-resolution images are obtained and these images can be compared to the contralateral extremity with minimal effort. There is minimal artifact with implanted metal and complete safety in patients with implanted pacemakers. The machines are getting smaller, are more portable, and are relatively inexpensive. There is no radiation and no known contraindications. Frequently after a diagnosis is made, an intervention using an ultrasound-guided injection or procedure can be done at the same time.

The limitations of ultrasound must be recognized. There is a limited field of view and limited penetration of tissues. Sound is reflected by bone which makes it difficult to image inside a joint and only the leading edge of bone can be visualized. There is variability between machines and some operators are more skilled than others.

Diagnostic MSK ultrasound examinations are divided into complete assessments of a joint or anatomic region or limited assessments of specific structures such as the patellar tendon. The American Institute of Ultrasound in Medicine (AIUM) has defined what these complete assessments are [5]. All relevant structures within an anatomic region must be examined including muscles, tendons, joints, and sometimes nerves to identify abnormalities. Most joints are divided into quadrants (anterior, posterior, lateral, medial); however, a complete shoulder evaluation includes all relevant structures including the rotator cuff, biceps tendon, ACJ, and GHJ. As is true with all imaging modalities, structures must be examined in at least two planes, long axis and short axis.

Bone Scintigraphy

Bone scintigraphy is an imaging technique in nuclear medicine allowing for visualization of bone metabolism rather than simple anatomic structure. As opposed to radiographic or CT imaging, bone scintigraphy involves intravenous injection of a radiopharmaceutical, which emits ionizing radiation that can subsequently be captured by specialized cameras. The most commonly used radiopharmaceutical for bone scintigraphy is methylene diphosphonate (MDP) coupled with radioisotope technetium-99 m. Essentially, MDP will be absorbed in regions with active bone turnover, and the greater uptake will lead to increased regional radioactivity by imaging with a gamma camera. Traditional gamma camera imaging is planar with imaging acquired in the anterior and posterior planes. Three-phase bone scans refer to imaging acquired during the flow phase, blood-pool phase, and delayed phase, which increases the diagnostic yield. Additionally, SPECT (single-photon emission computed tomography) allows for the obtained images can be viewed in cross-section similar to a typical CT and allow for improved localization. Although there is a large variance among ordering practices from one institution to another, clinicians are typically less familiar with nuclear medicine and often it is not the first-line for sports-related injuries. However, bone scintigraphy will provide for diagnoses for sports-related injuries and can be considered when MRI results are inconclusive [6, 7].

Bone scintigraphy does involve ionizing radiation, which should be taken into consideration when imaging athletes who would obtain similar diagnostic information from MRI studies. Additionally, many outpatient imaging centers will not have the radiopharmaceuticals readily available and the necessary equipment to perform routine nuclear medicine studies; therefore, the feasibility may be decreased outside of the hospital setting.

Sports Injuries by Anatomical Region

Lower Extremity

One of the most recognized and earliest developed guidelines for practitioners is the Ottawa Ankle Rules (OAR). Ankle injuries are one of the most common reasons for visits to both the emergency room and outpatient setting estimated at 200 visits per 100,000 person-years [8, 9]. However, the relative low incidence of fractures inclined Stiell et al. to develop and publish a set of guidelines in 1992 to identify patients that could be safely treated without radiography based on statistical likelihood of a fracture [10]. In short, the OAR guidelines are as follows: (1) inability to bear weight immediately after the injury, (2) point tenderness over the medial malleolus, the posterior edge or inferior tip of the lateral malleolus, talus, or calcaneus, or (3) inability to ambulate four steps in the emergency department. The Ottawa Ankle Rules have become a successful example of a decision support algorithm because they are reliable and easily applicable and can be assessed quickly and accurately. The ACR Appropriateness Criteria have incorporated many well-established guidelines, such as the OAR, while adding additional potential complicated factors that may influence management [8].

Standard ankle radiographs should consist of at least three views: anterior-posterior (AP), lateral, and an ankle mortise view. Specific radiographic imaging technique or additional special views may also help establish a diagnosis, when there is clinical concern for a particular injury. For example, when physical examination is concerning for a Lisfranc injury, weight-bearing radiographs are indicated; these may demonstrate widening or malalignment at the Lisfranc articulation, which may not necessarily be present on a non-weight-bearing examination. As advanced imaging modalities have become more readily available, the need for special views or

projections has been diminished, although radiologists may suggest additional views when the initial radiographs are concerning for a particular findings. As often is the case however, radiographs will not be sufficient to delineate the extent of an injury.

Other common ankle injuries can be accurately diagnosed with ultrasound. Achilles tendinitis can be differentiated from an acute tendon rupture or even a partial tendon tear (Fig. 6.5). Insertional calcific Achilles tendinosis can be differentiated from retrocalcaneal bursitis with MSK ultrasound. Dynamic ultrasound imaging of an acute ankle sprain can often delineate the degree of an ankle sprain and may replace the need for stress radiographs [11].

Certain injuries discovered by radiograph in one location should direct the search for associated findings elsewhere based on well-established injury mechanisms. For example, when a tibiofibular syndesmotic and/or deltoid ligamentous complex injury is suspected at the ankle, it is important to then assess the proximal fibula for a spiral fracture; this combination of findings is known as a Maisonneuve fracture and usually caused by pronation-external rotation mechanism and will likely require surgical stabilization (Fig. 6.6). Another example of coexisting injuries is seen with bilateral calcaneal fractures (also known as the "lover's fracture" or "jumpers fracture"), which are typically caused by an axial loading mechanism, possibly occurring after jumping from a height while trying to escape a lover's spouse. Given the distribution of forces with axial loading, it is important to evaluate the spine, specifically the lumbar spine, for concurrent injury.

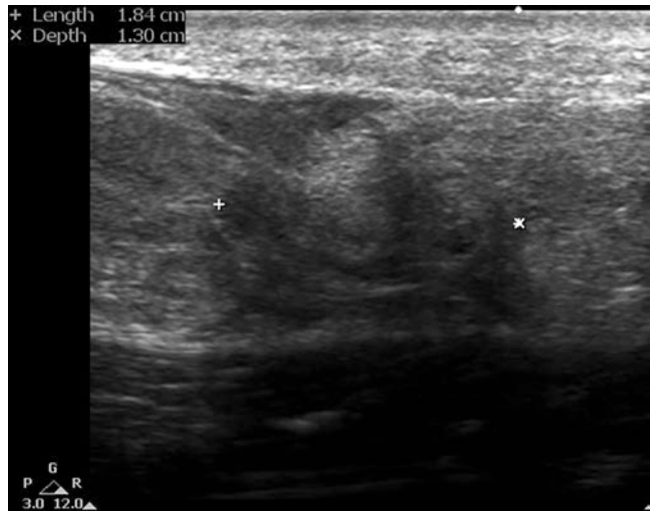


Fig. 6.5 Acute right Achilles tendon rupture (1.84 cm gap) in a recreational soccer player. Ultrasound confirmed the complete rupture and patient did not get MRI

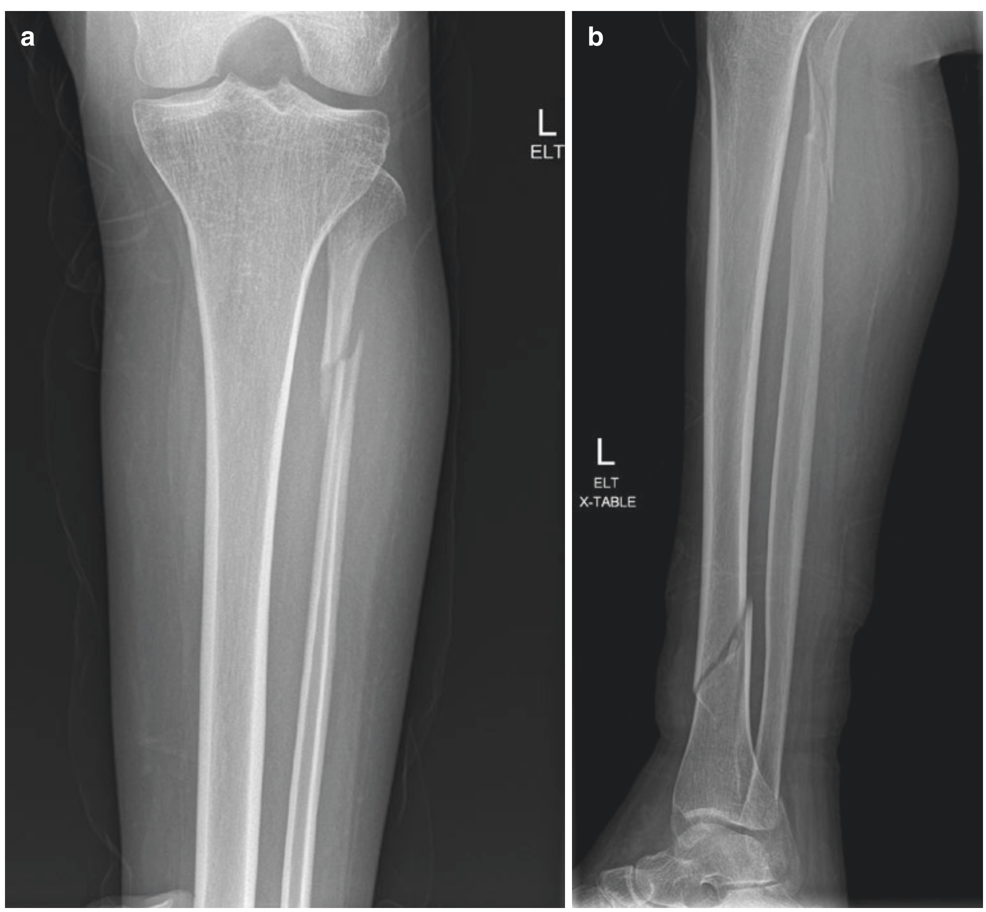


Fig. 6.6 Maisonneuve (distal tibia and proximal fibula) fracture in a 36-year-old female (**a**, **b**)

CT imaging can be quite useful in evaluating the extent of an osseous injury involving the lower extremity. Specifically, many fractures of the foot and ankle after poly-trauma are often discovered on CT after not being identified on the initial radiographs; one study suggested that 25–33% of midfoot fractures identifiable on CT were not detectable on radiographs [12, 13]. If there is a concern for a subtle Lisfranc injury with normal initial radiographs, then CT or MRI is indicated, especially when a patient is unable to bear weight. CT is far better at delineating the multiple metatarsal and cuneiform fractures that typically accompany a Lisfranc ligamentous injury. When evaluating talar or calcaneal fractures at the ankle, CT imaging is also the modality of choice, allowing for visualization of the extent of both intra-articular and extra-articular components. The Sanders CT classification of calcaneal fractures is a widely used system based on intra-articular extension, which correlates well with long-term prognostic outcome [14, 15].

Sports-related trauma involving the knee will often prompt providers to order radiographs as the initial test for evaluation. However, acute fractures involving the knee are actually a relatively uncommon finding with prior studies suggesting only 5% of patients with acute knee pain demonstrate a fracture by radiograph [16]. The most common and well-validated clinical decision rule set is the Ottawa Knee Rules published by Stiell et al. in 1995, with a reported 97–100% sensitivity for fracture; additionally this resulted in a 28% relative reduction in the number of radiographs ordered by the emergency departments in the study [17]. Several prospective follow-up studies were performed using these rules with similar results; however, it is important to recognize the limitations or confounding factors, such as the pediatric population, intoxicated patients, acute high-speed (e.g., skiing) injuries, or those with diminished sensation. Essentially, radiographs should be the initial imaging study after a fall or twisting injury with one or more of the following: focal tenderness, joint effusion, or inability to bear weight on physical exam. Clinical judgment should always determine the best way to manage a patient. Radiographs can be helpful in establishing other findings aside from fractures; the presence of joint effusion, anterior tibial translation (Fig. 6.7), or patellar malalignment is often associated with internal derangement or soft tissue injury needing further evaluation with MRI.

MRI is the workhorse at the knee given the superior soft tissue assessment compared to other imaging techniques while also providing significant information related to the bones. In the ER setting, 93.5% of patients presenting for acute knee trauma will have a soft tissue injury as opposed to an osseous injury [16, 18, 19]. Therefore, any patient with a recent fall or twisting injury and no tibial plateau fracture on the initial radiographs would benefit from an MRI. Additionally, an MRI is indicated in patients with a Segond fracture (avulsion fracture at the lateral tibial plateau)

Fig. 6.7 Anterior tibial translation in a patient with acute ACL rupture

given the high incidence of concurrent ACL injury (Fig. 6.8). MRI is highly sensitive for evaluating meniscal, ligamentous, and tendinous pathology and frequently coexisting osseous pathology such as contusion or fracture [20]. Some of the superficial injuries about the knee are visualized well with ultrasound. Patellar tendinitis, medial collateral ligament sprain, and quadriceps tendon ruptures can be quickly and accurately assessed. In addition to this, acute injuries to the gastrocnemius and quadriceps muscles (Fig. 6.9) are able to be quickly diagnosed. A rapidly developing hematoma can be seen which would allow the blood to be drained under ultrasound guidance and then compressed to prevent further bleeding into the tissue and possibly prevent a compartment syndrome from developing [21].

In cases of knee fractures, generally involving the tibial plateau, CT imaging is appropriate and can improve surgical planning, specifically when using the Schatzker classification system [16]. Additionally, the diagnostic utility of CT is important after posterior knee dislocation, which can occur in high-impact sports; vascular or nerve injury represents a serious potential complication related with studies suggesting a frequency between 5% and 11% within the relative low-velocity mechanisms such as sports-related activities [22, 23]. A CT-arteriogram protocol with intravenous contrast is indicated to evaluate for vascular injury in these patients. Further advances in CT imaging, specifically the

Fig. 6.8 Segond fracture (avulsion fracture at the lateral tibial plateau) in a 22-year-old female patient with acute ACL rupture as a result of a ski injury

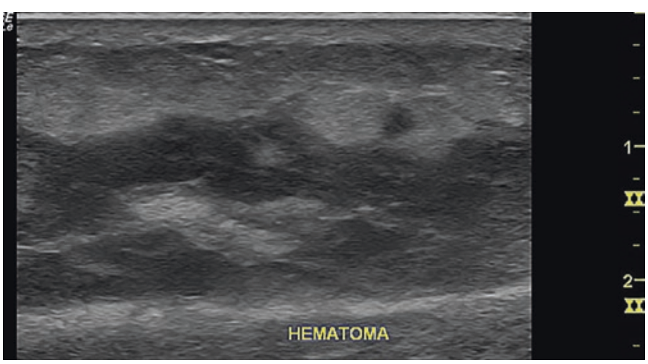


Fig. 6.9 Ultrasound image of gastrocnemius hematoma which is organized without evidence of acute bleeding. This is a hematoma which cannot be aspirated in this stage

development of dual-energy techniques, have shown significant promise with improved soft tissue resolution and even the visualization of bone marrow edema, which could certainly expand CT's role in the acute setting [24].

Hip and groin pain in the athlete is quite common; fortunately, fractures and dislocations are rare. Occasionally, traumatic dislocation and fracture will be seen with high-impact sports, such as football or skiing, often related to underlying dysplasia or other predisposing factors. Radiographs are always appropriate; however, additional CT imaging should be performed to evaluate complex acetabular and pelvic fractures [25].

Additionally, many athletes experience chronic hip and groin discomfort related to sports injuries. Radiographs are indicated in this clinical scenario as well with significant obtainable information, which may be helpful in guiding clinical management. For example, entities such femoroacetabular impingement (FAI) morphology, hip/acetabular dysplasia, and acetabular retroversion are typically assessed on radiographs; numerous standardized measurements by radiograph have been developed to classify the degree of both developmental and acquired conditions that are commonly seen in athletes [26, 27]. For athletes with chronic hip or groin pain, MRI is again the mainstay of imaging. Specifically, if there is clinical concern for labral or chondral pathology at the hip, athletic pubalgia, and certainly stress fractures or other overuse injury, an MRI is usually indicated [28–30]. MR arthrography, preferably direct with intra-articular injection, can be helpful for evaluating the labrum and cartilage [25, 31–33].

After a sports-related injury, MRI is the modality of choice to evaluate for muscular and tendinous injury that is commonly seen in the calf and thigh. MRI will diagnosis and grade injuries such as muscle strains and tendon tears while providing important information for surgeons such as tendon retraction. Soft tissue hematomas or contusions will also be visible by MRI. Even entities such as delay-onset muscle soreness or exercise-induced compartment syndrome may be unmasked as intramuscular edema in athletes [34, 35].

MSK ultrasound can also be used in the subacute setting to diagnose the same injuries as described above. In addition to these injuries, ultrasound can help differentiate the causes of athletic pubalgia which might be caused by a partial thickness tear at the origin of the adductor longus or the insertion of the rectus abdominis. The pain could come from entrapment of the genitofemoral nerve or a sports hernia could be causing the athletic pubalgia. Dynamic imaging of the lower quadrant of the abdomen with the use of Valsalva can demonstrate bulging of the sports hernia which is not possible with any other imaging modality [36].

Overuse or stress-related injuries are a common cause of pathology encountered in sports medicine. Conventional radiographs can be helpful in identifying patients with periosteal reaction related to stress response or healing stress fracture, although sensitivity is limited in the initial examinations, ranging from 15 to 35% and subsequently increasing to 30–70% on later follow-up examinations [7, 37]. MRI allows for recognition of subtle osseous changes not identifi-

able by radiograph and should be performed when stress syndromes are clinically suspected after negative radiographs; reported sensitivities for MRI are nearly 100% [7]. Tibial stress fracture and stress response is the most common site of overuse injury and should be evaluated with MRI; several studies grading medial tibial stress injuries using MRI findings have proposed time intervals to healing based on the degree of marrow signal abnormality, which may aid in treatment and management.

Upper Extremity

Hand and wrist anatomy is complex consisting of multiple dynamic components with numerous different structures that are vulnerable to injury during sports-related activities. The vast majority of abnormalities should initially be imaged by radiograph, and it is of utmost importance to provide an accurate and specific history when ordering these studies due to the number of different radiographic projections used to appropriately evaluate the various structures. For example, with concern for a scaphoid fracture after a FOOSH injury, a "scaphoid series" should be performed, which includes a dedicated view angled to visualize the scaphoid in long axis without any additional overlapping osseous structures (Fig. 6.10). In case of any concern for scapholunate ligament rupture, a clenched view can help detect subtle widening (Fig. 6.11). If there is concern for fracture of the hook of the hamate, pisiform, or trapezium, a supplemental carpal tunnel view (Fig. 6.12) can be performed allowing for better visualization of these structures.

Given the risk of complications related to untreated fractures at the hand and wrist, specifically radiographically occult nondisplaced scaphoid or distal radius fractures, it is recommended that follow-up or repeat radiographs be performed in 10–14 days after immobilization in certain situations when the initial radiographs are negative [38]. Alternatively, MR and CT imaging is considered an appropriate study with suspicion for these fractures or other radiographically occult fractures, metacarpal dislocations, and distal radioulnar joint subluxation. However, when there is concern for ligamentous, tendinous, or volar plate injury at the hand or thumb, MRI should be performed. MRI is also useful for delineating osseous contusion that frequently occurs in conjunction with fractures in the acute setting or in patients with unexplained symptoms [39].

Fig. 6.10 Scaphoid (ulnar) view reveals a nondisplaced mid scaphoid fracture in a 23-year-old female as a result of a snowboarding injury

Fig. 6.11 A clenched view demonstrates widening of the scapholunate space (>4 mm) in a 45-year-old male as a result of a fall while snowboarding

Fig. 6.12 A normal carpal tunnel view

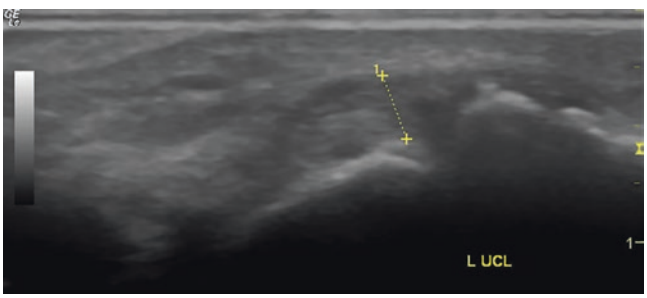


Fig. 6.13 Ultrasound image of left 1st CMC joint with evidence of thickening of the UCL without evidence of joint space widening with stressing the joint

Ultrasound is helpful in similar situations, such as a UCL sprain of the first CMJ (Skier's thumb). Clinically it is often difficult to assess the degree of ligament sprain. Gentle traction can be applied to the joint while examining the UCL under high frequency ultrasound. If a thickened, hypoechoic (edema) ligament is seen but no laxity is assessed, then you can confidently manage this sprain as a grade 1 UCL sprain (Fig. 6.13). In contrast, if the same gentle pressure causes the joint space to easily open and little resistance is noted, you might even see the ligament within the joint space (Stener's lesion) and then you can quickly diagnose a grade 3 or complete rupture of the ligament and refer the patient for operative management [40].

Acute sports-related injuries involving the elbow and shoulder may result in fracture and/or dislocation, for which radiography would also be the initial imaging study of choice. As discussed earlier, accurate history will provide the radiologists and technologists with the information to perform the diagnostic study with the highest yield. At the elbow, anterior-posterior (AP) and lateral radiographs are performed; however, additional oblique views will increase the sensitivity for fracture visualization. The presence of an elbow joint effusion can signify a radiographically occult fracture or other ligamentous/tendinous injury. At the shoulder, typical radiographs include AP views while in internal rotation and external rotation, scapular Y or lateral view, and often an axillary view. Numerous additional special views with various names can be performed in certain clinical scenarios; for example, a Stryker notch view can be helpful after anterior shoulder dislocation to evaluate for a Hill-Sachs defect or osseous (Bony) Bankart lesion. After dislocation or in the presence of complex fractures, CT imaging should always be performed to evaluate the extent of injury and articular surface involvement prior to surgical intervention at the shoulder and elbow [41].

However, similar to other locations and clearly a recurring theme, radiographs provide a quick initial assessment, although in sports-related trauma, soft tissue injury is quite common and MRI is the more important modality. Numerous ligamentous and tendinous injuries are encountered at the elbow, especially in overhand throwing athletes, golfers, and racquet sport athletes, when considerable stress is placed on these structures [42, 43]. Additionally, the shoulder is vulnerable to injury in nearly every sport with common soft tissue injuries involving the labrum and rotator cuff. MRI is the most appropriate study to evaluate the complex muscle-tendon units and ligamentous structures at both of the elbow and shoulder. Arthrographic technique at the elbow and shoulder will improve the diagnostic yield and should be considered when available, specifically in younger patients that may benefit from surgery. Additionally, radiologists can tailor MRI study protocols to evaluate for particular structures when provided with a clear history. For example, when evaluating distal biceps pathology, adding a FABS (Flexed elbow, ABducted shoulder, Supinated forearm) sequence to the MRI protocol will improve visualization of the distal tendon and subsequently provide greater diagnostic accuracy. At the shoulder, an ABER (ABduction and External Rotation) sequence can improve visualization of the anteroinferior labrum, which is a common location for labral injuries to occur. It should be noted that these sequences are not performed in every examination because MRI is not a quick process and each additional sequence extends an individual's time in the imaging bore.

Ultrasound is another alternative way to image the soft tissues of the shoulder. The MRI gives a more complete visualization of the labrum, but ultrasound can diagnose complete rotator cuff tears and even subtle partial thickness tears

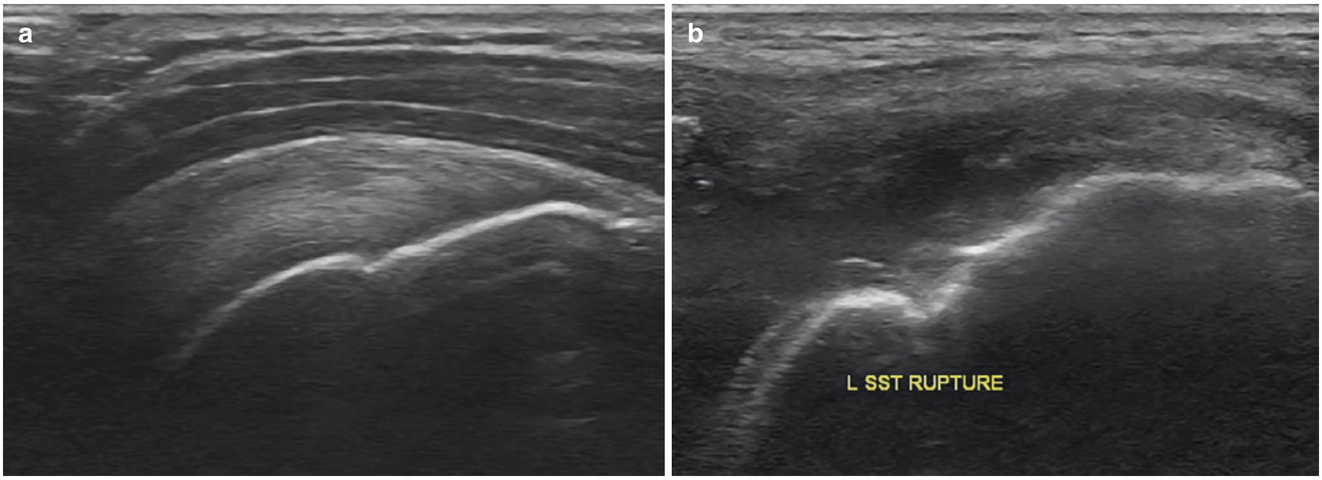


Fig. 6.14 Ultrasound images of normal insertion of the left supraspinatus tendon (SST) (**a**) and complete rupture of the right SST (**b**)

of the rotator cuff which are not always seen with MRI [44]. Examples of normal and complete full thickness rupture of the supraspinatus are seen in Fig. 6.14.

Clavicular fractures are quite common occurring with an incidence of 24.4 fractures per 100,000 person-years with sports activities involving 45% of these injuries, predominantly related to cycling, football, and soccer [45]. The decision regarding operative versus non-operative management for clavicular fractures is usually determined with radiographs only, noting that orthogonal views to better appreciate displacement and angulation will improve evaluation. ACJ injuries can be accurately assessed and graded using routine radiographs, which should be the initial study of choice. Stress views (weight-bearing) may improve the sensitivity of ligamentous disruption when the initial radiographs are normal, but are not usually indicated.

In addition to the acute sports-related trauma, a vast array of chronic injuries occur due to overuse in the athletic population with the most common locations including the elbow, shoulder, and wrist. At the elbow, radiographs may demonstrate osteochondral defects, abnormal ossification center fragmentation, or premature fusion at the physis in the pediatric population. When radiographs are non-diagnostic, MRI may demonstrate marrow edema at the apophyses of the elbow not seen on radiograph. Entities such as distal radial physeal stress syndrome (Gymnast's wrist) may demonstrate osseous resorption on radiograph, although they are more clearly delineated with MRI with periphyseal marrow edema. At the shoulder, MRI is helpful to diagnose internal impingement or posterosuperior impingement, which is seen almost exclusively in athletes who repetitively place their arms in extreme abduction and external rotation (swimming, volleyball, tennis, and throwing sports). Additionally, distal clavicular osteolysis may be seen by radiograph, although MRI may provide earlier recognition and distinction from similar entities such as acromial apophysiolysis [46].

Dynamic ultrasound imaging of the shoulder while the patient is actively moving the shoulder into 90° of abduction is an excellent way to assess either true impingement or relative impingement caused by poor posture and poor scapular mechanics [44].

Ribs, Chest, and Abdomen

Blunt trauma to the ribs, chest, and abdominal walls are encountered routinely during numerous sports, such as football, hockey, soccer, and winter sports. The vast majority of these can be treated without imaging; however, much depends on both the extent of injury and the level of play. At the professional, collegiate, and high-amateur settings, imaging with CT or MRI may establish a diagnosis that could guide a recovery and return-to-play timeline [47]. For a suspected rib fracture after minor trauma, a posterior-anterior (PA) chest radiograph is usually appropriate to diagnose any clinically relevant rib fracture; additional dedicated rib views may improve sensitivity for detection of nondisplaced or minimally displaced fractures; however, these often will not impact or change treatment [48, 49]. The most significant clinical impact from a rib fracture is usually the secondary complications, such as pneumothorax (Fig. 6.15), hemothorax, pulmonary contusion, or cardiovascular injury, which may be identified on the initial radiograph but would almost certainly require further investigation with cross-sectional imaging, namely, CT [50].

Ultrasound is also frequently used to diagnose pneumothorax. Sonographic signs, including "lung sliding," "B-lines" or "comet tail artifacts," "A-lines," and "the lung point sign," can help in the diagnosis of a pneumothorax. Ultrasound has a higher sensitivity than the traditional upright AP chest radiography (CXR) for the detection of a pneumothorax [51].

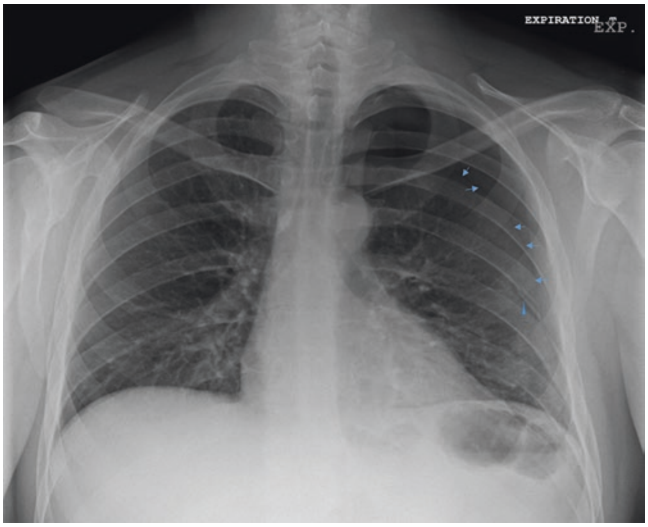


Fig. 6.15 Posterior lateral 6th rib fracture (arrowhead) and moderate pneumothorax (arrows) as a result of a tree strike while skiing in a 30-year-old male

Abdominal wall musculature injuries are often managed without imaging; however, high-performing athletes may benefit from an MRI to evaluate the extent of muscle or soft tissue injury and for the presence of hematoma. Although quite rare in the typical sports setting, intra-abdominal injury is possible after considerable blunt-force trauma and cross-sectional imaging with CT or ultrasound (FAST exam) would be the imaging modality of choice when concern for visceral pathology (see Chap. 42).

Head and Spine

Standard of care for imaging of head or spinal trauma uses cross-sectional modalities, including both thin-section multidetector CT and MRI, depending on the particular injury. Facial bone radiographs are still ordered; however, given the anatomic complexity of the region with overlapping osseous structures and decreased study volume experienced by most imaging centers and radiologists, this may lead to an increase in misinterpretation (see Chaps. 43 and 44). Therefore, CT imaging is preferred, which can now be obtained at most imaging centers in a time frame similar too or faster than the typical multiple radiographic projections previously performed in a facial bone series.

Head trauma is incredibly common in many different sports and across all levels of play with concussion serving as one of the most widely discussed topics regarding sports-related injuries. Currently, the primary role of imaging in the acute setting is to exclude an underlying more serious pathology such as hematoma or parenchymal contusion. Typically, non-contrast head CT is performed; however, providers are consistently faced with the difficult question regarding when to proceed to imaging in patients with minor or mild closed-head injury. The New Orleans Criteria (NOC), Canadian CT Head Rules (CCHR), and National Emergency X-Ray Utilization Study II (NEXUS-II) are published guidelines that can aid clinicians with these decisions [52]. MRI is usually not performed in the acute setting due to the time expense and limited additional information in patients with potentially serious injuries requiring rapid intervention. However, in the subacute or chronic setting, when neurologic symptoms persist or are unexplained by CT, MRI is useful to evaluate traumatic brain injury. Often, both CT and MRI are performed in conjunction and complementary to one another to assist in diagnosis. Advanced neuroimaging techniques including diffusion tensor imaging (DTI), functional MRI, MR spectroscopy, and nuclear medicine techniques (SPECT and PET) may play a role in evaluating chronic traumatic brain injury. Lastly, calvarial fractures after head trauma would benefit from CT imaging rather than MRI or radiography.

In 2009, Bailitz et al. demonstrated a drastic improvement in sensitivity for cervical spine injuries detected using CT for initial evaluation after trauma; radiography identified only 36% of the injuries diagnosed by CT, the majority of which were deemed significant [53]. Cervical spine trauma is quite common in sports-related injuries and similar to head trauma; there are two prominent guidelines published from large studies known as the National Emergency X-Ray Utilization Study (NEXUS) and the Canadian C-Spine Rule (CSR) [54]. Both incorporate several criteria that can be easily and relatively rapidly assessed to guide imaging choices. In many instances, both CT and MRI serve as complementary studies in the spine, with CT allowing for evaluation of the osseous structures and MRI providing information about the soft tissues, spinal canal compromise, and the spinal cord. If there is clinical or imaging concern for arterial injury, intravenous contrast with an arteriogram protocol is indicated.

In the pediatric population, specifically under the age of 14, studies have varied regarding the utility of radiography for initial evaluation after spine trauma. Younger children tend to experience different injuries than adults and adolescents leading to different imaging recommendations. Given the increased radiation doses of CT imaging, each patient should be treated individually regarding both clinical exam and injury mechanism when determining the most appropriate imaging modality; however, radiography may still play a role in diagnosis.

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Part II

General Synopsis of Acute Musculoskeletal Care in Sports

Morteza Khodaee

7 Anatomy and Physiology

Val Bratinov and Mark E. Lavallee

Key Points

- Bones, cartilage, tendons, ligaments, fascia, and joints are component of the musculoskeletal system.
- The musculoskeletal system has close interactions with neurologic and vascular systems.
- The Musculoskeletal system is responsible for human movement.
- Injury to musculoskeletal, neurologic, and vascular systems can cause significant physical and mental impairment.

The musculoskeletal system is comprised of bones, cartilage, tendons, ligaments, fascia, and joints. A major function of the skeletal system is to provide scaffolding for the rest of the body. Bones also serve a protective role for the vital organs. The brain, heart, and lungs are all encased in their own osseous confines like the skull and the thoracic cavity. Life is the pursuit of motion and cartilage, bones, tendons, ligaments, joints, and muscles interact together to generate movement.

V. Bratinov (✉)
UPMC Sports Medicine Fellowship Program, Pittsburgh, PA, USA
e-mail: bratinovvg@upmc.edu

M. E. Lavallee
York Sports Medicine Fellowship Program,
York, PA, USA

Pennsylvania State University College of Medicine,
Hershey, PA, USA

USA Weightlifting, Sports Medicine Society,
Colorado Springs, CO, USA

International Weightlifting Federation, Budapest, Hungary

York Hospital, York, PA, USA

Cartilage

Cartilage is a connective tissue that can be thought of as a matrix of extracellular fibers. Its composition varies based on the function that it performs. The main role of cartilage is to support soft tissue, to decrease friction over articulation surfaces, and to allow for the development of bone. The type of cartilage is divided into several subcategories known as hyaline, elastic, and fibrocartilage [1]. Classically, high friction areas of the body like the knee, responsible for caring the weight of the body during movement, have areas of cartilage that are high in collagen composition. This would be an example of hyaline cartilage as it is most often found around articulation surfaces of the bone. Areas of low force and friction, like the epiglottis and ears, require less collagen. This composition is known as elastic cartilage. Finally, fibrocartilage is comprised of few cells in a large extracellular milieu of fibrous tissue and collagen cartilage (Fig. 9.1). Such composition allows it to be flexible yet tough, making it integral to the structure of the meniscus and intervertebral disc.

Bones

Bone is a dynamic, living, connective tissue that comprises the skeleton. Its composition consists of calcified, mineral, matrix that contains a cellular matrix. This combination of mineral and organic structures allows bones to have properties of excellent mechanical strength while maintaining pliability and being lightweight. Bone provides support for structures in the body and protection for vital organs, serves as mineral reservoirs, acts as levers on which muscles pull, and produces hematopoiesis. To fulfill the specific function of various bones, the matrix composition is in a constant process of modeling and remodeling using cells called osteoblasts and osteoclasts. The body deposits increasing amount of bone over areas that experience high mechanical load using osteoblast activity. It removes bone mass, through a process known

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as resorption, from areas with low mechanical demand or in areas undergoing healing using osteoclastic activity. There exists excellent evidence suggesting that bone mass responds to mechanical load as seen during sports-specific training aimed at increasing muscle strength [2]. Likewise, inactivity and aging both contribute to resorption of bone mass in conditions such as osteopenia or osteoporosis.

Bones share a general anatomical composition. Cortical bone forms the dense outer body of bone that surrounds the spongy cancellous bony interior. Despite a large mineral composition in the cortical body, there is significant metabolic activity. Osteocytes, bone-forming cells, are in communication with osteoclasts, bone-resorbing cells, through a complex microscopic system of canals within the bone. The cancellous bone also contains bone marrow, which is the site for hematopoiesis. In the bone marrow, the body creates blood cells which comprise our red blood cells, white blood cells, and platelets.

Bones are frequently divided based on their shape. Long bones form cylindrical structures as found in the femur or humerus. Short bones are cuboidal like the individual bones of the ankle or wrist. Flat bones are spongy bone sandwiched between layers of compact bone and, for example, form the composition of the skull. Sesamoid bones are round bones that develop in tendons or muscles and serve the purpose of transmitting force by acting as a fulcrum. The largest sesamoid bone in the human body is the patella and acts as a pulley to transmit force from the thigh to the lower leg.

Joints

The connection between two osseous components of the skeletal system is known as a joint. Joints have both structural and functional classifications. Synovial joints, for instance, are skeletal elements separated by an articular cavity. Such joints allow for the largest range of motion. A second type of joint is known as a solid joint and is characterized by connective tissue holding together skeletal elements without the presence of a cavity. These joints exhibit significantly less movement than synovial joints. Examples of this joint type include cartilaginous joints which tend to have cartilage between bones. They allow for some degree of movement but are largely fixed. Ossification centers of developing bone or physes and the symphysis between separate bones, such as in the pelvis, are examples of cartilaginous joints. The least movable joints are fibrous joints. In the skull, adjacent bones are connected via suture ligaments. When bones are connected through an interosseous ligament, they create syndesmoses, such as in the tibiofibular joint.

Synovial Joints

Articular surfaces transmit large amount of force, especially during sports. Without lubrication between bony components, significant tensile and shear stress would result between adjacent articulating surfaces. As a result, the body has developed different ways to avoid bony surfaces from coming into direct contact. Freely movable joints with ample synovial fluid like the knee are known as synovial joints [3]. The joint space is composed of a joint capsule. Inside this capsule, a specialized tissue known as the synovial membrane produces a lubricating fluid called synovium. The fluid bathes the articular surface and provides the smooth sliding of the joints. Similar closed sacs of synovial fluid occur in other parts of the body, outside of joints, between other areas of high friction. Bursa sacs line the high force areas between joints and skin, tendon and joints, and skin and bones.

Tendons and Ligaments

The ability to transmit generated force from one part of the body to another is facilitated by tendons and ligaments. Tendons serve primarily to transfer the pull of muscles to bone, while ligaments connect one bone to another bone. They are composed of collagen fibers and can carry great tensile strength. Much like bone, tendons and ligaments once thought to be static structures are dynamic and capable of altering their composition and tensile strength. Healthy tendon and ligaments are composed mostly of parallel arrays of collagen fibers packed firmly together and separated by proteoglycan threads. Cell bodies are able to communicate with each other through gap junctions. This avenue for communication exists throughout the entire tendon and ligament structure, allowing for coordination of cellular response to mechanical loading. As collagen cells come together, they form fibrils. As fibrils combine, they form fibers. As fibers come together, they form fiber bundles. Finally, as bundles come together, they can make fascicles [4]. A tendon sheath runs along the exterior of fascicles, provides structure, and produces synovial fluid that bath internal structures of the tendon. Much like synovial joints, tendon sheaths prevent the tendon from adhering to itself or local tissue, thus ensuring a smooth lubricated movement.

Muscle

Muscles allow the body to convert chemical potential energy into mechanical energy to generate force and power. Muscles are contractile structures that are composed of a motor unit called a muscle fiber. There are actin and myosin chains that

allow the muscle to contract and relax. This is triggered by an action potential across the sarcolemma from the influx of calcium from the sarcoplasmic reticulum. There are three different muscle types.

Skeletal Muscle

The skeletal muscle forms the 40% of total body weight and contains as much as 75% of total body protein [5]. By working in an organized fashion, muscle constriction brings together bones which animate the skeleton, allowing the body to maintain posture and produce movement. The skeletal muscle is composed of large amounts of muscle fibers. Muscle fibers are composed of myofibrils organized in a strict linear fashion. These myofibrils are made up of the functional units of muscle, actin and myosin crosslinked filaments (Fig. 7.1). The actin and myosin are responsible for converting stored energy, ATP, into mechanical energy. As the functional units contract, they are able to work in parallel to generate the needed force to sustain a muscular contraction capable of movement [6]. The control of a synchronized contraction is voluntary and mediated by the somatic nervous system [7].

Cardiac Muscle

The cardiac muscle shares the same actin/myosin functional but lacks the overall strength and voluntary control as discussed with the skeletal muscle [8]. Its organization is branched and individual cells are electrically linked and produce coordinated contractures in the heart. Though the strength of these contractures is less than that of the skeletal muscle, the cardiac muscle is much less resistant to fatigue. The cardiac muscle is nonvoluntary and innervated by the visceral motor system [9].

Smooth Muscle

The smooth muscle is involuntary, non-striated, muscle capable of sustained contracture. The functional molecular structure is again actin/myosin; however, the myocytes are organized in a fusiform shape. Smooth muscle tissue has the ability to maintain contractility even in a stretched state. It is found in the walls of blood vessels and body organs [10]. The smooth muscle is nonvoluntary and innervated by the visceral motor system.

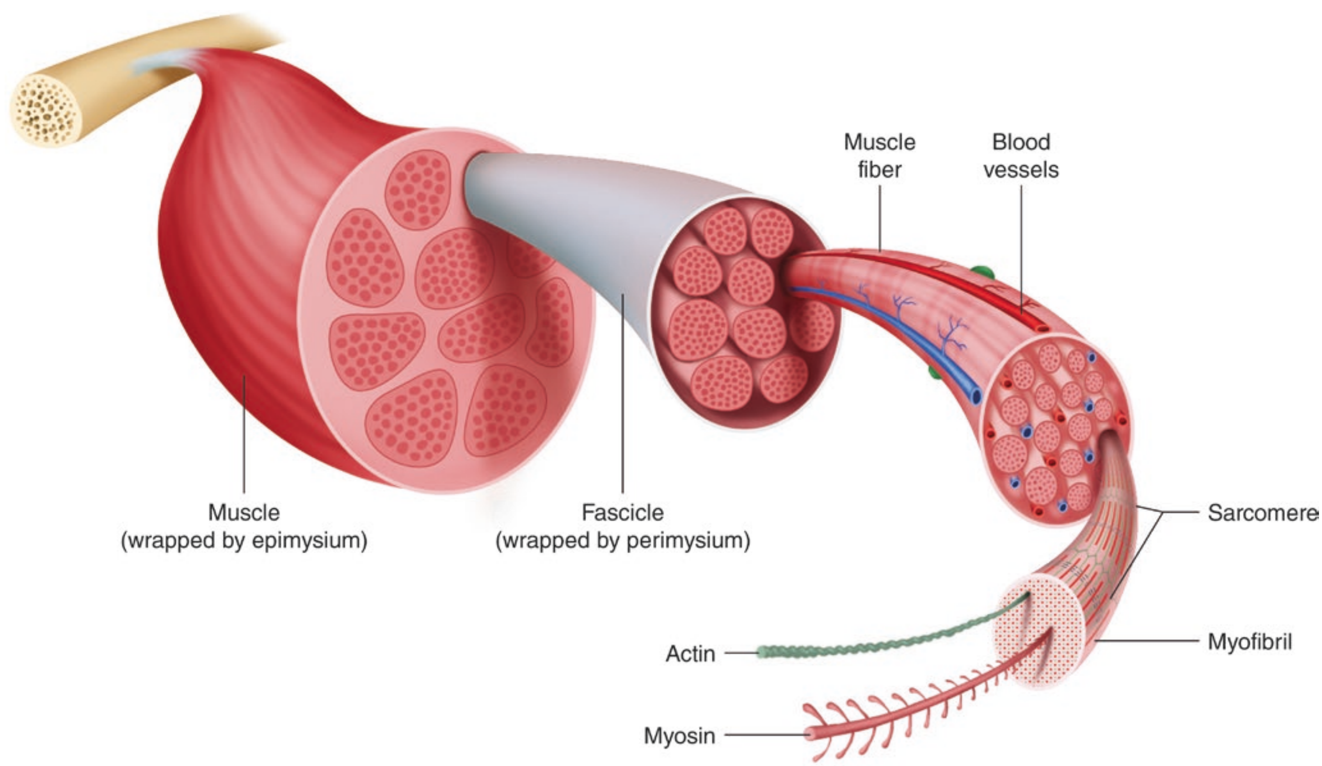

Fig. 7.1 The skeletal muscle divided into anatomical subunits. Muscle contraction, at the most basic level, occurs as a result of myosin movement against actin

Fascia

Fascia is a connective tissue that has grown in research interest. It is a thin layer of tissue surrounding muscles, bones, organs, nerves, and blood vessels. It separates, compartmentalizes, and provides continuity of tissues and organs allowing for movement of structural groups based on their anatomical function. Fascia promotes sliding, uptakes friction, and distributes force during motion of these functional groups. Fascicular fascia provides force transmission and proprioceptive feedback to muscles for movement and control. This type of fascia also maintains protection for nerves and vessels during forceful muscle contraction by forming intramuscular, extramuscular, and neurovascular sheaths. Such compartmentalization also plays an important role in limiting the spread of infection [11].

Neurologic System

The nervous system is composed of three divisions, the central nervous system which composes the brain and spinal cord, the peripheral nervous system which compromises the voluntary nerves located outside the spinal column, and lastly the autonomic or involuntary nervous system which regulates essential body function like breathing, digestion, and heart rate.

The central nervous system is comprised of two essential organs, the brain and the spinal cord. The brain is the central processing unit for the human body. The brain processes conscious and unconscious thought, voluntary and involuntary motions, emotions, memory, and all bodily function. The spinal cord is the main connector between the brain and the rest of the body. Major injury to the central nervous system is often permanent as it lacks prompt regenerative properties. In the realm of sports-related trauma, injuries to either of these two organs can cause a range of disorders like anxiety, depression, concussions, spinal shock, or even traumatic brain injuries.

The peripheral nervous system is the part of the somatic nervous system that controls voluntary action. It can vary in function, working in conjunction with the skeletal muscle, to cause the body to run, jump, breath, or even blink. It also acts as the reflex center by allowing the body to respond rapidly to specific sensory inputs with almost automatic motor responses. An example of the reflex arch is the patellar reflex which is activated by striking the patellar tendon. Unlike the central nervous system, the peripheral nervous system can regenerate itself but only under certain conditions.

Vascular System

Arteries and veins are part of the circulatory system which is responsible for the delivery of oxygen and nutrients to cells and the removal of carbon dioxide and waste products from cells. Arteries carry oxygenated blood away from the heart and veins carry blood back toward the heart. Arteries carry blood being pumped from the heart and are thus equipped to handle high pressure. Their composition consists of an outer layer made primarily of connective tissue, and middle layer made primarily of smooth muscle and elastic cells, and an inner layer made primarily of thin cells capable of nutrient and gas permeability. Based on systemic demands, the cross-sectional size of arteries can change based on smooth muscle stimulation [12]. These properties play important roles in systemic vascular resistance and blood pressure.

Veins carry deoxygenated blood from tissues back toward the heart. Their composition is similar to arteries, except that their middle layer contains much less smooth muscle. Venous blood is of much lower pressure and the venous system does not function in a contractile manner like the arterial counterpart. Veins, because they are a low-pressure system, also have valves to prevent backflow of blood.

Lymphatic system, though not necessarily part of the vascular system, is another low-pressure system that carries lymph around the body to help fight infection. This system can be injured during athletic trauma causing lymphedema or Morel-Lavallée lesion.

In the realm of sports trauma, injury to these vascular structures can cause acute blood loss, placing athletes at risk for hemorrhagic shock or even death. Being able to monitor and control bleeding during traumatic injury is essential for this reason. In terms of healing, the vascular system again plays a vital role as it transports nutrients, clotting factors, progenitor cells, and other essential components to injured tissue.

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8 Fracture Types and Definitions

David Cole, Joseph Medellin, Ryan C. Wennell, and Mark E. Lavallee

Key Points

- Fractures are common in sports.
- A history and physical examination including a vascular and neurologic assessment will always be primary in identifying the level of acuity of care required in fracture situations.
- Classification of fractures is important to direct management and risk stratify healing as well as long-term outcomes.
- Pediatric fractures can and will vary from those of adults.
- Identification of pathologic fractures can be discovered with a thorough clinical history and examination.

Introduction

Bone is essentially a series of interwoven strands of type I collagen. Compact or cortical bone is comprised of densely packed strands of collagen surrounding a vascular canal and is the primary bone type of adults. Sponge or trabecular bone is more disorganized and contains more space between the strands of type I collagen. Sponge bone is more immature and there is more of it present in children. Bones may also be separated by axial (skull, vertebrae, ribs, and sternum) versus appendicular (limbs, pelvis) and further divided by shape (flat, tube [long, short], irregular, sesamoid, and accessory).

Fractures may occur at any one or more of any of the locations of the bony structures that make up the human skeleton. The purpose of this chapter is to discuss the various classifications of fractures that are based on distinct characteristics such as the anatomic site, the visual appearance (open versus closed), the radiographic appearance (e.g., displaced, compressed, fragmented), and the positioning and alignment of fragments if present.

Fracture Descriptions

Anatomical Sites of Fractures

Long bones are divided into different anatomical areas (Fig. 8.1) and fractures can occur in one or multiple of these sites. The long bone shaft is called the diaphysis with each end called an epiphysis, while an area between the epiphysis and diaphysis, the neck in long bones, is called the metaphysis. In children there is the presence of growth plates that are called physis that is part of the metaphysis, and in adulthood the area is referred to as the epiphyseal line. Bones also develop areas of protuberance or outgrowth called apophysis that will serve as tendon and ligament attachment sites. Fractures may also extend into the joint surface and are referred to as being intra-articular.

D. Cole
Department of Primary Care/Sports Medicine, One Medical, San Francisco, CA, USA

J. Medellin
Department of Sports Medicine, Henry Ford Allegiance Health, Jackson, MI, USA

R. C. Wennell (✉)
Sports Medicine, Wellspan York Hospital, York, PA, USA

M. E. Lavallee
York Sports Medicine Fellowship Program, York, PA, USA

Pennsylvania State University College of Medicine, Hershey, PA, USA

USA Weightlifting, Sports Medicine Society, Colorado Springs, CO, USA

International Weightlifting Federation, Budapest, Hungary

York Hospital, York, PA, USA

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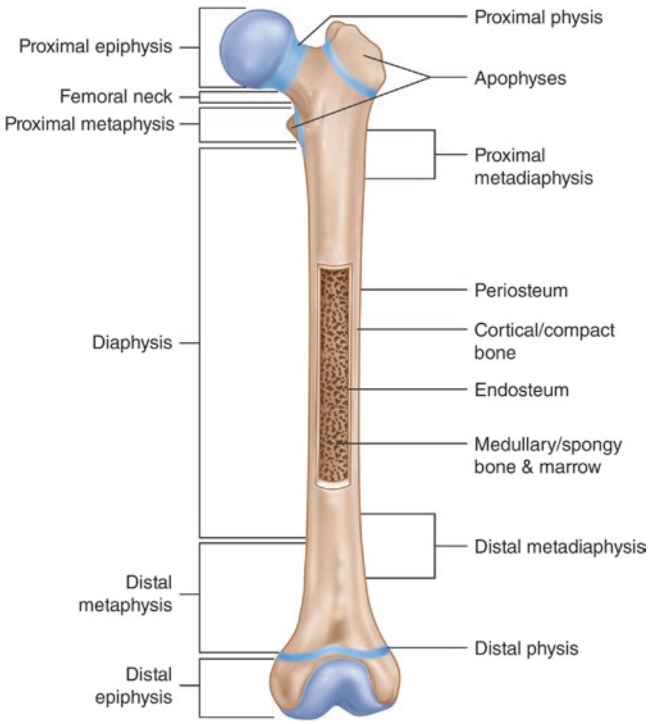

Fig. 8.1 Schematic anatomy of long bones

Fig. 8.2 Intra-articular patellar fracture causing significant joint effusion/hemarthrosis (arrows) and pre-patellar soft tissue swelling/hemorrhagic pre-patellar bursitis (arrowhead) in a 46-year-old female as a result of a direct fall on ice

Fracture Correlation with Joints

An articular fracture involves the joint surface and is typically described in relation to the percentage of the joint space that is disrupted [1]. Intra-articular fractures are a bone fracture that crosses into the joint surface. Hemarthrosis (Fig. 8.2), bleeding into the joint space, can be a consequence of intra-articular fractures. Hemarthrosis will present with moderate to severe joint effusion within hours of the traumatic injury or inciting event and can be evaluated by physical examination, x-ray, ultrasound, and MRI [2, 3].

Intra-articular fractures will always result in damage to cartilage and have a high risk of subsequent development of arthritis [4, 5]. Posttraumatic osteoarthritis is a result of the initial impact to the cartilage, combined with the ensuing pathomechanical and pathobiological response of the cartilage [4]. Treatment for intra-articular fractures is focused on restoring joint surface congruity to avoid chronically elevated articular surface contact stresses and/or instability usually achieved by surgical intervention [4]. Extra-articular fractures are bone fractures that occur near but outside a joint space [6].

Fracture Types

There are a variety of terms that can be used to describe the types of fractures that occur. Fractures that circumferentially traverse the shaft of bone perpendicular to the axis are transverse fractures and can be with or without displacement [6]. Spiral fractures are produced by rotational or twisting forces, while compression fractures are crumpled or collapsed bone injuries caused by a traumatic force usually due to loss of bone mass. A comminuted fracture involves an injury where the bone is fragmented into two or more pieces. An avulsion fracture is one in which a tendon or ligament tears off a fragment from the main bone.

Osteochondritis dissecans (OCD) refers to a fracture that occurs in the pediatric population due to repetitive stress causing osteonecrosis of the bone and ultimately a subchondral stress fracture. Most common location is the knee medial femoral condyle [7].

Fracture Displacement

Fracture displacements refer to the position of the fractured bone in relation to the normal anatomic configuration. Fractures without any abnormal anatomic configuration are nondisplaced. Displaced fractures show a separation of the fragment(s) with a loss of anatomic position of the distal fragment with respect to the proximal fragment. The amount and direction of positioning from the anatomic position are described as direction of translation, the amount of which should be reported [1]. Minimal displacement refers to anatomic anomalies of 3 mm or less of translation. Angulations of bone may also occur concomitantly with the fracture and are areas of bending or angle anomaly that differ from normal anatomic position. Bone length changes may occur in instances of impacted, compression, or bayonet-type apposition (side-by-side position as opposed to end-to-end). "Dimpling sign" (Fig. 8.3) can be observed as a result of such and healing may be disrupted depending on the amount of shortening and what may be considered acceptable based on the deformity [1]. Rotational displacement (Fig. 8.4) is often only detected clinically and is dependent on the type of trauma sustained, gravity, and muscular spasms.

Open Versus Closed Fractures

Closed fractures indicate a fracture area that does not visibly communicate externally to the body (e.g., intact skin pattern). Open fractures communicate externally to the body and the break in the skin pattern may be very subtle (Fig. 8.5) or an obvious open wound (Fig. 8.6). Open fractures are an urgent situation and evaluation should commence following primary survey and resuscitation protocols in an emergency department.

Pediatric Considerations

Greenstick Fracture

The large amount of immature, biologically active bone in the pediatric population makes for higher rate of bowing or

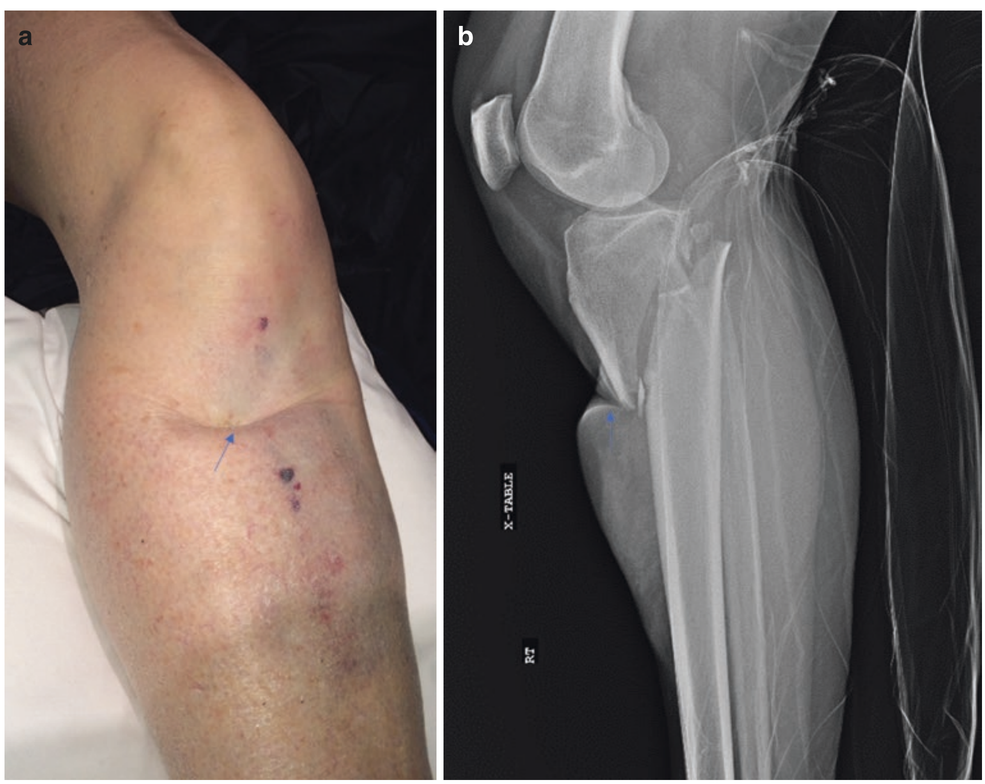

Fig. 8.3 Dimple sign (arrow) as a result of a significantly displaced and shortened tibial fracture in a 67-year-old female as a result of a ski injury (**a**, **b**)

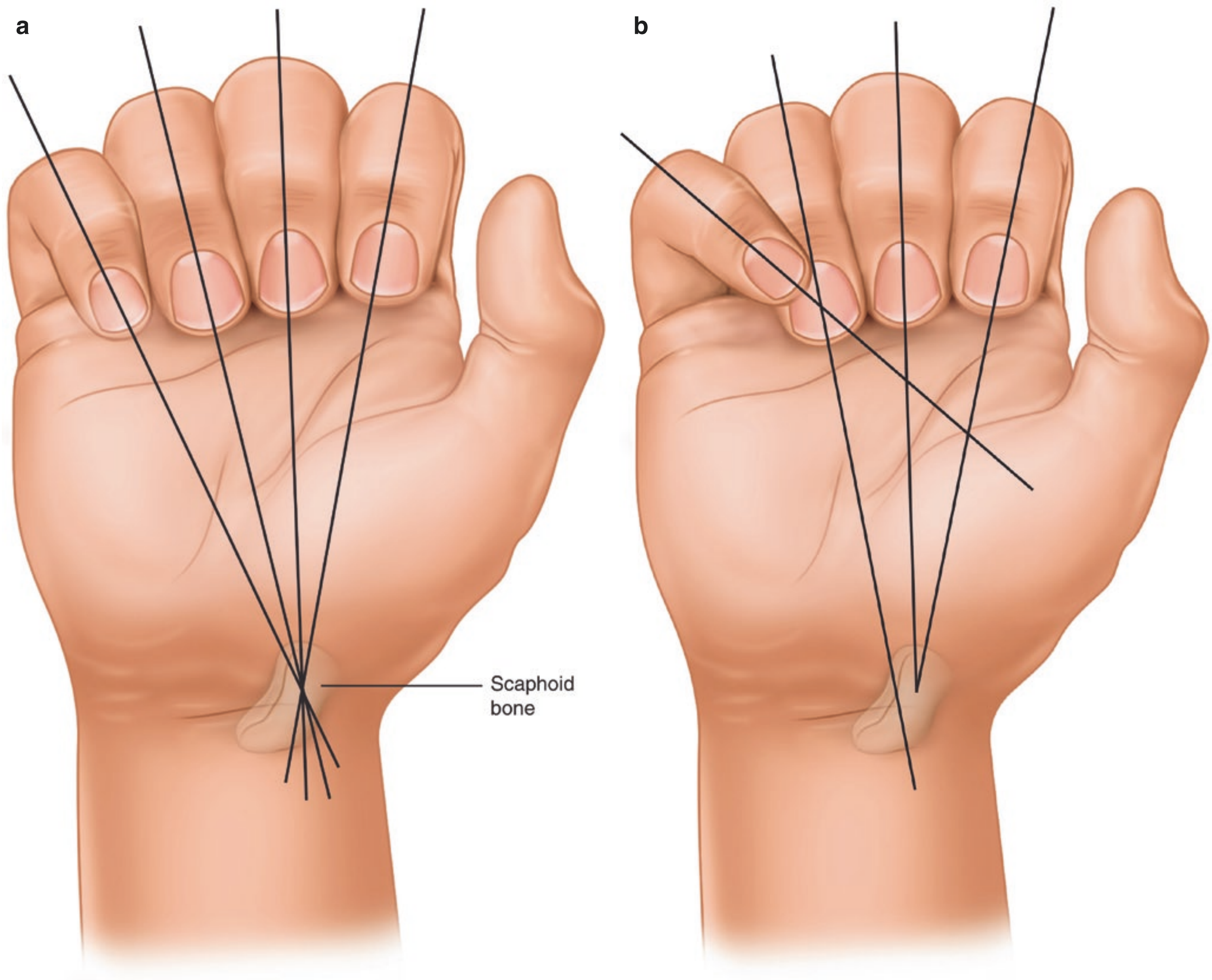

Fig. 8.4 Normally, all fingers should line up on the same location at scaphoid (**a**). Rotational displacement of the little finger is present (**b**)

bending, and a greenstick fracture (Fig. 8.7) may occur. The fracture is a portion of the bone on one side, while the opposing side of the same bone will have a buckled or bowed appearance. The fracture itself does not cross through the width of the bone. These typically occur in the long bones and require immobilization and casting as quickly as possible following confirmation.

Torus (Buckle) Fracture

Torus or buckle fractures refer to compression injury of the cortex that occurs in the region between the metaphysis and diaphysis (Fig. 8.8). There may also be involvement of the periosteum that can be dependent upon the type of trauma and the amount of force exerted. They are considered stable fractures and are usually treated with splinting.

Physeal Fracture

Physeal (growth plate) fractures are the most common fractures to occur in children. Their involvement in injury could result in abnormal growth patterns and often require close monitoring depending on the type and location of the fracture. Premature closure of the growth plate causing asymmetrical limb shortening and stimulation of longitudinal growth causing asymmetrical limb lengthening are both possible occurrences.

The Salter-Harris classification has become and continues to be the most widely used system for describing physeal

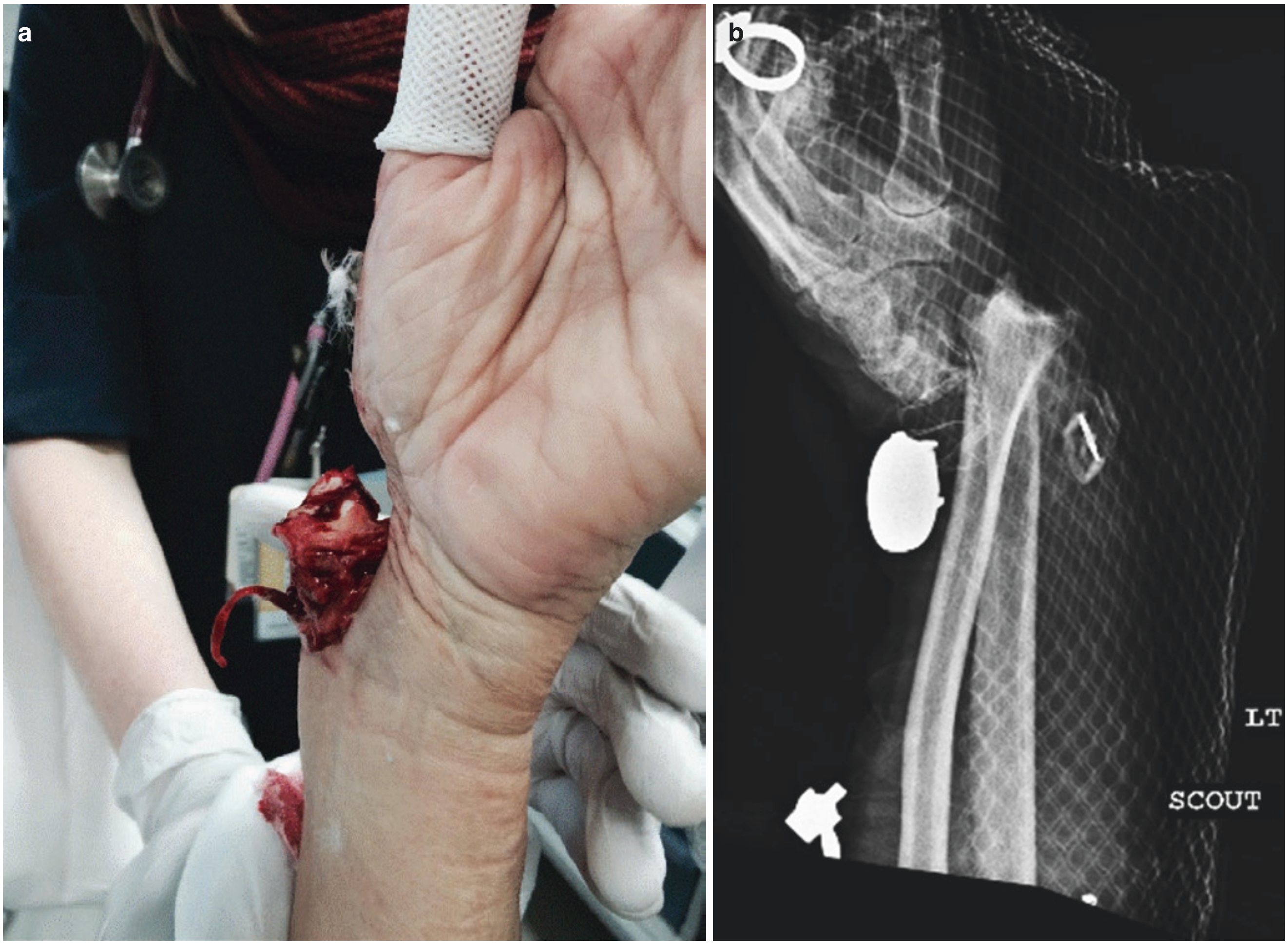

Fig. 8.5 Open fracture/dislocation of the distal radius in a 71-year-old female as a result of a fall while skiing (**a**, **b**)

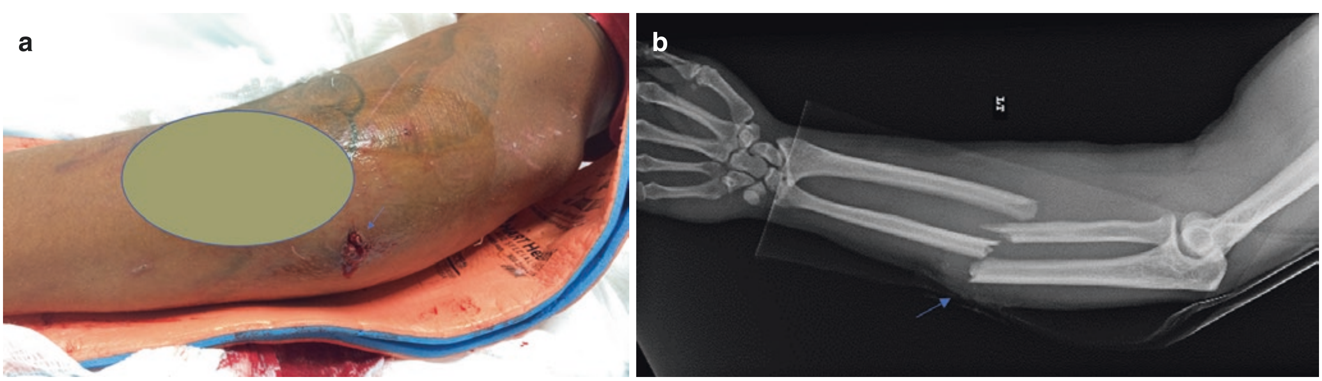

Fig. 8.6 Radius and ulna shafts fracture with component of an open ulna fracture (arrows) in a 28-year-old male as a result of a fall while mountain biking (**a**, **b**)

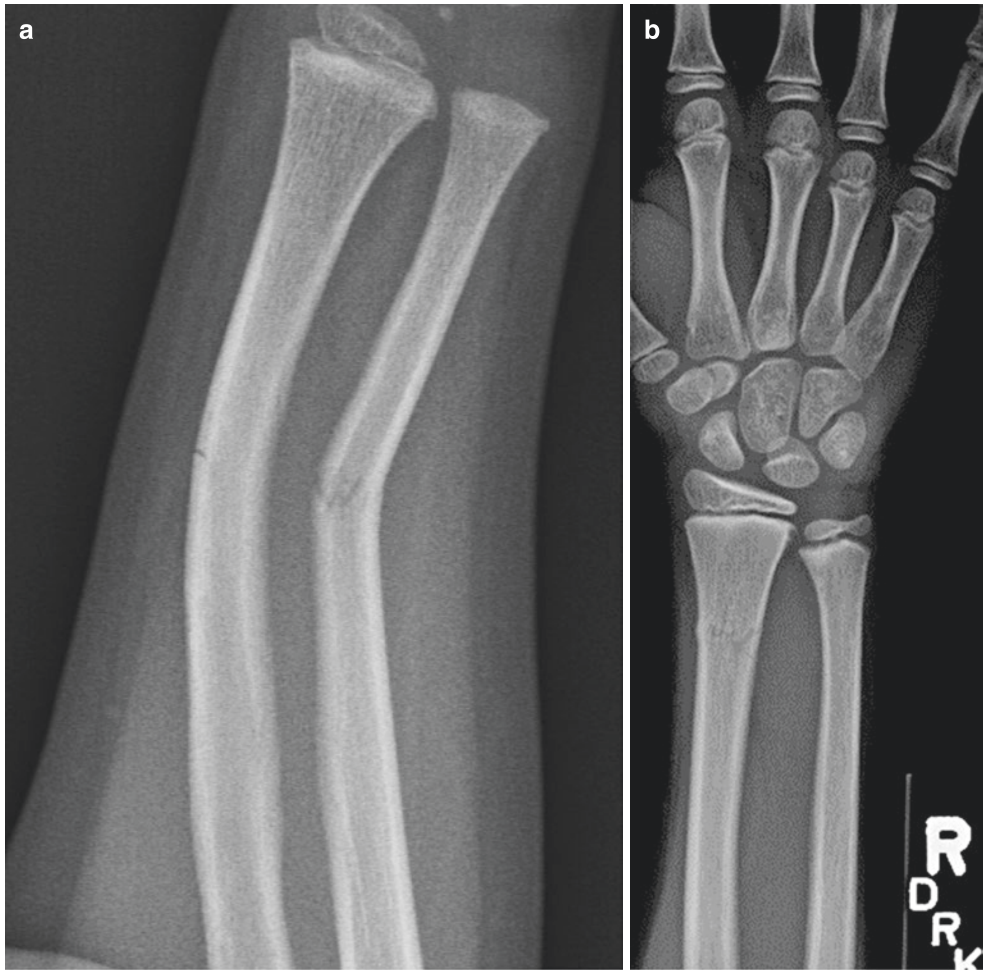

Fig. 8.7 Greenstick fracture of the radius and transverse fracture of the ulna in a 3-year-old girl (**a**). Greenstick fracture of the distal radius in a 9-year-old boy (**b**)

fractures during childhood (Fig. 8.9). Salter and Harris developed a classification of fractures as they relate to the growth plate in children in 1963 [8]. There have been nine types described; however, only the first five are typically used. Type I accounts for approximately 5% [9] of injuries and is a fracture that passes through the physis alone and does not involve either the metaphysis or the epiphysis. Type II accounts for approximately 75% [9] of fractures and is one that passes through both the physis and metaphysis. Type III accounts for 10% [9] and involves the physis and the epiphysis. Type IV accounts for 10% [9] of fractures and involves the physis, metaphysis, and epiphysis. Type V accounts for 1% [9] or less of fractures. Type V is described as a crush or collapsing injury that results in a nondisplaced compression of the physis.

In general, the risk of abnormal growth patterns increases with the increase in number. Type II fractures tend to vary the most with respect to healing and growth and are extremely dependent on the anatomic location and the amount of the physis involved. Poorer growth outcomes are almost universal with type IV due to the involvement of each portion of the bone and frequent occurrence of comminution.

Pathologic Fractures

These are fractures that tend to occur in individuals who have a bone structure that is weakened or altered by a local or systemic process. These processes can be benign in origin including history of previous fracture, prolonged immobilization, cysts (Fig. 8.10), tumor, osteopenia/osteoporosis, and congenital diseases like osteogenesis imperfecta. They may also be the result of more serious conditions that may be first recognized following a fracture including infections, sarcomas, metastatic disease, and leukemias/lymphomas.

Fig. 8.8 Torus (buckle) fracture of the distal radius (arrow) in a 10-year-old female

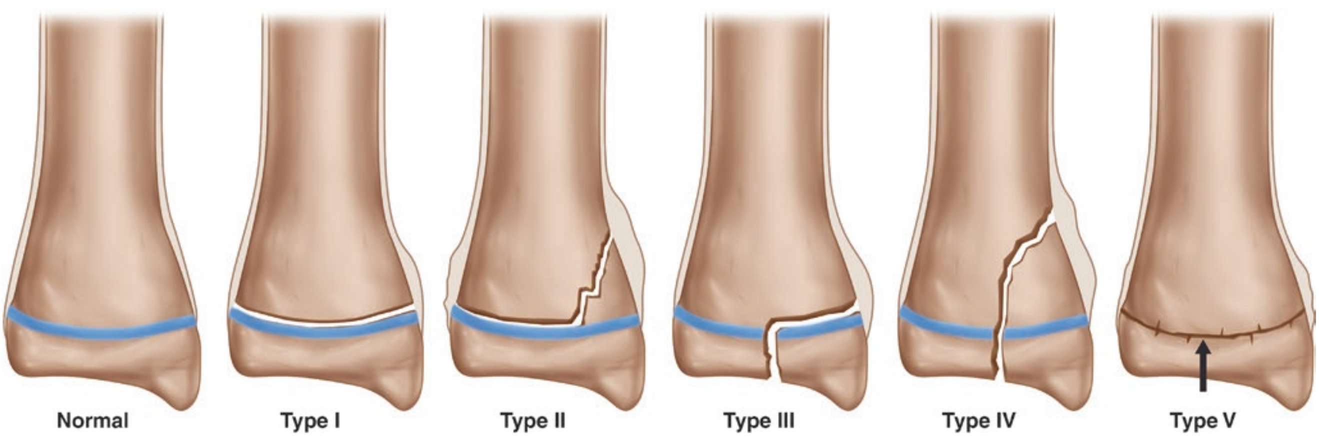

Fig. 8.9 Salter-Harris physeal fracture classification [8]

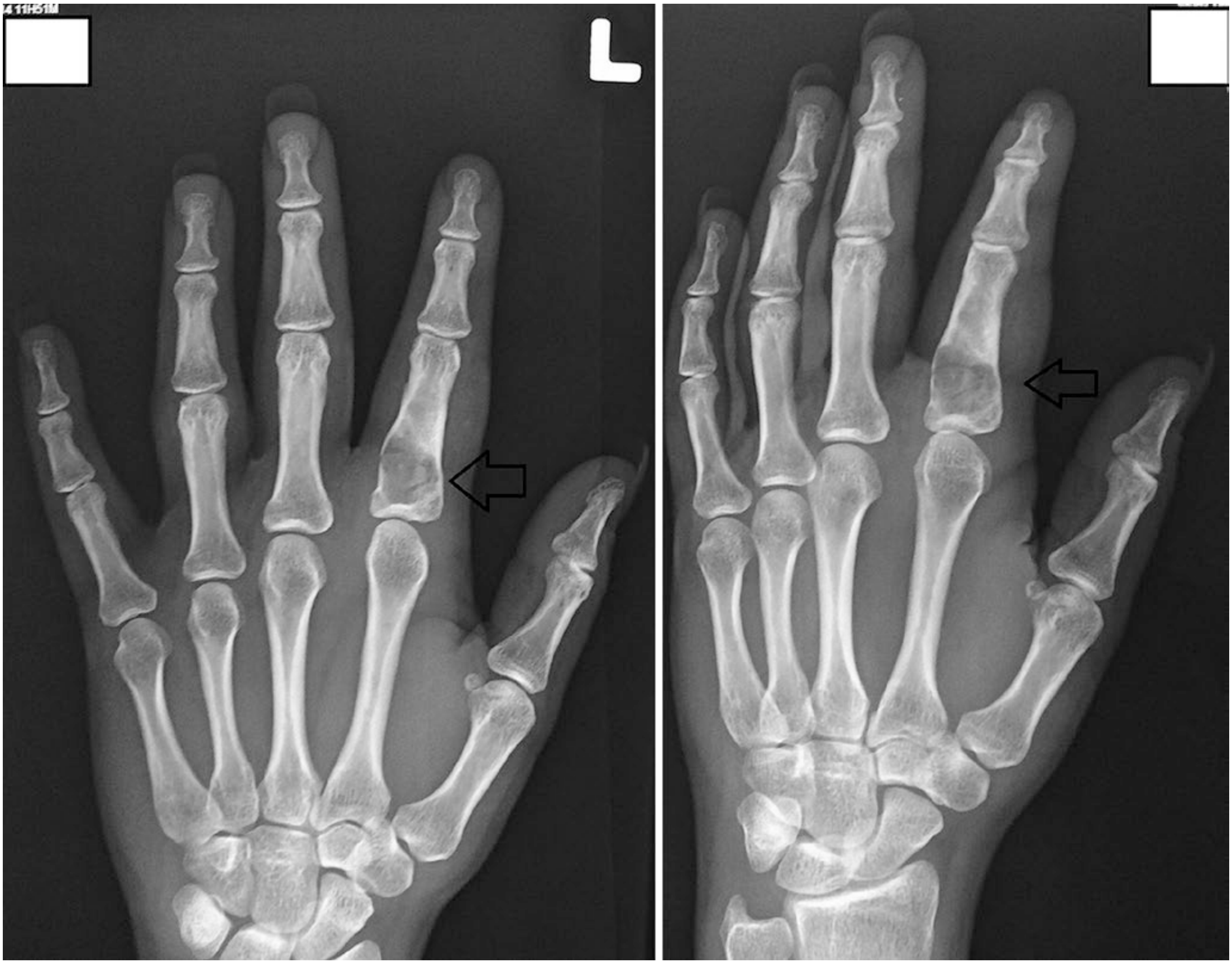

Fig. 8.10 Pathologic (underlying bone cyst) fracture (arrows) at the 2nd proximal phalanx in a 28-year-old female as a result of an injury while playing football in her backyard when the football hit her index finger

In children, the most common pathologic fracture is due to benign tumors including unicameral or aneurysmal bone cysts and nonossifying fibromas [10, 11]. Fractures that may be due to an underlying pathology should always include a thorough evaluation and work-up and a differential should always consider the female athlete triad when appropriate [12].

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Dislocation Types and Definitions

9

Katherine M. Edenfield and Jocelyn R. Gravlee

Key Points

- Joint dislocations are relatively common in sports.
- Taking a comprehensive history of the injury and a focused physical examination including neurovascular status is recommended.
- An attempt to reduce a dislocated joint should be considered depending on the specific joint, concomitant injuries, and the skill level of the provider.
- Follow-up radiography should be performed to confirm the reduction.

Introduction

Joints, or articulations, join bone to bone, bone to cartilage, or teeth to boney sockets. Some of these articulations allow mobility; however, they are considered the weakest points of the skeleton.

There are two different joint classifications. The functional classification is defined by the degree of mobility where synarthroses are immovable joints, amphiarthroses are joints that have some movement, and diarthroses are freely moveable joints. The structural classification is defined by the material that binds them and includes fibrous, cartilaginous, and synovial joints.

Fibrous joints include sutures, syndesmoses, and gomphoses (Fig. 9.1).

- *Sutures* are seen in the skull where bones are tightly bound by a minimal amount of fibrous tissues to allow growth as a child and ossify in adulthood. This joint is immoveable.
- *Syndesmoses* join bones by ligamentous and fibrous tissue and can be of varying sizes in the body. The anterior tibiofibular ligament connecting the distal tibia and fibula is short allowing minimal movement, whereas the interosseous tissue connecting the radius and ulna is long and forms a freely moveable joint (dysarthrosis) between these bones as the radius rotates around the ulna (supination and pronation).
- *Gomphoses* are seen only in the mouth where a peg-in-socket joint is created by the tooth and bone and connected by the periodontal ligament.

Cartilaginous joints include synchondroses and symphyses (Fig. 9.2).

- *Synchondroses* are immovable joints and include epiphyseal plates where hyaline cartilage joins bones and costocartilage joins the first rib and the sternum.
- *Symphyses* are slightly moveable joints. Examples include intervertebral discs and the pubic symphysis where fibrocartilage joins bones. Hyaline cartilage lines the ends of bones to form articular cartilage in this joint, but it is not the main structural component of the symphysis.

Synovial joints are all diathroses and make up the majority of joints in the body. These joints consist of the following:

- Articular cartilage lines the ends of opposing bones
- Articular capsule consists of a fibrous outer capsule that provides strength to the joint and the inner synovial membrane that produces synovial fluid
- Synovial cavity is unique to a synovial joint

K. M. Edenfield (✉) · J. R. Gravlee
Department of Community Health and Family Medicine, University of Florida College of Medicine, Gainesville, FL, USA
e-mail: kedenfield@ufl.edu

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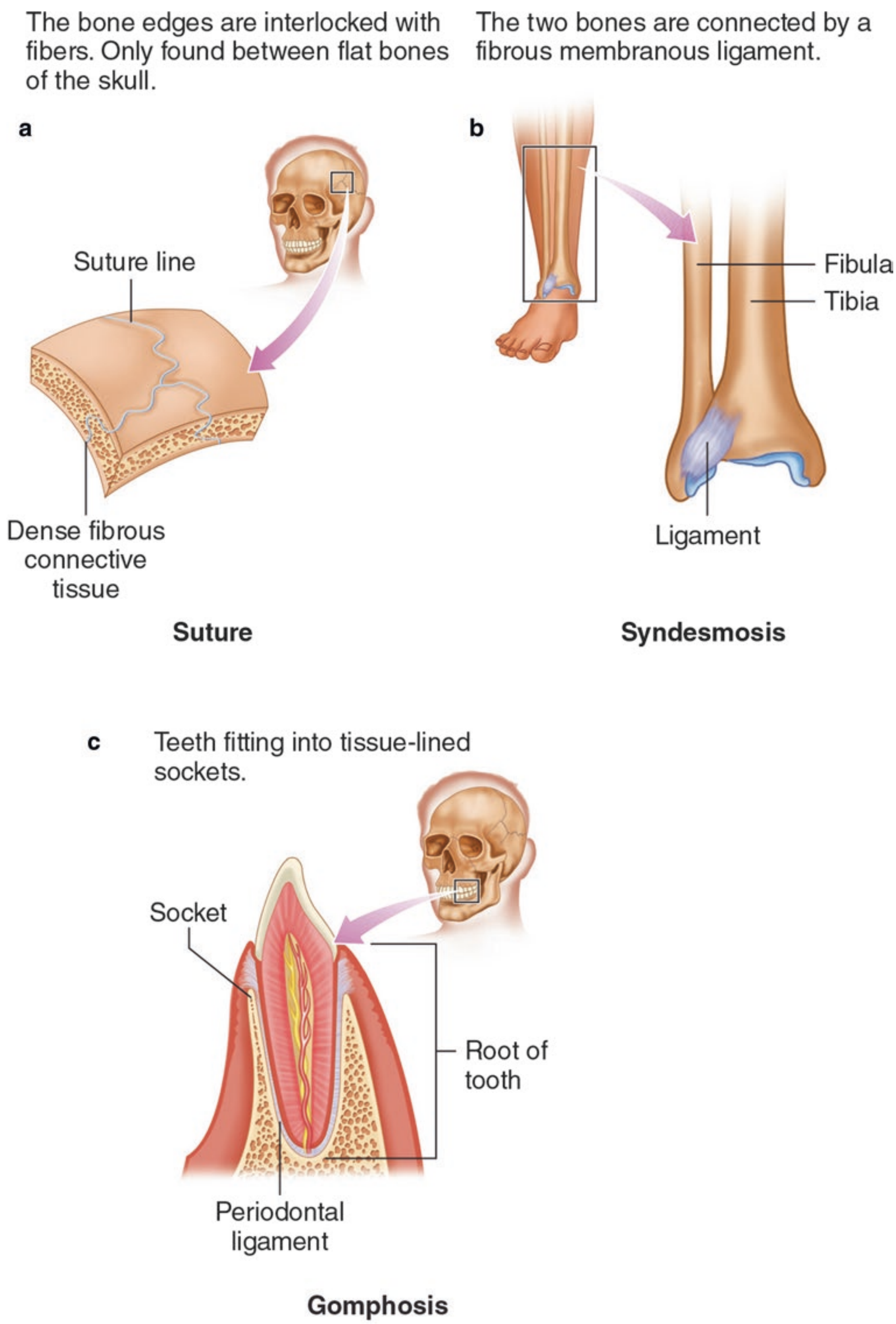

Fig. 9.1 Types of fibrous joints

- Synovial fluid is the viscous filtrate of blood that lubricates joints and provides nutrients to the articular cartilage

Simple synovial joints have two articulating surfaces

Compound synovial joints have more than two articulating surfaces such as the elbow.

Classification of Synovial Joints by Shape (Fig. 9.3)

- Plane joint – flat plane, gliding movements such as intertarsal or intercarpal joints
- Hinge joint – the cylindrical end of one bone fits into a trough-shaped surface of the other such as the ankle and the elbow
- Pivot joint – the rounded end of one bone fits into a ring-shaped opposing bone such as the atlas and dens of the axis

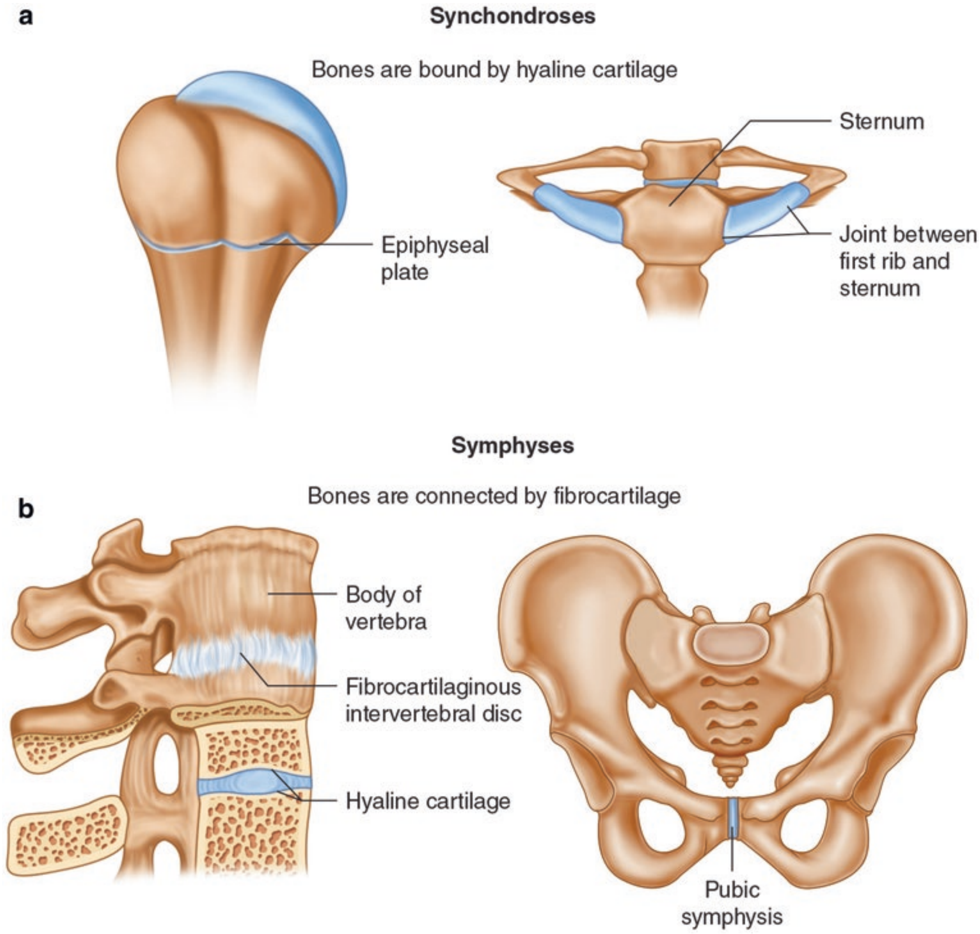

Fig. 9.2 Types of cartilaginous joints

- Condyloid joint – the convex surface of one bone fits into the concavity of the opposing bone such as the metacarpophalangeal joints
- Saddle joint – each joint surface has both a convex and concave areas seen in the 1st carpometacarpal joint
- Ball and socket joint – the spherical head of one bone fits into the socket of the other as seen in the shoulder and hip joints

Stability of a synovial joint depends on the following:

1. *Shape of the articular surface*
   The deep ball and socket joint of the hip provides static stability; however, most joint sockets are too shallow to provide adequate stability. In some joints such as glenohumeral and hip joints, there is an extra cartilaginous rim (labrum) which can increase the stability.
2. *Ligaments*
   Ligaments and the joint capsule provide stability to a joint; however, when ligaments are stretched after excessive forces are applied, they tend to stay stretched contributing to joint instability.
3. *Muscle tone*
   Muscle tone provides stability by exerting a constant contractile force on tendons across joints outside the joint capsule. The muscle tone of the rotator cuff is critical in providing stability at the shoulder.

Definitions

- *Dislocation:* complete separation of two joint surfaces where relocation (reduction) is needed to restore the normal anatomy.
- *Subluxation:* partial separation of two joint surfaces.
- *Joint laxity:* asymptomatic movement of a joint at the upper end of normal physiological range. This could be idiopathic or acquired from repetitive use such as overhead athletes.
- *Joint instability:* abnormal movement of a joint resulting in dislocation or subluxation and some degree of pain.

Dislocations make up about 3.6% of all high school athletic injuries [1]. When a joint dislocation occurs, supporting structures stretch, tear, or avulse and may heal with abnormal alignment of the joint or fail to heal all together, resulting in instability and an increased risk of recurrent dislocation or subluxation. Patients with inherent laxity of their joints can develop

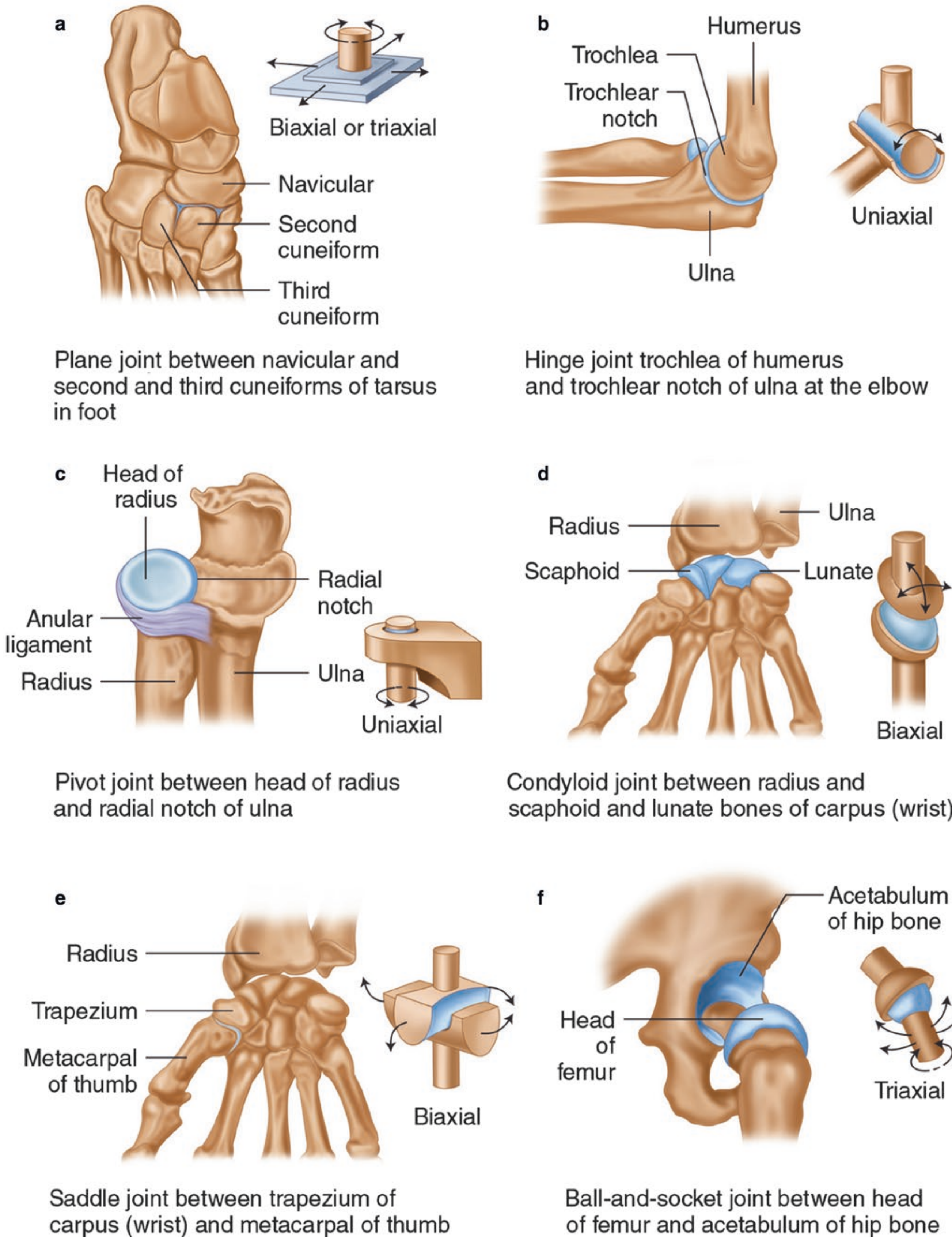

Fig. 9.3 Types of synovial joints

microscopic tissue damage from low-level forces applied across the joint resulting in instability. Other risk factors for joint dislocation include younger age [2, 3], male gender (for anterior shoulder and finger dislocations) [4, 5], and contact sports.

Joint Hypermobility Syndrome (JHS)

It would be prudent to consider hypermobility syndrome in a patient with recurrent dislocations. Based on 1998 diagnostic criteria (*Brighton* criteria), JHS is diagnosed in the presence of the two major criteria, one major and two minor criteria, or four minor criteria [6]. Two minor criteria suffice when there is an unequivocally affected first-degree relative. JHS is excluded by the presence of Marfan or Ehlers-Danlos syndromes (Table 9.1).

Major criteria:

- A Beighton score of ≥4 out of 9 (currently or historically)
- Arthralgia for longer than 3 months in four or more joints

Minor criteria:

- A Beighton score of 1, 2, or 3 out of 9 (or 0 if at least 50 years of age) (the major and minor criteria using the Beighton score are mutually exclusive).
- At least 3 months of arthralgia in one to three joints or back pain or spondylosis or spondylolysis/spondylolisthesis (the major criterion of arthralgia and this criterion are mutually exclusive).
- Dislocation/subluxation in more than one joint or in one joint on more than one occasion.
- Three or more traumatic or overuse injuries, such as epicondylitis, tenosynovitis, or bursitis).
- Marfanoid habitus.
- Abnormal skin: striae, hyperextensibility, thin skin, and papyraceous (or papyrus-like) scarring. The soft and silky texture of the skin and the ability to stretch it more than might be found in a non-hypermobile person is qualitative. This is best assessed by pulling the skin upward on the dorsum of the hand or over the olecranon at the elbow.
- Eye signs: drooping eyelids, myopia, or “antimongoloid” slant (i.e., a lateral downward slant or lateral canthal dystopia).
- Varicose veins, hernia, or uterine/rectal prolapse.

Table 9.1 Beighton scoring system for hypermobility [7]

Clinical maneuvers performed and scored as 0 or 1. A summative score of ≥4 suggests hypermobility. The examination is performed bilaterally (Fig. 9.4)
Little finger: passive dorsiflexion >90°
Thumb: passive dorsiflexion to make contact with the flexor side of forearm
Elbow: hyperextension >10°
Knee: hyperextension >10°
Trunk: from a standing position, forward flexion with straight knees allows the palms of both hands to rest on the floor

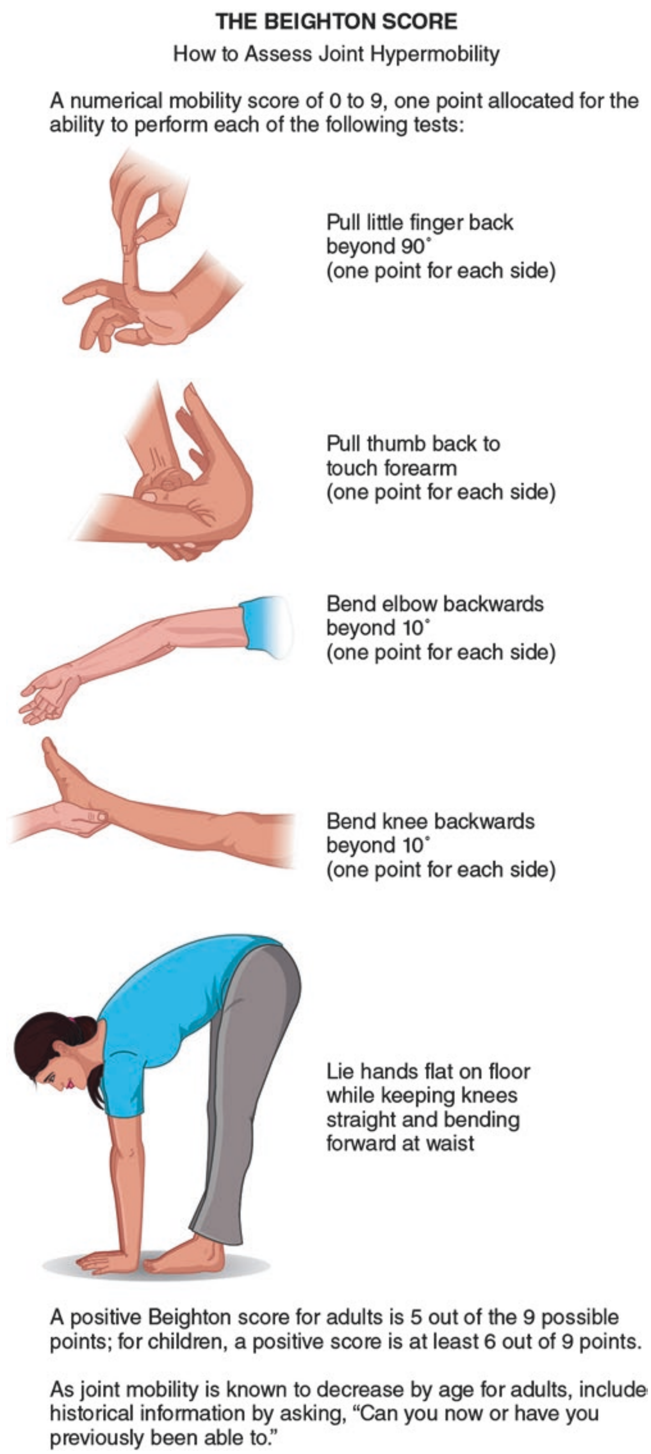

Fig. 9.4 Five clinical maneuvers performed to calculate Beighton score

Dislocation Description

It is important to accurately identify the direction of the dislocation before attempting relocation (reduction) and be able to communicate the position of the related structures to a consultant if referral is needed.

A dislocation is described by noting the distal bone’s position in relation to the proximal bone. This position can

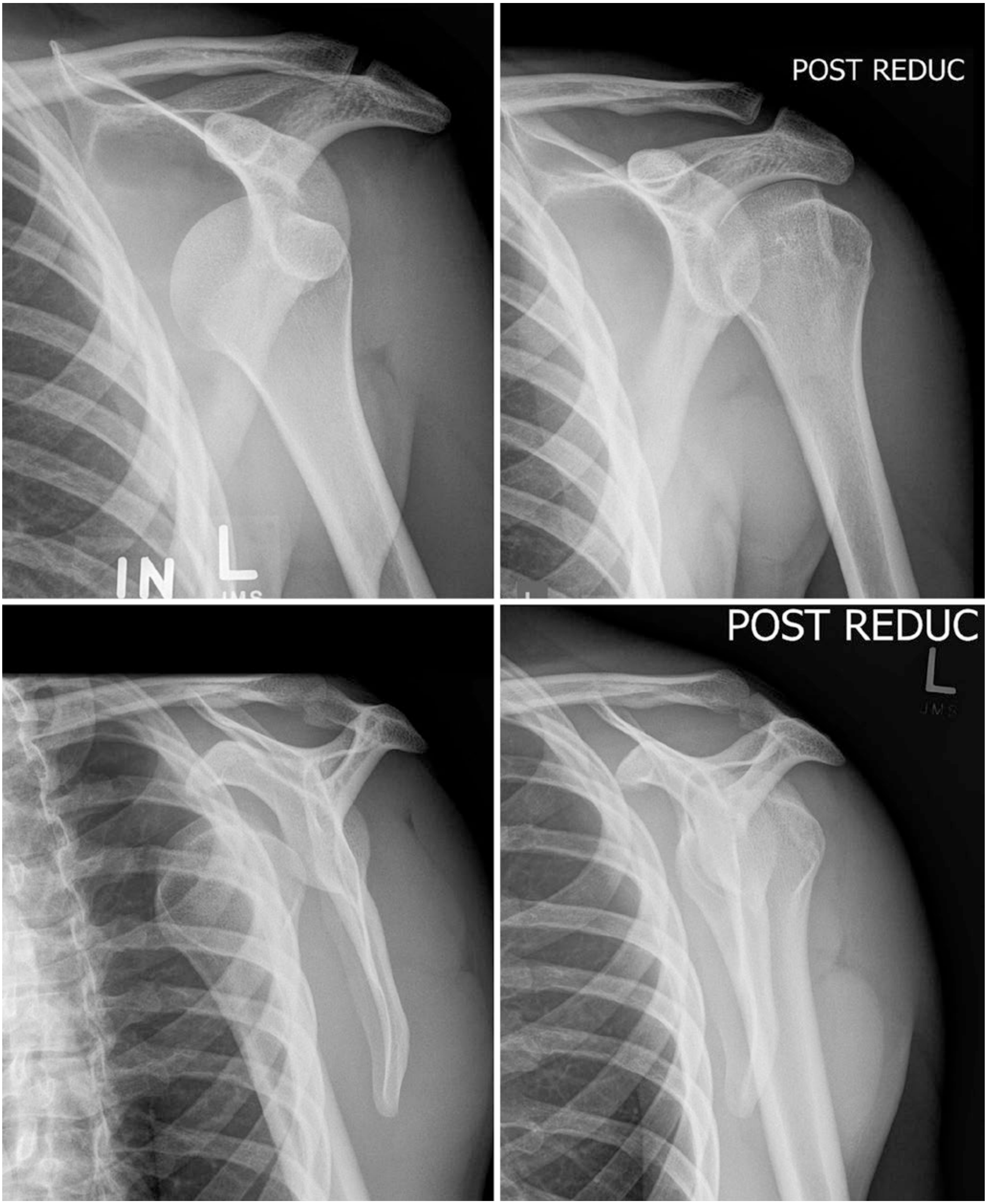

Fig. 9.5 AP and Y view radiographs showing an anterior shoulder dislocation with the humeral head located anterior to the glenoid and post-reduction radiographs. (*From the University of Florida Student Health Care Center Radiology Teaching File, with the assistance of Joan M. Street, RT*)

result in anterior/posterior, dorsal/volar, or medial/lateral dislocation. For example, in an anterior glenohumeral dislocation, the humerus dislocates anterior to glenoid (Fig. 9.5). With a dorsal finger dislocation, the middle phalanx dislocates dorsal to the proximal phalanx (Fig. 9.6). Patellar dislocations occur most commonly in the lateral direction (Fig. 9.7).

Simple dislocation: absence of fracture, and a closed reduction is performed to reduce dislocation.

Complex dislocation: associated with fracture, or there is soft tissue interposed in the joint necessitating an open reduction (Fig. 9.8).

Association with fractures:

It is important to assess for any fractures that may be associated with a joint dislocation, and extreme caution should be taken when relocating a fracture-dislocation prior to obtaining imaging. There is increased risk of displacing the fracture fragment or converting a simple dislocation into a complex one. The clinician has to weigh the risks and benefits before attempting reduction prior to obtaining imaging. The risks of associated fracture with dislocation are higher in some joints such as elbow, ankle, wrist, and hip joints in comparison to finger, patellofemoral, and shoulder joints (Fig. 9.9).

Overview of Acute Management

Initial Assessment

The initial evaluation of a person with a suspected joint dislocation should always include a focused history including mechanism involved, any other associated injuries, previous injuries (including dislocation) to the involved joint, relevant medical history, and allergies. Assessment of neurovascular status is the next most important step which includes checking for pulses, capillary refill, sensation, and motor function [8]. Further examination, including inspection of the injured joint, comparison to uninjured side if possible, and palpation for

Fig. 9.6 A lateral finger radiograph showing a dorsal finger dislocation with the middle phalanx located dorsal to the proximal phalanx. (*From the University of Florida Student Health Care Center Radiology Teaching File, with the assistance of Joan M. Street, RT*)

fractures and associated injuries should also be performed. The decision to attempt immediate relocation depends on multiple factors. Factors that may promote the decision are neurovascular compromise, previous dislocation of that joint, clinicians' experience, and no immediate access to radiographs. It can be much easier to relocate a joint prior to muscle spasm setting in. Factors that might weigh against it would be suspicion for associated fracture, uncertainty about the diagnosis, and ease of obtaining radiographs and specialty consult.

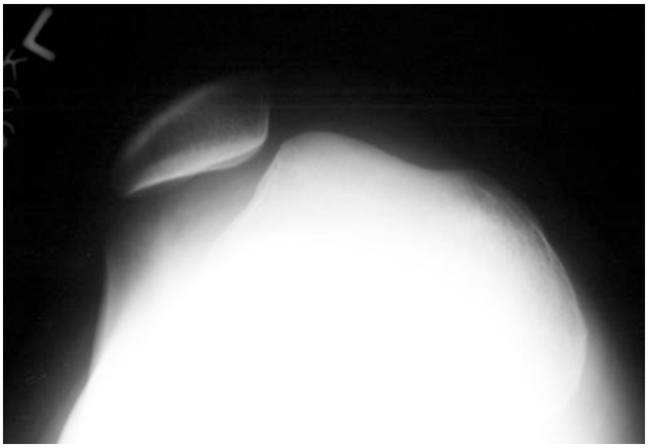

Fig. 9.7 A lateral patella dislocation with the patella located lateral to the femoral groove seen here on sunrise view. (*From the University of Florida Student Health Care Center Radiology Teaching File, with the assistance of Joan M. Street, RT*)

Urgent Considerations

Early consideration and recognition of urgent conditions requiring immediate treatment is important in preventing long-term sequela and complications.

Life-Threatening Conditions

Rarely, life-threatening conditions can be associated with a joint dislocation. These are usually associated injuries related to the trauma required to dislocate a stable joint. For example, the hip joint requires a significant force to dislocate the femur from the acetabulum. The trauma required for this to occur may also cause internal organ damage, bleeding, pelvic fractures, and shock which could be life-threatening [8].

Fig. 9.8 AP hip radiographs showing a complex dislocation of the right hip with fractures of the acetabulum and femoral head and post-reduction image. (*From the University of Florida Student Health Care Center Radiology Teaching File, with the assistance of Joan M. Street, RT*)

Fig. 9.9 Posterior left elbow dislocation with coronoid process avulsion fracture in a 29-year-old male as a result of a snowboarding injury. (Courtesy of Morteza Khodaee, MD, MPH)

Arterial Injury

Arterial injury can occur with joint dislocations, some more commonly than others (e.g., knee), and circulation should always be assessed quickly in a suspected dislocated joint with palpation of distal pulses, capillary refill, and extremity temperature, inspection for pallor and mottled skin, and assessment for decreased sensation. It is always recommended to compare to the other side.

If vascular status is compromised on initial exam, immediate attempt at restoring vascular status with a relocation maneuver should be attempted by the most experienced provider available. If the first attempt fails, it is not recommended to try again unless emergency transport is unavailable for extended amount of time [8].

A high index of suspicion for damage to the popliteal artery should be used for any suspected knee dislocation. Sports injuries cause up to 33% of knee dislocations [9] and the incidence of popliteal artery disruption with a knee dislocation is believed to be between 20% and 40% [10, 11].

Avascular necrosis of the femoral head can be a complication with hip joint dislocation. If the hip is reduced in a timely fashion (< 6 hrs), it decreases the chance of this complication [8, 11].

The vascular exam should be repeated after any manipulation maneuvers or with any change in symptoms, and all vascular exams should be documented in the medical record.

Nerve Injury

Nerve injuries can occur with joint dislocations and a focused assessment of nerve function should accompany any suspected dislocation. Shoulder, knee, and hip dislocations are most commonly associated with nerve injury.

Damage to the axillary nerve accounts for the majority of cases of nerve injury with a shoulder dislocation due to its fixed position in the quadrangular space and traction that occurs with downward displacement of the humeral head [12]. This can be assessed easily checking sensation with light touch to the lateral deltoid.

The common peroneal nerve is the most common nerve injury seen with knee dislocations due to its location passing around the fibular neck [8–10]. Sensation over the dorsum of the foot and motor examination including ankle dorsiflexion should be performed to assess function.

The sciatic nerve can be injured with laceration, stretching, and compression during a hip dislocation where it runs inferior/posterior to the hip joint or can occur as a late sequela if it becomes enclosed in heterotopic ossification, and nerve function may not recover in all cases [13, 14].

Compartment Syndrome

Acute extremity compartment syndrome can be seen after a joint dislocation but is usually caused by the related soft tissue injury or associated fracture that might be seen with a knee, hip, or elbow dislocation. The increase in compartmental pressure seen with the soft tissue injury and local swelling leads to tissue ischemia. The first and most important sign of possible compartment syndrome is severe pain out of proportion to exam or injury, unresponsive to analgesia. Pain with passive stretching of the affected area can be seen and paraesthesia may be seen early. It is important to note that the traditional "pallor, paralysis, and pulselessness" taught in the pneumonic about the five P's of compartment syndrome are more signs of arterial ischemia and might not be present at all or be present very late in the course [15]. If not identified and treated, acute compartment syndrome can lead to consequences including permanent dysaesthesia, ischemic contractures, muscle dysfunction, loss of limb, or death [15].

Open Injuries

A fracture-dislocation may be considered open anytime the bone is exposed to the environment outside the body through a break in the skin even if the bone is not visible at the time of exam. (see Fig. 17.17). This is an orthopedic emergency requiring irrigation, surgical debridement, and intravenous antibiotic administration to limit consequences such as infection and poor healing [16].

Irreducible

Occasionally, a joint dislocation may be irreducible at the time of exam. This is usually either because of soft tissue interposition into the joint, preventing return of normal alignment, or because of muscle spasm and pain onset. Soft tissue interposition is often seen with volar proximal interphalangeal (PIP) joint dislocations. These dislocations are much less common than dorsal or lateral dislocations of the PIP joint but when occur may have the central slip, lateral band, or torn collateral ligament be interposed within the joint [17]. In any joint dislocation where soft tissue prevents relocation, open surgical reduction is often indicated (Fig. 9.10).

Fig. 9.10 Posterior left 2nd and 3rd MCPJ dislocation in a 25-year-old male as a result of a fall while skiing (**a**, **b**). The 2nd MCPJ joint was successfully reduced at the clinic (**c**, **d**), but due to soft tissue entrapment, the 3rd MCPJ dislocation required open reduction in the operating room. (Courtesy of Morteza Khodaee, MD, MPH)

Radiographic Studies

The decision on if and when to obtain radiographic studies in an athlete with suspected joint dislocation may depend on the mechanism of injury (traumatic or atraumatic), timing of presentation (witnessed on sideline of event or later in clinic or emergency room), neurovascular status at time of presentation, and patient history of previous injuries or dislocation of involved joint. If available and if it does not significantly delay the reduction, plain radiography should be obtained. If neurovascular compromise is present, reduction should be attempted immediately to try and restore neurovascular status [8]. If there was a high-force trauma involved, suspicion for a fracture is present, the physician is clinically uncertain of joint position, or it is a first-time dislocation for the patient, radiographs are indicated [18]. Another important consideration regarding level of suspicion for fracture is that fractures are more common than dislocations in the elderly and pediatric (skeletally immature) populations [8].

Radiographs are generally indicated post-reduction to rule out any associated injuries that may affect healing or treatment and to ensure adequate alignment.

Relocation (Reduction)

Relocation of a joint should always follow a joint dislocation. The timing and ability to relocate the joint are two considerations. Indications for immediate attempts at relocation include compromised neurovascular status, preservation of skin, pain reduction, and ease of splinting for transportation [8].

Knowledge of multiple reduction techniques for different joints can aid in ability to relocate a joint in different situations. General concepts in reduction of a joint dislocation include applying gentle traction to the involved joint. Occasionally this is all that is needed. If it does not reduce with gentle traction, gently recreating the position causing the dislocation while continuing to hold traction is usually performed. Specific techniques to each joint will be covered elsewhere.

The clinician should use their judgment based on the potential risks and benefits, as well as comfort level with reduction techniques, when deciding on whether or not to reduce a joint.

When reduction is attempted, but fails, multiple attempts should only occur if an extended period of time is expected to be able to get to the nearest medical facility or specialty care.

Immobilization

All relocated joint dislocations benefit from a period of immobilization to allow healing and prevent recurrent or further injury. The type of immobilization may range from buddy tape to immobilizer and depends on the joint involved, the severity of the injury, location of the injury, concurrent injuries, and need for surgical intervention.

Other Acute Measures

Pain is important to consider and sometimes pain medications and muscle relaxants may be needed to be administered for severe injuries or to aid in relocation of a joint. Aspiration can be considered in cases of significant hemarthrosis for symptomatic relief. Intra-articular injections can be performed for analgesia relatively quickly and simply. In recalcitrant cases, procedural sedation may be needed under appropriate monitoring.

Pediatric Considerations

Children have special considerations to consider. Because their bones are still growing, the growth plate is a relatively weak area of bone and more prone to injury, than the relatively stronger ligaments and tendons. With the same injury mechanism and force, a child is more likely to hurt their bone, while an adult is more likely to hurt their ligaments and tendons. For this reason, dislocations are less likely in the pediatric population and the clinician should be alert for other injuries like a fracture.

The clinician also must have a high level of suspicion for injuries and complications as pediatric patients may not be able to verbalize symptoms and history.

Certain genetic conditions, such as Ehlers-Danlos syndrome (EDS), may present for the first time with joint dislocation or hypermobility. In one study of 205 patients, 15% had a documented dislocation during a 4-year period. The most common sites were the patella followed by the shoulder [19].

When dislocations do occur in the pediatric population, treatment and surgical planning may differ due to the skeletal immaturity and requires careful planning and protection of growth plates. The incidence of patellar dislocation has increased with the rising participation in youth sports. Approximately half of young patients' patellar dislocations occurred during athletics [20].

Follow-Up Care

Non-urgent Referral Considerations

Patients with first-time dislocations of the GHJ and the patella may need a referral to an orthopedic surgeon to discuss possible surgical options (this is discussed in depth elsewhere in the book). Young, active patients with a shoulder dislocation may have less recurrence and better functional outcomes after surgery [18]. After an initial patella dislocation, skeletally immature patients frequently have a recurrence, and each additional dislocation carries a substantial risk of further cartilage damage and future patellofemoral arthritis [20].

Additional referral considerations may include level of competition of the athlete and age of the patient, as well as hand dominance and occupation for upper extremity dislocations.

Return to Sports After Reduction

There is no consensus on return to play after different joint dislocations. Return-to-play considerations include whether it is a first time dislocation, association with other injuries, ability to brace, level of play, and timing during the season. As a general practice, many athletes are allowed to return to play when they can demonstrate full, pain-free range of motion and strength in the affected joint and perform sports-specific activities.

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Musculoskeletal Healing Process

10

Jessica Devitt

Key Points

- Musculoskeletal (MSK) healing is generally described as having three phases post injury: inflammation, repair/proliferation, and maturation.
- The duration and degree of healing varies by injury location, the type of tissue involved, and the severity of injury.
- As repair requires additional energy, consuming sufficient calories in the form of foods high in amino acids, trace minerals, and vitamins is imperative.
- Screening for nutritional deficiencies may be beneficial in high-risk patients and when healing is delayed.
- NSAIDs, corticosteroids, smoking, and excessive alcohol intake should all be avoided as they can delay and compromise repair.
- While weight bearing may be avoided acutely, weight bearing is critical to MSK tissue repair.
- In bone healing specifically, extracorporeal shock wave therapy (ESW), autologous bone marrow, autologous bone grafts, fibroblast growth factor-2 (FGF-2), platelet-derived growth factor (PDGF), and parathyroid hormone (PTH) have shown promising results in promoting bone healing.

Introduction

MSK pathology can easily be described as an epidemic. According to a 2012 US National Health Interview Survey, approximately 126.6 million adults, representing over half of the estimated population over 18, reported MSK medical conditions [1]. MSK conditions were diagnosed in up to 18% of health care visits in 2011 [1]. MSK problems are so common they are an expected part of life for many populations. For this reason, it is important to understand the pathology behind MSK injury, the mechanisms of repair, and how to promote effective MSK healing. In this chapter, we will discuss the common stages of MSK healing, specific patterns by MSK tissue type (Table 10.1), and factors that influence and promote repair. Finally we will review novel interventions for interrupted or delayed healing, specifically for cases of nonunion fractures.

While specific details regarding the physiology of healing vary by injury location, the type of tissue involved, and the degrees of injury, there are general patterns shared across MSK injuries. MSK healing is generally described as having three phases post injury: inflammation, repair/proliferation, and maturation [2]. These basic phases are illustrated following a fracture in Fig. 10.1 and following a muscle injury in Fig. 10.2.

Inflammation (from Injury Through Days 4 to 6)

Hemostasis

With the exception of articular cartilage (see Table 10.1 for details) most MSK tissues bleed in response to injury. In order to achieve hemostasis immediately after injury, local blood vessels constrict and the coagulation cascade is set in motion. Clot formation results from platelet aggregation and consists of collagen, thrombin, and fibronectin. Platelets involved in the clot formation release cytokines and growth factors involved in the inflammation pathway. This primitive structure will serve as a “scaffold” for the inflammatory and rebuilding cells attracted to the area by signaling molecules. This process also results in subsequent vasodilation and vascular permeability, which aids in the invasion of specialized cells [2].

J. Devitt (✉)
Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA
e-mail: jessica.devitt@cuanschutz.edu

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Table 10.1 Description of MSK tissue function, injury, and mechanism of healing by type

	Muscle	Tendon	Ligament	Articular (hyaline) surface cartilage	Fibrocartilage (focusing on knee meniscus)	Bone
Function	Contracts to stabilize and facilitate motion in joints [2]	Connects muscle to bone	Stabilizes joints by connecting bone to bone	Lines joints allowing for smooth joint movement [2] and weight distribution [3]	Occurs in the meniscus (reduces contact pressures on hyaline cartilage and adds stability), the labrum, triangular fibrocartilage complex (TFCC), acromioclavicular (AC) joint, and the annulus of the intervertebral discs [4]	Bone provides scaffolding for soft tissues
Injury	Partial or complete tear, contusion, laceration [5]	Tendinopathy vs. tear increased noncollagenous ECM, hypercellularity, neovascularization [2]	Partial or complete tears	Partial and full thickness defects	Knee Meniscal Injuries: partial and full thickness tears in the peripheral vascular zone (PVZ) and the avascular zone (AZ) [6]	Fracture
Inflammation	In the first few days after injury, degeneration and inflammation begin [7]. Calcium is released and clot formation begins after the rupture of myofibers and capillaries [2]. Chemoattractants released by local activated macrophages prompt the migration of inflammatory cells to the area of injury. Damaged tissue is then cleared through phagocytosis, inflammatory mediators are released, and fibroblasts and satellite cells are activated. This process can last for several days [2, 7]	Homeostasis-achieved through hematoma formation, inflammation and fibroblasts and tenocytes proliferation at the injury site [2, 8]	Bleeding from initial tear eventually leads to clot formation. Clot forms in the gap between torn fibers and is infiltrated by a cellular matrix. Of note, for some ligaments, e.g., the ACL, have limited clot-forming ability and the clot may be resorbed before further repair can take place	*Partial thickness injury*: No or minimal inflammation due to the avascular and hypocellular nature of articular cartilage. *Full thickness injury*: The subchondral bone is injured triggering inflammation involved in bone injury (see next column). This stimulates the formation of a fibrin clot [9] within the articular cartilage defect	*Injury in the AZ:* No or minimal inflammation due to the avascular nature of this portion of the meniscus. *Injury in the PVS:* Bleeding from the initial tear leads to the formation of a fibrin clot which will serve as a scaffold for the repair [6]	Fracture disrupts blood vessels in the periosteum leading to bleeding and hematoma formation. A fibrin network is created and serves as a rudimentary matrix for the bone rebuilding cells (osteocytes and chondroblasts) attracted to the site by inflammatory signaling molecules. The gap created by the fracture is initially bridged by granulation tissue. This process generally occurs during the first week post injury [8]. An initial soft callous develops providing some limited stability several weeks after the injury [2, 10]

Repair/ proliferation	During the first week after injury, the coordinated interactions of fibroblasts and satellite cells begin the process of muscle regeneration which can begin around 24 hours post injury, though newly formed myofibers are not detected until at least 3–5 days post injury and this process peaks around 2 weeks [7]	Fibroblasts and tenocytes are responsible for forming a fibrous tissue at the injury site and rebuilding collagen (type III). This process can take weeks during which time the tendon is still weak and prone to re-injury [2, 8]	Successful proliferation/ repair depends on the size of the initial tear and whether or not any contact remains between the two ends of the ligament. In successful repair, if the clot is able to bridge the two ends of the torn ligament and establish a cellular matrix before it is resorbed, fibroblasts then produce collagen to form a bridge	*Partial thickness injury*: Repair and proliferation are not possible as the articular cartilage itself is incapable of generating the necessary inflammatory and reparative agents *Full thickness injury*: Injured subchondral bone releases a limited number of reparative cells from bone marrow [9]	*Injury in the AZ:* Repair and proliferation are very unlikely due to the inability of this tissue to generate inflammatory and reparative agents. *Injury in the PVS:* The perimeniscal capillary plexus and synovial fringe supply vessels that migrate along the fibrin scaffold along with mesenchymal cells [6]	The chondroblasts build a cartilaginous callous which is eventually transformed into primitive woven bone by osteoblasts forming a more stable hard callous. Formation of the hard callous (capable of load bearing) occurs several weeks to months after the initial injury [2, 10]
Repair/ maturation	During fibrosis, connective tissue forms between muscle fibers to form a scar (usually between the 2nd and 3rd week after injury) [7]	Over time repaired tissue decreases in cellularity and vascularity, the proportion of type I collagen increases, and collagen fibers and tenocytes begin to align with the direction of stress increasing the tendon's strength [2, 3]	Over time the collagen will become organized according to the planes of force increasing the strength of the repair (weeks to months depending on the ligament injured and degree of injury)	*Partial thickness injury*: These defects are unable to heal without intervention and often expand over time due to mechanical loading on edges. *Full thickness injury*: Fibrocartilage forms from the fibrin clot and reparative cells released by subchondral bone	*Injury in the AZ:* These tears are generally unable to heal without intervention. *Injury in the PVS:* A fibrovascular scar forms bonding the free edges of the tear together [6]	Also known as the remodeling phase, the immature bone of the hard callous is then transformed into more mature and stable lamellar bone along the lines of force returning integrity to the bone [2, 8, 10]

(continued)

Table 10.1 (continued)

	Muscle	Tendon	Ligament	Articular (hyaline) surface cartilage	Fibrocartilage (focusing on knee meniscus)	Bone
Duration of repair/ completion of healing	Often 4–6 weeks	Weeks to months	Weeks to months (often months for larger ligaments/more significant tears). If no contact remains between the two ends of a ligament repair may never occur without intervention	*Partial thickness injury:* Never heal without intervention. *Full thickness injury:* Weeks to months	*Injury in the AZ:* Rarely heal without intervention. *Injury in the PVS:* Often 3–4 months	This process can take months to years
Expected performance of repaired tissue	Muscle repair can lead to either partial (weaker and more prone to re-injury) or functional repair depending on the degrees of fibrosis that occurs (the degrees to which injured tissue is replaced with functional muscle tissue vs. muscle scar) [7]	Often the tendon will never regain preinjury strength	Repaired tissue will remain more cellular, vascular, have decreased collagen cross linking and size, and abnormal innervation rendering it less elastic and therefore more prone to injury than uninjured tissue	*Partial thickness injury:* NA *Full thickness injury:* Resulting fibrocartilage remains structurally weaker and less mechanically competent than uninjured hyaline cartilage	*Injury in the AZ:* NA *Injury in the PVS:* Resulting fibrovascular scar remains structurally weaker than the uninjured meniscal tissue	After full maturation healed bone can often resume to previous load-bearing capacity depending on the degree and displacement of the initial fracture

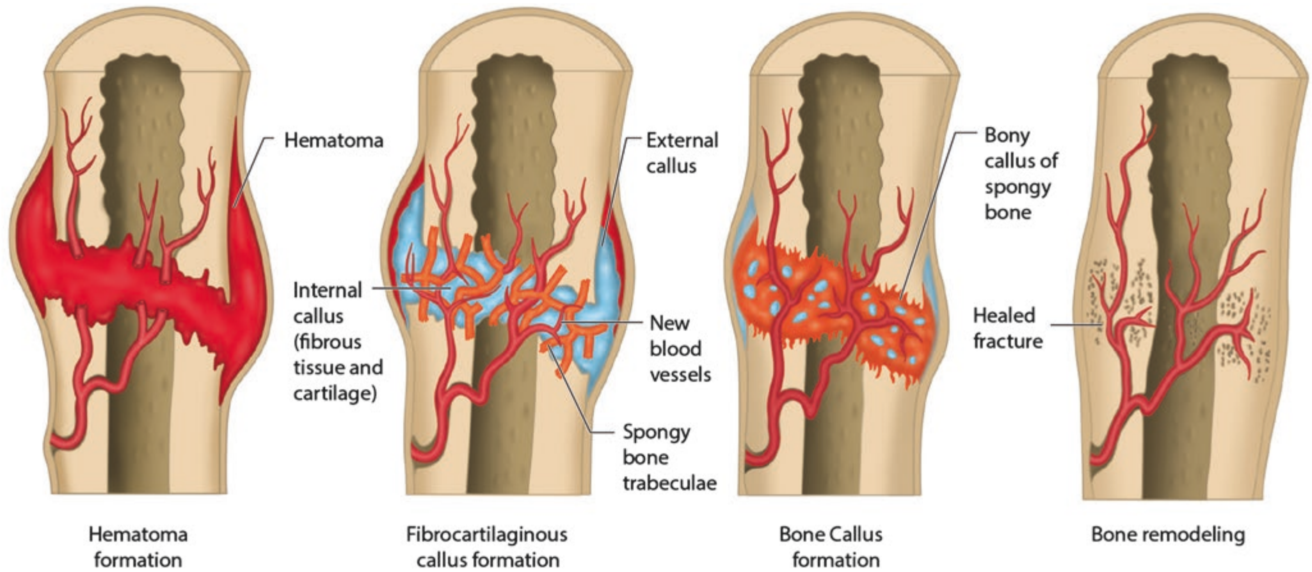

Fig. 10.1 Phases of healing post fracture: (1) inflammation/hematoma formation, (2) repair, (3) proliferation, and (4) maturation/bone remodeling

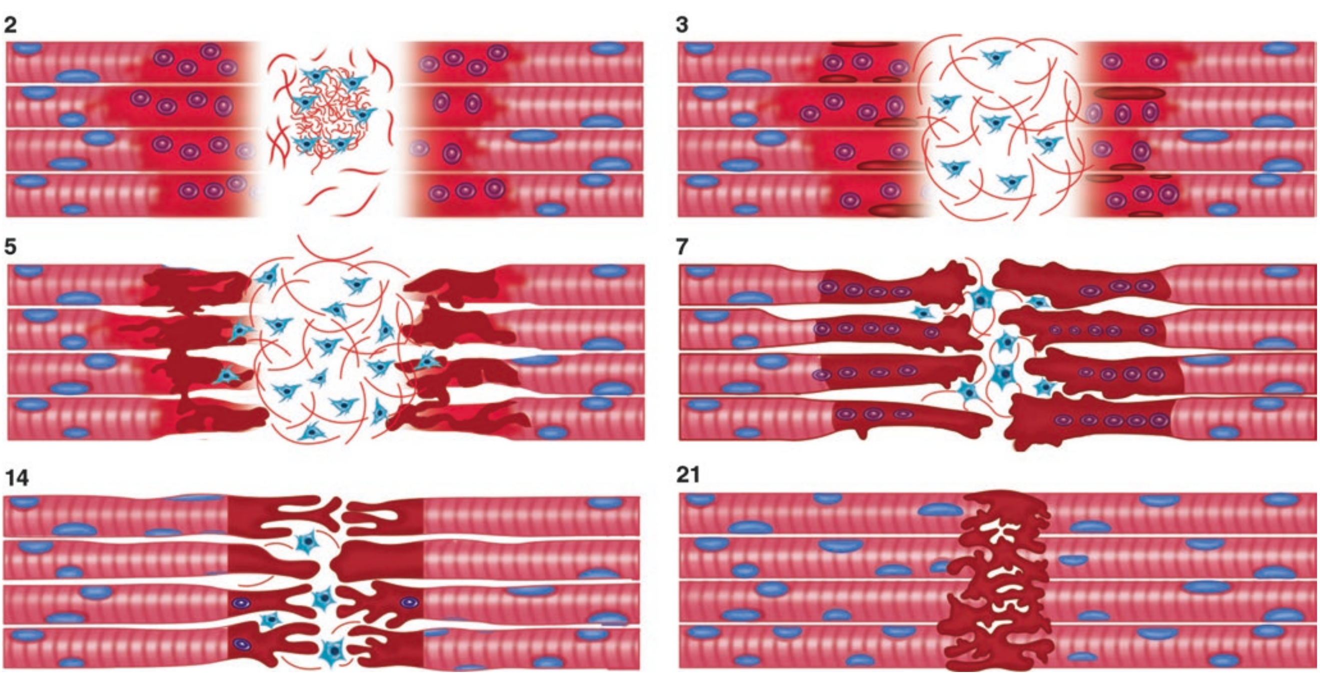

Fig. 10.2 Skeletal muscle healing stages. Day 2: inflammation – bleeding from torn myofibers contributes to clot formation while necrotized myofibers are removed. Days 3–14: repair/proliferation – satellite cells are activated, myoblasts fuse into myotubes, and these maturing muscle cells then penetrate the clot/scar to close the gap resulting from the tear. Day 21: maturation – the myofibers from each end of the tear have fused with little remaining scar and strengthening can begin

Inflammation

Signaling molecules also attract neutrophils which begin clearing cellular debris generally sparing uninjured tissue. Of note, the mechanism by which healthy tissue protects itself can be overwhelmed by a strong inflammatory response. Around 48 to 96 hours activated macrophages phagocytose debris and neutrophils, making way for the fibroblasts (rebuilding cells) they recruit to the area. This marks the transition to the proliferative phase [2].

Proliferation (Day 4 Through 14)

Angiogenesis/Fibroplasia

Vascular endothelial growth factor stimulates new capillary formation increasing blood flow to the injured site. Fibroblasts, responding to signals from macrophages and platelets, migrate to the site of injury and synthesize collagen and the scaffolding necessary for tissue repair [2].

Maturation and Remodeling (Day 8 to 1 Year)

During the maturation and remodeling phase, the extracellular matrix (ECM) is reinforced and collagen is produced in an organized structure which then grows "along lines of stress." While collagen regrowth often allows return of function, this repaired tissue will never be as structurally sound as a comparable tissue that has never been subject to an injury [2].

More details regarding each of these phases by specific tissue type can be found in Table 10.1.

Healing Modulators

Nutrition

It is widely understood and accepted that malnourishment impairs healing. However, one concept that may be counterintuitive, especially in the athletic population, is that the decrease in athletic activity that often follows injury does not necessarily equal a decrease in energy consumption/need. Rebuilding injured tissue consumes energy and, depending on the extent of the injury, can increase energy requirements by 15–50% [11]. Muscle protein synthesis (a task frequently required as a part of MSK injury healing) requires a significant amount of energy. A muscular male may spend up to 500 kcal/day on muscle protein synthesis alone (not including any activity) [11]. Additionally, having to adopt novel methods to perform activities of daily living including walking can increase energy requirements as well (e.g., ambulating with crutches may consume up to two to three times more energy than normal ambulation) [2, 11]. Therefore, though there may be a lull in athletic activity after an injury, it is important that patients maintain adequate caloric intake, with special attention paid to the micro- and macronutrients known to be important to MSK healing.

Good quality animal and soy products contain the essential amino acids needed for protein production and collagen formation. Ensuring that patients are meeting the standard dietary recommendations for trace metals (specifically zinc, copper, and magnesium), folate, and vitamins A, C, D, and E will also support the healing process. However, while deficiencies in these nutrients are associated with impaired healing, there is no evidence that supplementation in the absence of a deficiency is helpful. In that respect, one could also advocate for screening for low levels of the above trace minerals and vitamins as well as iron levels, albumin, and prealbumin to evaluate the patient's nutrition status in order to determine if supplementation would be beneficial, and especially in patients where healing seems to be delayed or impaired [2]. Regardless, in the absence of a deficiency or screening data, it stands to reason to advocate for a healthy diet to include some animal protein for trace metals and vitamins (red meat and poultry for zinc, fish and shellfish for copper, magnesium, and vitamins D and E), a good amount of fresh fruits and vegetables (especially dark leafy greens), beans, and nuts. Trace metals can also be found in whole grains, dark leafy greens, and other non-animal sources for patients that may adhere to a vegetarian diet.

Lastly, there is ample evidence to suggest that alcohol can impair or delay healing. Advising athletes who, in the face of injury may be tempted to treat sorrows and pain with alcohol, to limit if not abstain from alcohol may be wise [11].

NSAIDs and Corticosteroids

There is conflicting evidence regarding the use of NSAIDs and corticosteroids for MSK injuries, and their effect will surely vary by timing, dose, and duration of use. However, it may be safest to avoid NSAID use during the acute phase of an injury as there is growing evidence that they may impair MSK healing. Animal models have demonstrated a negative effect on the tensile strength of healing tendons exposed to NSAIDs as well as a negative effect on bone healing especially if used within the first week of fracture [2]. There is also evidence that a cox-2-specific inhibitor (e.g., celecoxib) may hamper skeletal muscle healing [7]. Though NSAIDs can decrease certain types of inflammation and may have a role to play in certain specific injuries (preventing the calcification of muscle hematoma), it would likely be wise to avoid their use in acute injuries of muscles, tendons, ligaments, and bones until further research is done [2, 7].

Pressure

Edema can cause elevated tissue pressures which can limit blood supply to the injured area and delay healing [2]. For this reason, compression and elevation are part of routine post-acute injury RICE (rest, ice, compression, elevation) recommendations. Reducing edema may help expedite healing. However, later in the course of recovering from injury, restricting blood supply and therefore oxygen during strength

training of atrophied muscles while recovering from an injury may actually be beneficial. Studies have shown that strength training with vascular restriction (achieved through the use of a compression device, e.g., wrap, blood pressure cuff) resulted in increased muscle size and strength [12]. While further study is needed, this form of therapy could prove beneficial to patients who are unable to participate in high-intensity resistance training after injury or surgery.

Mechanical Forces

While full weight bearing may need to be avoided immediately after an injury to a lower extremity and until pain with weight bearing alone subsides, weight bearing is critical to healing. Loading the injured area promotes the reorganization of collagen and the ECM essential to the maturation/remodeling phase and improves circulation at the site of the injury. Although further study is necessary to provide specific loading recommendations for each MSK tissue type and their respective function, current evidence suggests that low-magnitude and high-frequency loads punctuated by rest have the greatest effect on bone healing and eccentric exercise therapy has become the main treatment for tendinopathies [2].

Comorbidities and Behaviors That Impair Healing

There is a variety of comorbid conditions and behaviors that can negatively impact healing. It is important to be familiar with these negative modulators to be able to identify patients who are at risk of delayed or impaired healing that might benefit from intensive healing support. It is also important to consider screening for these conditions and risk factors in a patient who is not healing as expected, especially if other signs, symptoms, or risk factors are present for any of these conditions/behaviors (Table 10.2).

Table 10.2 Comorbidities and behaviors that impair MSK healing [2, 8]

Conditions
Any condition that compromises blood flow can impair healing. These conditions include both microvascular disease (e.g., diabetes) and macrovascular disease (e.g., peripheral artery disease)
Any condition that impairs immune function or effects protein or collagen formation (e.g., HIV, chronic steroid use)
Any condition that effects protein or collagen formation (e.g., connective tissue disease, hypothyroidism)
Neuropathy
Organ failure
Extreme age
Estrogen deficiency
Chronic site infection
Obesity
Deformity
Behaviors
EtOH use
Smoking

Specific Therapies for Fracture Healing

Fractures are a common injury affecting almost 8 million people in the United States each year. Approximately 5–10% of those fractures are complicated by delayed or impaired healing. Multiple modalities have been tried and tested for the purpose of improving fracture healing including physical stimulation therapies and biologic therapies, both local and systemic. Commonly employed physical stimulation therapies include electrical stimulation, low intensity pulsed ultrasound (LIPUS), and extracorporeal shock wave (ESW) therapy. Unfortunately, studies of these modalities have not yet provided clear evidence of their efficacy. Basic science studies have shown a clear benefit of electrical stimulation while clinical studies have provided mixed results. LIPUS was found to be safe for the treatment of acute fractures in long bones in one trial, but a meta-analysis concluded that the evidence for the efficacy of LIPUS was limited and conflicting [11- > 13]. A systematic review concluded that ESW may promote healing of delayed or nonunion fractures but this was only a level 4 evidence [11- > 13]. Future research into the efficacy of these modalities is needed.

Local biologic strategies for enhancing bone healing include autologous bone marrow, autologous bone grafts, fibroblast growth factor-2 (FGF-2), platelet-rich plasma (PRP), platelet-derived growth factor (PDGF), and bone morphogenetic proteins (BMP). Of these interventions, autologous bone marrow, autologous bone grafts, FGF-2, and PDGF have the best evidence of efficacy; however, further studies are still needed. A large systematic review, though limited by variability in outcome measures examined by the included studies, found no significant benefit to PRP over control therapy in any of the reported outcomes. Similarly, while there was a trial that found a benefit to BMP 2 in tibial shaft fractures, other studies of both BMP 2 and BMP 7 have failed to demonstrate benefits [13]. That said, there is some evidence that the success of certain biologic strategies may also depend on applying them in conjunction with certain physical rehabilitation activities; therefore, it may be that some of these interventions could prove beneficial when coupled with some mechanotherapy [12]. Further research is needed in the area.

Two common systemic biologic strategies include treatment with parathyroid hormone (PTH) and bisphosphonates. PTH seems to have the most robust evidence, with its efficacy being demonstrated in both animal studies and clinical

trials. In one study, PTH significantly improved healing of certain fractures in post-menopausal women [13]. Regarding bisphosphonates, their efficacy in improving fracture healing has been demonstrated in animal studies but clinical trials are lacking [13].

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11 Complications

Andrew M. Wood

Key Points

- Most complications from sports trauma have significant morbidity for the athlete, and this is lessened by prompt diagnosis and treatment.
- Taking a comprehensive history of the injury and a focused physical examination including neurovascular status is recommended.
- Emergent treatment of complications such as compartment syndrome, fat embolism, and vascular/nerve injuries is important for improved outcomes.

Introduction

Sports-related trauma can take many forms ranging from lacerations to the skin, broken bones, joint dislocations, and many others. Most injuries will heal without issue, but a small percentage can develop complications. Complications from sports injuries can range from acute problems like acute compartment syndrome to chronic issues such as fracture nonunion.

Infection is one of the most common complications related to sports injuries. They can present in many ways, a study looking at the rate of infection following simple hand lacerations reported around 5% of lacerations will become infected [1]. Sports-related surgery can also have postoperative infection which can complicate the healing of an injury. Postoperative infections are rare; for example, only 0.68% of anterior cruciate ligament (ACL) reconstructions get infected [2]. Postoperative infections can be devastating, resulting in loss of range of motion, graft failure, and osteomyelitis [2].

Clostridium tetani spores enter the body through broken skin. Once in an anaerobic environment the spores germinate and produce toxins blocking neurotransmitters and leading to unopposed muscle contraction. Tetanus has an incubation period averaging 10 days with the initial sign being spasm of the jaw or “lockjaw.” Tetanus is a medical emergency with goal of immediate treatment with human tetanus immune globulin. Dirty and penetrating wounds are considered higher risk for tetanus. If the tetanus status is uncertain, then the tetanus toxoid-containing vaccine should be given. Also, if a patient’s last dose of a tetanus vaccine was received five years or longer then a booster vaccine should be administered.

Acute peripheral nerve damage can be due to acute injury from a direct blow or chronic injury secondary to repetitive microtrauma causing nerve entrapment. Typically, if not catastrophic, treatment involves rest, anti-inflammatories, splinting, and physical therapy. For example, in the shoulder, common nerve injuries include spinal accessory nerve injuries usually from direct blows to the neck. These result in trapezius paralysis with sparing of the sternocleidomastoid muscles. Long thoracic nerve injuries result in paralysis of serratus anterior and scapular winging (Fig. 11.1). Suprascapular nerve injuries result in rotator cuff weakness.

Fig. 11.1 Right scapular winging in a 30-year-old male

A. M. Wood (✉)
Department of Family Medicine, Denver Health Medical Center, Denver, CO, USA

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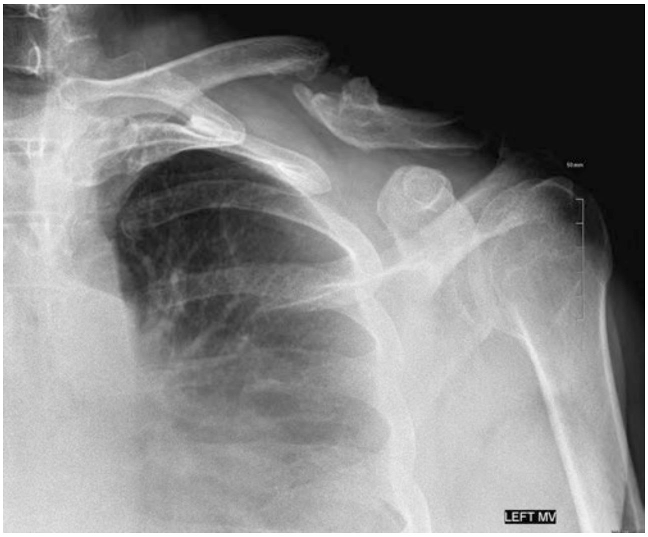

Fig. 11.2 Left mid-shaft clavicle fracture nonunion

Axillary nerve injuries are associated with anterior shoulder dislocations [3].

Fracture care can have a wide range of complications. Nonunion of a fracture occurs when there is incomplete bridging and lack of osseous healing of a fracture (Fig. 11.2). It has been suggested that nonunion rates are around 5–10% of all fractures [4]. Risk factors for nonunion of fractures include severe fractures (open fractures, multiple fractures), obesity, smoking, and alcoholism [5]. Women have a slightly higher rate of fracture, but men have higher risk of nonunion [5]. In addition to nonunion of fractures there can be malunions. These occur when a fracture ossifies but with a deformity like angulation or rotation. All fractures can develop malunions but the most common location is clavicle fractures. It is reported that around two-thirds of mid-shaft clavicle fractures will have some malunion [6]. Most of these fractures will cause no functional limitations and mainly be cosmetic. Risk factors for symptomatic malunion of clavicle fractures are shortening of clavicle length by 1.4–2 cm [6]. When malunion occurs with other pediatric radial fractures for example, there can be a loss of pronation and supination of wrist motion [7].

Deep vein thrombosis (DVT) is a significant complication as well. Anytime there is tissue trauma this can contribute to thrombosis. DVT with fracture care mainly occurs with major fractures of the hip, pelvis, and femur. For example, with knee and hip replacements the incidence of DVT without prophylactic anticoagulation is 27% [8]. Fatal pulmonary embolism ranges around 1.4–7.5% within three months of hip fracture surgery [8]. Due to these major orthopedic procedures being such high risk for DVT, use of prophylactic anticoagulation to reduce this risk is recommended. Thromboembolism prophylaxis reduces the rate of DVT by approximately 60% [8]. DVT can also occur with prolonged immobilization, especially in the lower extremity as a result of inactivity of the ankle pump mechanism. The rate of DVT with lower extremity casts is 1.8% in one study [9]. Several factors increase risk for thrombosis such as previous clotting problems, cancer, older age, obesity, heart failure. If patients have no risk factors then prophylactic anticoagulation is usually not necessary. Rate of DVT after arthroscopic procedures is very low. Currently, there are no guidelines for anticoagulation for arthroscopic procedures. The overall incidence of DVT was 0.5% among the 1410 knee arthroscopy patients and 3.1% among arthroplasty patients [10].

Hemorrhage

Hemorrhage and vascular injuries are rare in sports but can also be devastating complications. Most bleeding can achieve hemostasis with direct pressure. However, if the bleeding is severe or uncontrolled then electrocautery, chemocautery, and/or tourniquet use may be required. Tourniquets in the setting of acute hemorrhage and trauma on the sideline or in the emergency room can be a life-saving measure. In general, two hours is a reasonable duration to use a tourniquet to prevent long-term complications [11]. Prolonged tourniquet use is associated with muscle and nerve damage with around six hours of tourniquet use being associated with severe muscle damage and likely amputation [11].

Subdural hematoma is a life-threatening complication related to head injury. If there is parenchymal injury, the mortality rate can range between 35% and 50% [12]. These are caused by low-pressure venous bleeding into the subdural space after sudden brain movement and usually affect the elderly. Epidural hematoma is a high-pressure arterial brain bleed between the skull and the dura mater. Epidural hematomas usually present after a direct blow to the head. It can also present with a decreased level consciousness with a lucid interval. This lucid level can last for several hours and can lead to misdiagnosis. Subarachnoid hemorrhage is defined as bleeding between the arachnoid and pia mater. This type of bleeding can be spontaneous or traumatic. It is often associated with aneurysms, hypertension, and arteriovenous malformations. It is usually associated with severe headaches, which can be described as the patient's worst headache ever. Treatment for these intracranial bleeds require emergent care. In addition to intracranial hemorrhage, femur fractures can cause large amounts of bleeding. The thigh has a large potential space and can lead to life-threatening bleeding. Traction splinting is recommended to help with pain control and bleeding (Fig. 11.3). The rationale for decreased bleeding potential is that the thigh under traction is shaped like a cone with decreased area compared to a displaced fracture with shortening, which is more cylindrical. Intra-

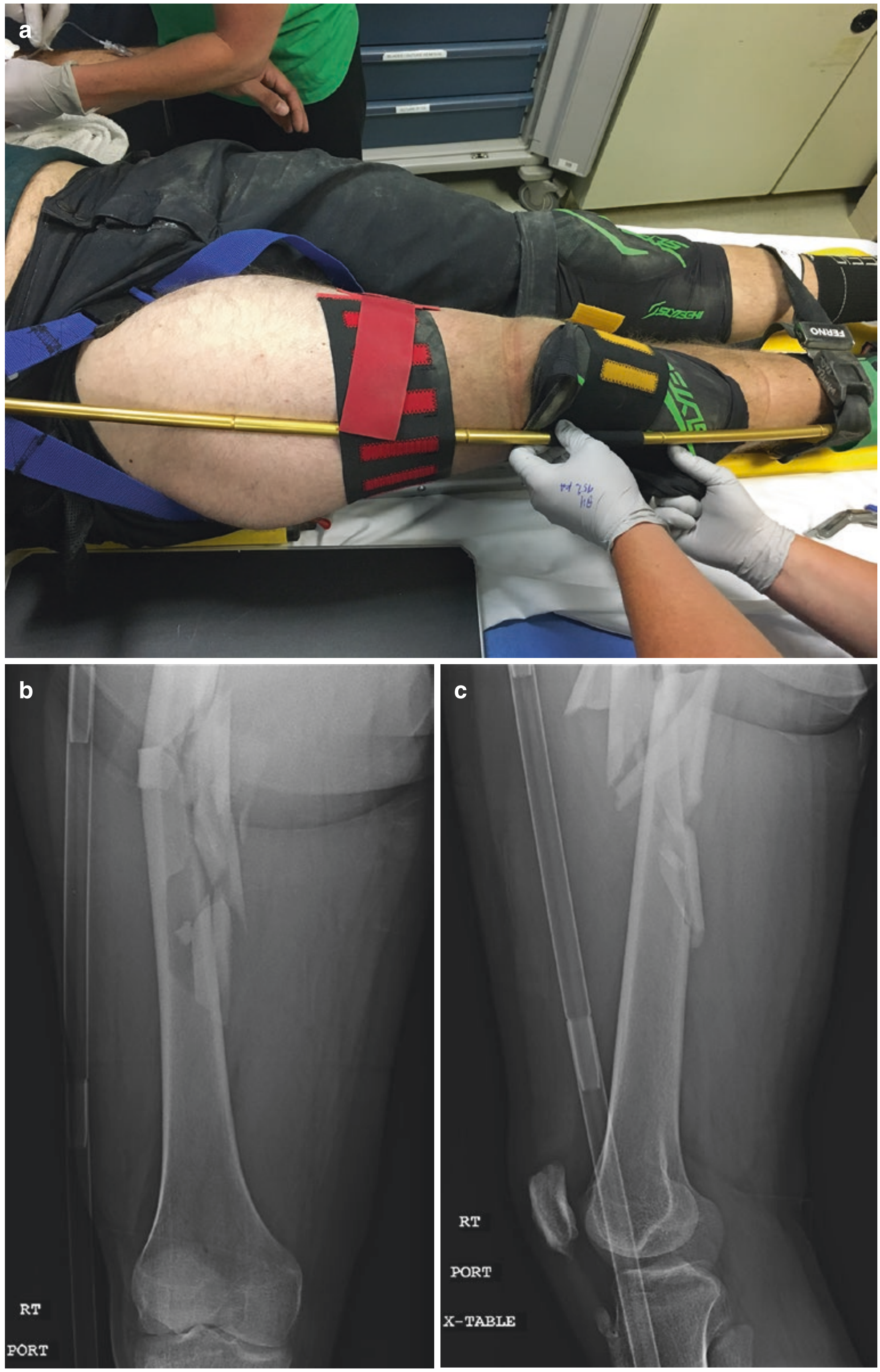

Fig. 11.3 Right comminuted mid-shaft femur fracture in a 32-year-old male as a result of a fall while mountain biking. Femur traction splint was applied (**a**). AP (**b**) and lateral (**c**) radiography views show the traction splint

abdominal bleeding is also a complication from sports-related trauma. Usually it is related to high velocity blunt force or penetrating trauma. Having a high index of suspicion and serial abdominal exams are important to diagnosing abdominal hemorrhage. Etiology of bleeding is usually from splenic laceration, but can occur with injury to the liver, small bowel injury, diaphragm, kidneys, or pancreas. In addition to an exam and labs, focused assessment with sonography for trauma (FAST) can be used to evaluate as a screening test for blood. Positive FAST exams in blunt

and penetrating trauma have a strong association with intraperitoneal bleeding. Blunt force trauma has a positive predictive value of 97.4%, and penetrating trauma has a specificity of 94.1–100% [13]. It is important to note that FAST exam is highly operator-dependent and can have a high false-negative rate (low negative predictive value). So, if there is concern for significant bleeding, more definitive imaging (CT scan) is required.

Acute Compartment Syndrome

Epidemiology

Acute compartment syndrome (ACS) is a rare, but serious complication from sports injuries. It is caused by either decreased muscle compartment volume or increase in pressure in a compartment. It can affect both the upper and lower extremity with common sites being the lower leg and forearm but can include other regions such as the foot, thigh (Fig. 11.4), and gluteal region [14]. Approximately one-third of cases result from closed tibial shaft fractures, and this is the most common cause of compartment syndrome. One-fourth of cases are secondary to soft tissue limb trauma, and around 20% of cases are related to radius-ulna shaft fractures [15]. Of all tibial diaphyseal fractures, around 7–8% developed acute compartment syndrome [16]. Patients who sustained a combined arterial and venous injury had a 41.8% likelihood of developing compartment syndrome, whereas the likelihood was 5.9% for an open fracture and 2.2% for a closed fracture [16]. The anterior compartment of the leg is the most common site for ACS. The compartment contains the four extensor muscles of the foot, the anterior tibial artery, and the deep peroneal nerve. Other possible causes are burns, blunt force trauma, and penetrating trauma. Male gender is a risk factors for developing ACS. The increase in incidence in males compared to females is significant, with the average annual incidence being 0.7 per 100,000 in women and 7.3 per 100,000 men [17]. Another risk factor for ACS includes younger age [18]. Other rare causes of ACS in sports-related trauma are ruptured baker's cyst, bleeding disorders, malignancies, and diabetes-associated muscle infarction [19, 20].

Clinical Presentation

The classic signs of ACS include the six "P's": pain, paresthesia, poikilothermia, pallor, paralysis, and pulselessness, with the most common complaint being worsening pain.

Fig. 11.4 Quadriceps tendon rupture (**a**) in a 65-year-old male as a result of a ski injury (**a**). Ultrasound confirmed (arrow) the diagnosis (**b**). He developed an acute thigh compartment syndrome requiring a fasciotomy (**c**)

Diagnosis

Anytime there is a limb that has a serious mechanism of injury that raises the risk for ACS, there should be a high index of suspicion, especially with severe pain as a component. Paralysis, pulselessness, and paresthesias usually present later in ACS and when present are usually associated with irreversible nerve and muscle damage. Therefore, paralysis, pulselessess, and parastesias should not routinely be a part of the initial diagnostic criteria for ACS. Patients with concern for possible ACS should have repeat physical examinations, and when there is concern they should be transferred to an emergency room for evaluation and possibly compartment pressure testing [21]. To test ACS pressures, after being zeroed, the pressure needle is usually inserted perpendicular to the skin into the muscle compartment in question. A small amount of sterile saline (usually about 0.3 mL) is injected into the compartment to ensure there is an appropriate pressure seal on the testing device, which will allow an accurate measurement.

Compartment pressures of greater than 30 mmHg have traditionally been used as criteria for decompression. However, various pressure levels have been considered as the criteria for surgical intervention. For example, if the compartment pressure is 20 mmHg but there is concern for ACS, this would be a reasonable patient for a fasciotomy [22]. Another consideration is continuous or interval pressure testing and looking at delta change, as most compartments have a resting pressure of 0–10 mmHg. There is evidence for decreased capillary perfusion at 20 mmHg and necrosis at 30 mmHg [23].

Initial Management

While covering a sporting event or working in an office setting, if there is concern for ACS, compartment pressure testing should be considered or the patient should be referred to a hospital for an urgent surgical consultation. Any dressing, splint, or cast should be removed. There is no benefit of elevating or lowering the limb and the limb should be maintained at heart level. Elevating the limb risks decreased arterial perfusion and lowering increases dependent swelling. Pain control, improving volume status if dehydrated, and supplemental oxygen are helpful measures. Fasciotomy is the definitive treatment.

Indications for Orthopedic Referral

As stated above any concern for ACS should be referred for emergent surgical evaluation.

Follow-Up Care

If ACS is treating promptly and appropriately, it usually has a good outcome. Of patients receiving a fasciotomy, there is however a high risk of complication ranging from 2% to 37% [24]. Complication rate was affected by co-morbidities, the type of procedure performed, the duration from onset of symptoms to surgery, and type of limb affected [25, 26]. In patients receiving fasciotomies complications include soft tissue necrosis, wound dehiscence, skin graft infection, missed or incomplete opened compartment, or need for tissue debridement [27]. Rhabdomyolysis may result from the muscle necrosis secondary to ACS. With rhabdomyolysis there is a significant release of myoglobin which can lead to acute kidney injury (AKI) [28]. The rate of rhabdomyolysis after ACS has been reported is 44.2% [28]. Of the patients that develop rhabdomyolysis, 14.4–39.1% develop AKI [28]. Unfortunately, amputation is also a complication of ACS with males and patients with vascular injury at higher risk [29]. Long-term consequences of compartment syndrome include chronic pain, disability from work, foot drop, and unsightly scars.

Return to Sports

After hospitalization, most athletes can return to activity once pain has improved and they have regained full function of their limb with full strength and range of motion.

Volkmann's Ischemic Contracture

Epidemiology

Volkmann's ischemic contracture (VIC) is a deformity with fixed flexion of the elbow, pronation of the forearm, flexion at the wrist, and finger extension. VIC is usually associated with delayed treatment of ACS and forearm/elbow fractures or burns [30]. It occurs at a rate of 0.5% in upper extremity pediatric long bone fractures.

Clinical Presentation

VIC presents as sequela from ACS or fracture. Presentation of Volkmann's contracture is usually based on the Tsuge classification as followed. Mild cases involve superficial volar group of muscles and result in flexion contractures of digits 2 and 3 with minimal sensory changes. Moderate cases usually involve deep flexor muscles and intrinsic muscles with median and ulnar nerves affected. Severe cases affect both flexor and extensor forearm muscles and results in severe deformity and atrophy of the forearm.

Diagnosis

Diagnosis is based on the clinical presentation and physical examination. When a patient presets acutely after an injury, the diagnostic process and index of suspicion is similar to compartment syndrome. Therefore, compartment pressure testing is useful in the acute phase. If the presentation occurs after the acute phase, then usually a diagnosis can be established using the Tsuge classification as above.

Initial Management

Treatment mainly focuses on prevention with emergent fasciotomy when increased compartment pressure occurs. Once Volkmann's contracture occurs, treatment is consistent with contracture treatment.

Indications for Orthopedic Referral

If Volkmann's contracture is suspected, then a multidisciplinary approach will be needed.

Follow-Up Care

Several different options exist for continued care including dynamic splinting, physical therapy, and contracture surgeries.

Return to Sports

Volkmann's contracture usually results in permanent disability and might preclude athletes from returning to full recovery.

Avascular Necrosis

Epidemiology

Avascular necrosis (AVN) is caused by an interruption of blood flow to bone. AVN usually affects the ends of long bones with the femoral head being the most common site. The shoulders and knees also have a relatively high risk of being affected. Anatomic suboptimal blood flow can also set up bones for AVN. The scaphoid and the base of fifth metatarsal bone have a higher risk, as they have perfusion distally to proximally. Other causes of disruption of blood flow can lead to AVN such as fractures, compartment syndrome, and fat embolism. There is a male predominance as men have a four times greater risk of developing AVN [31]. AVN is usually diagnosed between the third and fifth decades of life [32].

Approximately 20,000–30,000 new patients are diagnosed with AVN of the hip each year in the United States [33]. AVN can be post-traumatic or spontaneous. Post-traumatic necrosis occurs with hip fractures and hip dislocations. Risk factors for developing avascular necrosis includes glucocorticoids, alcohol, fat emboli, Gaucher's disease, radiation, hyperlipidemia, gout, HIV, renal failure, sickle cell, and coagulopathies. About 80% of spontaneous cases are a result of steroids and alcohol [34]. The pathogenesis of AVN from alcohol is related to fatty deposits in blood vessels disrupting blood flow.

Clinical Presentation

Avascular necrosis usually presents with worsening hip pain and decreased range of motion. It affects a patient's bilateral hips 80% of the time and accounts for 10% of all total hip arthroplasties.

Diagnosis

Standard views of plain radiography of the affected anatomic location such as pelvis with AP and frog-leg views can show subchondral collapse called a crescent sign (Figs. 11.5 and 11.6). Severe cases of AVN show femoral head collapse. Bone scans can show disease but is much less sensitive than MRI (56% vs. 100%) [35]. MRI is the current gold standard method of diagnosis and is helpful with predicting collapse based on severity of disease.

Initial Management

Treatment options are not entirely clear but consist of non-operative management, joint-preserving procedures, and joint replacement. Non-operative treatment can provide satisfactory improvement in pain and function in approximately 22% of cases [36]. There is some evidence that shows bisphosphonates can be helpful and could decrease the need for hip replacement [37]. One study had patients receive alendronate for 25 weeks and showed a decrease in rate of collapse of 29% in the placebo group to 2% in the alendronate group [38]. Other treatments that can be considered are statins, anticoagulants, iloprost, hyperbaric oxygen, and electrical stimulation.

Joint-preserving procedure success varies widely based on severity of disease, type of procedure, and surgeon's expertise ranging from 53% to 71% [39].

Fig. 11.5 Large focus of sclerosis, subchondral lucency, and articular collapse of right femoral head consistent with severe AVN in a 49-year-old male

Fig. 11.6 Right humeral head AVN in an 18-year-old male after traumatic posterior shoulder dislocation two years earlier

Unfortunately, hip replacement has poorer prognosis compared to hip replacement for hip arthritis. There seems to be a trend of improving outcomes with newer data. A recent study from 2016 with 149 patients with AVN and 133 patients with osteoarthritis had a revision rate of 19 and 6, respectively [39].

Indications for Orthopedic Referral

Prior to evidence of femoral head collapse, non-operative measures can be considered, but even early AVN can be considered for core decompression or other joint-preserving procedures. So, early referral to an orthopedic surgeon is recommended.

Follow-Up Care

Close follow-up is needed to assess for possible worsened hip disease.

Return to Sports

Could be considered early in disease but symptoms will largely dictate return to sport.

Fat Embolism

Epidemiology

Fat embolism is a very rare process that occurs when fat particles enter the bloodstream and pass into the small vessels of the respiratory syndrome or other sites such as the brain causing endothelial damage and symptoms related to vascular occlusion. It can occur with bone marrow transplantation, osteomyelitis, and pancreatitis, but usually is related to orthopedic surgery and trauma. The incidence varies but is reported around 3–4% of long bone fractures [40]. The exact incidence of fat embolism is unknown as the diagnosis is often overlooked or undetectable clinically.

Clinical Presentation

The main symptoms are respiratory insufficiency, and usually this is the initial presenting symptom. Additional findings are neurological abnormalities including irritability, agitation, confusion, delirium, seizure, and coma. Petechial rash is another known component which may also occur. Related sequelae from fat embolism are anemia, thrombocytopenia, and retinal scotoma.

Diagnosis

Having a high degree of concern for respiratory changes after an orthopedic surgery or major MSK trauma is important. Usually, symptoms will present in 24–72 hours after surgery or fracture. Most imaging studies will be negative, and therefore fat embolism syndrome is largely a clinical diagnosis, after excluding other etiologies for the patient's symptoms. MRI of the brain can show embolism syndrome lesions located usually in the deep white matter, brain stem, and cerebellum [40].

Initial Management

Treatment for fat embolism is largely focused on decreasing further risk of emboli and supportive care. In some cases, the respiratory symptoms are severe requiring ICU care and mechanical ventilation. Early immobilization decreases the risk of fat embolism. For severe cases of fat embolism, clinicians can consider a short course of high dose steroids. Preoperative steroids are not routinely given as side effects have not been shown to outweigh possible benefits [41]. The petechial rash related to fat embolism usually subsides in around a week.

Indications for Orthopedic Referral

Any suspicion for fat embolism or any patients with major MSK trauma such as femur fracture should be immediately transported to ED. If patients develop worsening hypoxia, dyspnea, sudden drop in hemoglobin, or if they have neurological changes, this is an indication for intensive care. Around 10% of cases develop respiratory failure [42].

Follow-Up Care

Most patients with fat embolism syndrome recover fully without long-term disease burden. Mortality rates have been widely variable but are likely around 1–2% [43].

Return to Sports

Once an athlete is pain free and has no residual symptoms, they likely would be cleared to return to their sport.

Fracture Blisters

Fracture blisters occur in areas where the skin adheres tightly to bone without overlying subcutaneous fat [44]. Of all fractures, there is a 2.9% incidence of fracture blister formation and 5.2% of fractures of the ankle, wrist, elbow, and feet as they are the common location of fracture blisters.

There are two types of fracture blisters: fluid-filled and blood-filled. Patients usually present with concern of new blister formation 1–2 days after trauma. There is a correlation of delayed time to surgery to increased risk of blister formation [45]. The diagnosis is mainly based on clinical presentation and blister formation.

Blister disruption should be avoided to decrease the risk of infection. If blisters do become disrupted then treating with appropriate wound care is needed. Usually, fracture blisters heal without further complication after 4–21 days [46].

Unless there is a major complication from a fracture blister, usually the fracture or acute process will be more limiting than the fracture blister.

Reflex Sympathetic Dystrophy

Epidemiology

Also called complex regional pain syndrome, reflex sympathetic dystrophy (RSD) is a syndrome that is associated with exaggerated response to an injury. There is a 4:1 ratio of female to male incidence with a median age of 46 [47]. It usually affects the upper extremity twice as frequently as the lower extremity. The most common cause of RSD is fractures, which cause 46% of cases [48]. There seems to be an increased incidence with HLA-A3, B7, and DR2 [45].

Clinical Presentation

It is usually associated with four clinical characteristics which include intense pain, vasomotor disturbances, delayed functional recovery, and various associated trophic changes.

Diagnosis

RSD is characterized by three stages [45]. Stage 1 begins with worsening pain without specific cause. The pain is characterized as a burning, throbbing pain and the limb feels cold. Plain radiography is usually negative. Stage 2 essentially shows worsened symptoms with thickening of the skin and muscle wasting. This phase usually occurs 3–6 months after the injury. Stage 3 is when the disease has progressed to severe disease. The patient will have a waxy skin appearance, contractures, and abnormal hair growth. Plain radiography will show severe demineralization. Overall RSD is a clinical diagnosis, but autonomic testing, standard imaging modalities, and sympathetic blockade can be used [45].

Initial Management

Prevention is important for RSD. Use of daily vitamin C (500 mg) has been shown to prevent distal radius fracture RSD [47]. Once RSD has occurred there are a wide range of pharmacologic treatments, such as NSAIDs, anticonvulsants, antidepressants, bisphosphonates, topical analgesics, nasal calcitonin, steroids, and alternative medical modalities [45].

Indications for Orthopedic Referral

If the patient has continued severe uncontrolled symptoms, surgery can be considered. Nerve blockage and spinal cord stimulators are procedures for severe RSD with limited efficacy [45].

Follow-Up Care

Usually until a patient's pain is well controlled, there needs to be frequent follow-up visits.

Return to Sports

If treated early, patients can have resolution of their symptoms. Children respond to treatment better than adults. Stage 3 of the disease is much more resistant to treatment.

Post-traumatic Arthritis

Arthritis can be an unfortunate complication of trauma. Post-traumatic arthritis can be attributed to around 12% of osteoarthritis cases [49]. It usually develops as a result of a joint injury and disruption of the articular surface. Post-traumatic arthritis can be a result of a single significant trauma or micro repetitive trauma. Different joints have varying predispositions to arthritis. Post-traumatic arthritis accounts for approximately 10% of all knee arthritis [50]. In general, ankle arthritis is less common than knee arthritis (10 times more common), but when it occurs it is rarely idiopathic. Varying studies suggest that 20–78% of all cases of ankle osteoarthritis are post-traumatic arthritis [51]. Intra-articular fractures develop arthritis with varying prevalence. For patients with acetabular fractures, 20% will develop post-traumatic arthritis after an average of 11.3 years [52]. About 44% of knee intra-articular fractures develop secondary traumatic arthritis with an average of 7.6 years follow-up [53].

In addition to fractures, ligament injuries can cause post-traumatic arthritis.

Following an ACL reconstruction at 10–20 years of age, 50% of patients will have osteoarthritis and functional impairment [54]. Another study looked at arthritis after a 14-year follow-up from ACL reconstruction. It showed an incidence of 57% of arthritis compared to 18% of arthritis on the contralateral knee, with the most common location being the medial compartment [55].

In a recent review by Chalmers et al., there was no difference in post-traumatic arthritis rate between conservative care and surgical ACL repair groups [56]. They included 1484 patients who were treated with ACL reconstruction and 685 who were treated non-operatively [56]. However, another meta-analysis by Ajuied et al. reported a higher rate of arthritis with non-operative care [57]. Reconstruction may also reduce the risk of subsequent meniscal injuries and improve passive flexion and extension of the knee [45].

Pediatric Considerations

Pediatric athletes have a significant improvement in bony remodeling and usually with appropriate care can get great functional outcomes. However, vascular and nerve complications from injuries or fractures can cause chronic issues. For example, with displaced supracondylar humerus fractures acute neurovascular complications occurred in 17% of patients [58]. Nerve damage is the most common complication with 10% of patients [58]. The median nerve is most commonly affected by supracondylar fractures [58].

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Part III

Acute Sports-Related Bones and Joints Trauma: Upper Extremity

Morteza Khodaee

12 Clavicle

Armando F. Vidal, Mark F. Riederer, Justin H. Bartley, K. Linnea Welton, Richard Bortz, and Morteza Khodaee

Key Points

- The clavicle, sternoclavicular (SC), and acromioclavicular (AC) joints serve as the attachment of the shoulder to the axial skeleton.
- The clavicle is the strut of the shoulder.
- Injuries to the AC joint and mid-shaft clavicle fractures are very common in contact and collision athletes.
- Most injuries to the AC joint and fractures of the mid-shaft of the clavicle can be treated non-operatively.
- Posterior dislocations of the SC joint warrant urgent evaluation due to the potential injury to the mediastinal structures including vascular injury.

A. F. Vidal
The Steadman Clinic, Steadman Philippon Research Institute, Vail, CO, USA
e-mail: avidal@thesteadmanclinic.com

M. F. Riederer
Department of Orthopaedic Surgery, University of Michigan, C.S. Mott Children's Hospital, Ann Arbor, MI, USA
e-mail: mriedere@med.umich.edu

J. H. Bartley
Department of Orthopedic Surgery, Lovelace Hospital, Albuquerque, NM, USA

K. L. Welton
Department of Orthopedic Surgery, MultiCare Health System, Auburn, WA, USA
e-mail: klwelton@multicare.org

R. Bortz
Winter Park Medical Center, Denver Health, Winter Park, CO, USA
e-mail: Richard.bortz@dhha.org

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Introduction

Sports-related clavicle injuries are common, particularly in contact and collision sports. These injuries include fractures, sternoclavicular (SC) and acromioclavicular (AC) joints sprains, subluxations, and dislocation.

Anatomy

The clavicle is a double S-shaped bone that articulates with the sternum at the sternoclavicular (SC) joint (Fig. 12.1) and the scapula at the acromioclavicular (AC) joint (Chap. 14; Fig. 14.1b). It serves at the attachment of multiple muscles, including the trapezius, deltoid, pectoralis major, subclavius, sternocleidomastoid, and sternohyoid (Fig. 12.2). It is the strut of the shoulder and connects it to the axial skeleton. It protects the underlying neurovascular structures from the neck down the arm, including the subclavian vein and artery. It is the first bone to ossify in fetal development at around 5 weeks of gestation, and the medial clavicular physis is the last to close, rarely before 20–25 years of age [1]. The SC joint is a diarthrodial joint with an intra-articular disc, capsule, and surrounding ligaments (Fig. 12.1). Although there is very little bony stability of the SC joint, the strong capsular, costoclavicular, and infraclavicular ligaments provide the majority of the stability. The AC joint is also a diarthrodial joint with an intra-articular disc, similar to the SC joint (Fig. 12.1). It is supported by a capsule and surrounding ligaments. The coracoclavicular (conoid and trapezoid), acromioclavicular (anterior, posterior, inferior, and superior), and coracoacromial ligaments are static stabilizers (Fig. 12.1), and the deltoid and trapezius muscles are dynamic stabilizers (Fig. 12.2).

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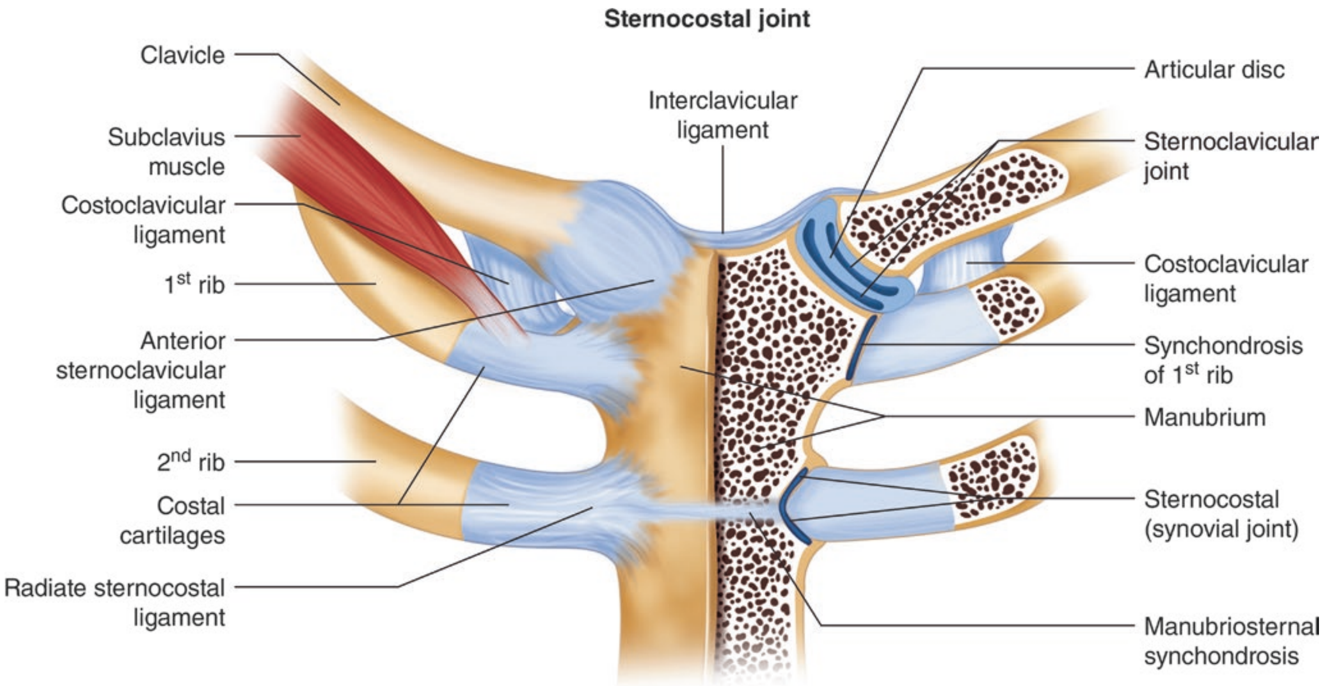
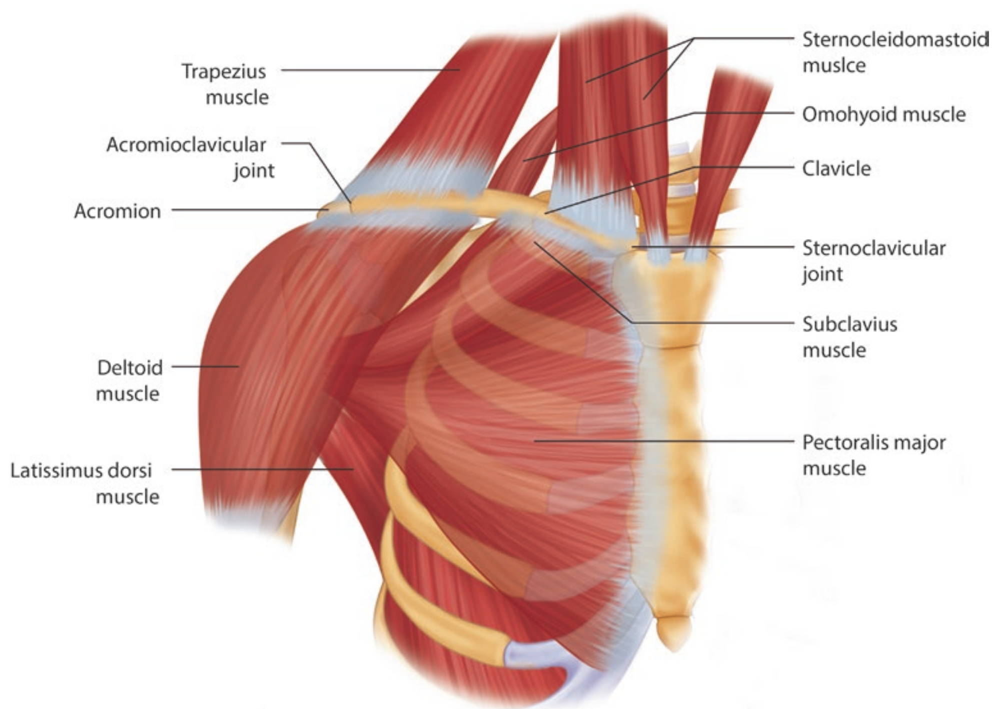


Fig. 12.1 Medial clavicle ligamentous anatomy

Fig. 12.2 Muscular attachments to the clavicle



Fractures

Mechanism of Injury in Sports

Historically, the mechanism of injury for a clavicle fracture was thought to be a fall onto an outstretched hand (FOOSH). However, it seems that direct fall onto the shoulder is the main mechanism of injury [1]. With regard to fractures to the medial clavicle, most injuries occur from high-velocity trauma, such as motor vehicle collisions. There are no reported studies of medial clavicle fractures in sports; however, high-speed sports (e.g., cycling, skiing, snowboarding) and collision sports (e.g., American football, ice hockey, and wrestling) have the highest risk for these injuries.

Epidemiology

Clavicle fractures are common in the general population. Up to half of clavicle fractures are sports-related [2–5]. Cycling, skiing, snowboarding, soccer, rugby, and American football are responsible for the majority of sports-related clavicle fractures [2–5]. Clavicle fractures are more common among men, particularly among young men [2–5]. Mid-shaft fractures are the most common type accounting for about 80% of all clavicle fractures [1, 2, 6]. Fractures of the medial (proximal) clavicle are uncommon, only representing <5% of all clavicle fractures [6–8]. In children, medial clavicle fractures are even less common. It seems that many medial clavicle fractures are missed on plain radiography [6].

Fracture Classification

Allman developed the most commonly used clavicle fracture classification in 1967 [9]. Using this classification system, the clavicle is broken up into thirds, creating three groups with different characteristics. One group (middle third), occurs most frequently, one group (distal third) heals least frequently, and one group (proximal third) heals the best and displaces the least [9].

In 1968, Neer further classified distal (lateral) clavicle fractures into two principal types in order to recognize the contribution of the coracoclavicular (CC) ligaments to the stability of the medial fracture segment [10]. In Type I, the fracture occurs distal to CC ligaments with minimal or no displacement (Fig. 12.3). In Type II, the medial fragment is discontinuous with CC ligaments. In Type III, there is an intra-articular fracture of the AC joint with intact CC ligaments.

Clinical Presentation

Despite its subcutaneous nature with little soft-tissue protection, open clavicle fractures are uncommon [11]. In a cohort study which included high-energy trauma, only 2 out of 535 isolated clavicle fractures were open [7]. It seems that the incidence of open, sports-related fractures of the clavicle is even less common. Patients will typically present with the

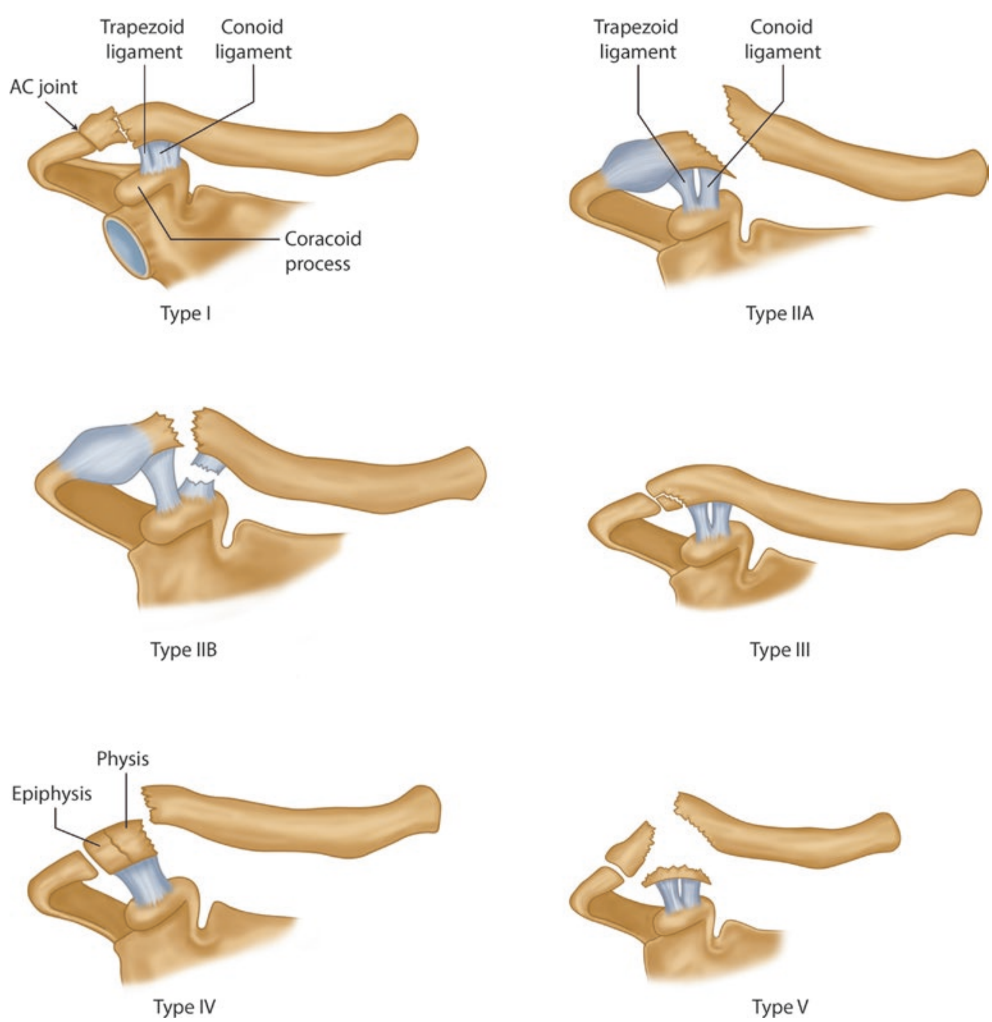


Fig. 12.3 Neer classification of the distal clavicle fracture

involved arm splinted against the side with some degree of drooping (ptosis) of the shoulder [11]. In the sports-related setting, the skin overlying the fracture must be exposed; therefore, protective gear such as shoulder pads must be removed to allow for careful inspection for open fracture or skin tenting (Fig. 12.4) that can lead to pressure necrosis. The area of injury is often evident with ecchymosis, swelling, or deformity (Figs. 12.4 and 12.5). Gentle palpation can help assess for level of comminution, displacement of the fracture fragment, or segmental injury. Sensorimotor testing of the involved extremity is necessary, paying special attention to the function of the ulnar nerve since the medial cord of the brachial plexus has been proposed to be closest to the fractured clavicle [12]. Range of motion and strength testing can be performed if tolerated, and the immediate post-injury examination should include auscultation of the lung fields to evaluate for pneumothorax. In addition, peripheral pulses should be palpated for strength and symmetry with any difference prompting blood pressure comparison, followed by further vascular work-up if concern exists [13]. Vascular complications associated with clavicle fractures, which may occur early or late, are usually evident by loss of pulse, pulsating hematoma, thrombus, numbness, coldness, weakness, or skin discoloration [14].

Diagnosis

The standard plain radiography AP view of the clavicle, including the entire clavicle, is often sufficient to make the diagnosis (Figs. 12.4, 12.5, 12.6, 12.7, 12.8, 12.9, 12.10, 12.11, and 12.12). However, multiple views with oblique imaging can be useful to allow accurate estimate of displacement, shortening, and characterization of the fracture. A posteroanterior (PA) 15° caudad oblique radiograph has been proposed to limit magnification error and provide a more accurate estimate of shortening at the fracture site [15]. For medial clavicle fractures, a 40° (Serendipity) cephalic tilt

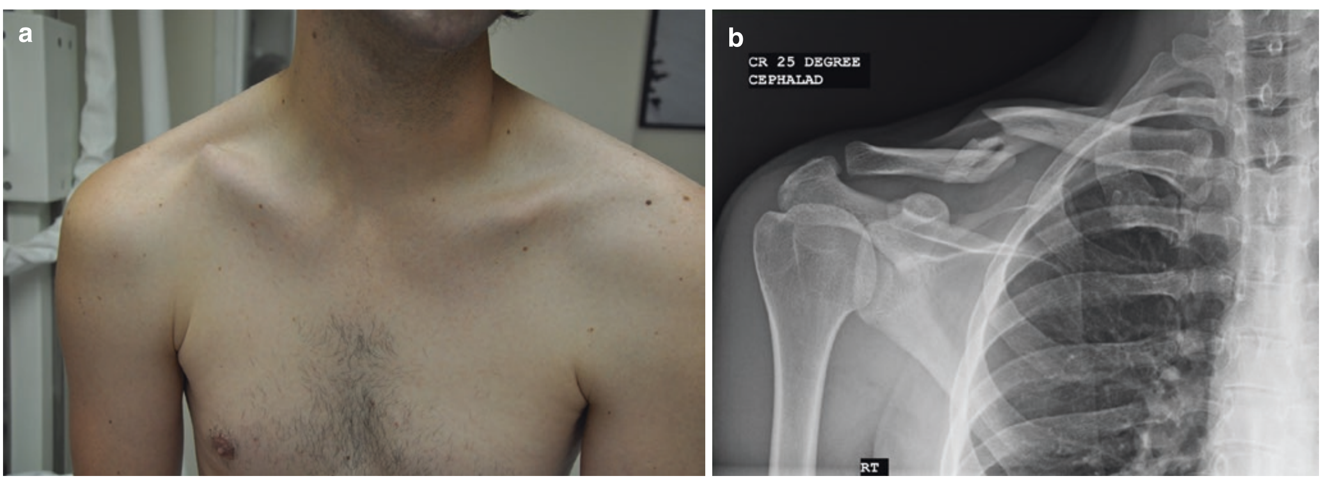


Fig. 12.4 Right clavicle tenting (**a**) due to a comminuted and displaced mid-clavicle fracture (**b**) in a 37-year-old male as a result of a mountain bike injury

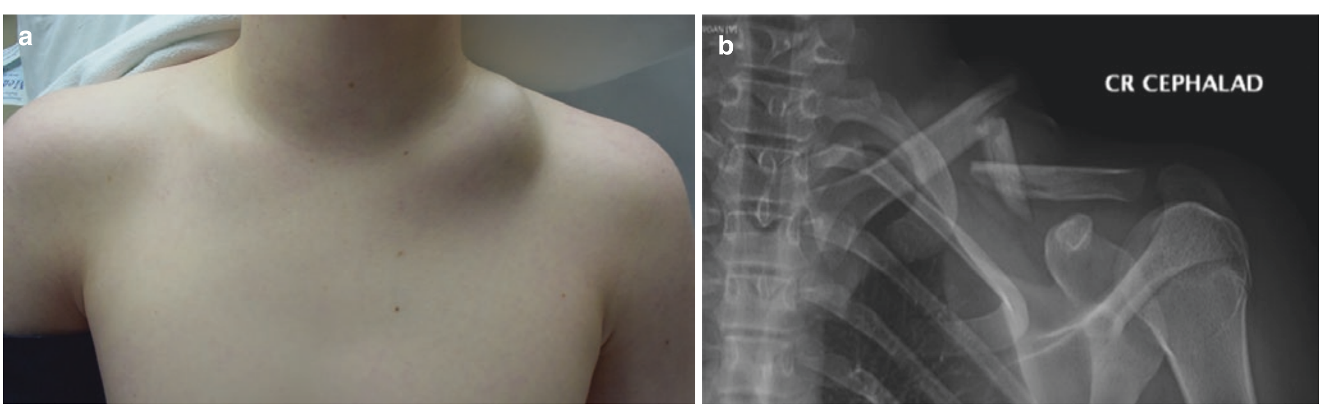


Fig. 12.5 Left clavicle deformity (**a**) due to a comminuted and displaced mid-clavicle fracture (**b**) in an 18-year-old male as a result of a snowboarding injury

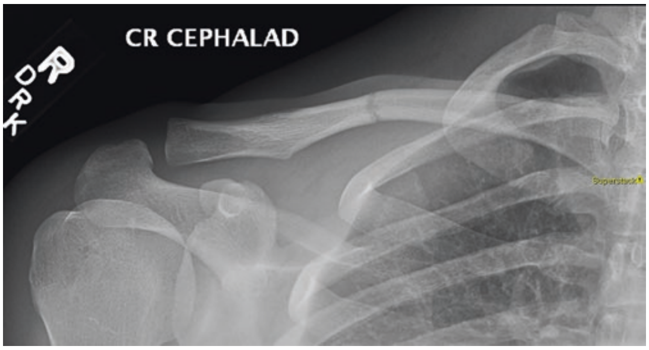


Fig. 12.6 Right non-displaced mid-clavicle fracture in a 20-year-old male as a result of a snowboarding injury

view of the bilateral medial clavicles and SC joints is most helpful. In addition, an upright chest X-ray should be conducted if a pneumothorax is suspected. CT is most accurate to evaluate for shortening at the fracture site (Fig. 12.7), but this modality is seldom necessary unless evaluating for nonunion [13, 16, 17]. If plain radiography is not diagnostic, CT scan is recommended for suspected fractures of the medial clavicle. However, the CT scan should be obtained if only the results may change the treatment.

Initial Management

The vast majority of clavicle fractures can initially be managed conservatively. Assuming the fracture is closed and the patient is neurovascularly intact, the patient can be placed in a sling. There are multiple methods of immobilization and sling treatments devised for the non-operative treatment of displaced clavicle fractures. The number of treatments attests to the difficulty of achieving and maintaining reduction [10, 13, 14, 16, 17]. Despite multiple studies comparing different immobilization techniques for the closed treatment of clavicle fractures, ultimately there is no superior method of immobilization [18–20]. In a randomized control trial comparing figure-of-8 with sling, there was no significant difference in functional or radiographic outcomes [18]. In addition, the figure-of-8 brace was poorly tolerated by patients, thus favoring sling treatment in most patients [18].

Indications for Orthopedic Referral

In general, immediate orthopedic involvement is recommended for skin tenting, open fractures, presence of neurovascular compromise, polytrauma, or floating shoulder [12, 14, 16]. Due to the risk of nonunions, shoulder dysfunction, and residual pain, all athletes with displaced clavicle fracture should be referred to a sports medicine clinic for a thorough discussion of treatment options [11, 13, 14, 16, 20–28].

For centuries, it has been believed that clavicle fractures require little more than benign neglect by clinicians [13, 28]. Neer's published series of 2235 patients in 1960 supported this belief, since non-operatively treated middle-third clavicle fractures were associated with a nonunion rate of 0.13% versus 4.6% in the 45 patients that were treated operatively [29]. Robinson et al. published a nonunion rate of 4.5% in 581 non-operatively treated middle-third clavicle fractures, but also revealed significant risk factors for nonunion including advancing age, female gender, displacement of the fracture, and the presence of comminution [30]. A systematic review published in 2005 emphasized the effect of these risk factors by publishing a nonunion rate of 15% in 159 displaced, mid-clavicle fractures that were treated non-operatively [31].

A multicenter, prospective randomized clinical trial of 132 patients conducted by the Canadian Orthopedic Trauma Society (COTS) studied the difference in outcomes between operative (open reduction and internal fixation [ORIF]) and non-operative (sling) treatment of displaced mid-shaft clavicle fractures [21]. They reported that Constant-Murley and DASH (The Disabilities of the Arm, Shoulder, and Hand) scores significantly improved, mean time to radiographic union significant decreased, nonunions significantly decreased, symptomatic malunions significantly decreased, and patients were more likely to be satisfied with appearance of the shoulder and with the shoulder in general following surgical treatment [21].

However, a new Cochrane systematic review concluded that the evidence for suggesting non-operative or operative management of mid-clavicle fractures are inadequate [24]. Based on low-quality studies, there were no additional benefits (e.g., function, pain, and quality of life) of operative treatment compared with non-operative treatment [24]. Operative management possibly causes fewer treatment failures compared with non-operative treatment [24]. Therefore, treatment options should be individualized after thorough discussion of the relative benefits and risks of each intervention with athletes [11, 13, 22, 24, 28]. Relative operative indications for patients are summarized in Table 12.1.

Most medial clavicle fractures are stable and can be managed non-operatively [16, 22]. Indications for surgical intervention are summarized in Table 12.1. Most distal third clavicle fractures are non-displaced and do not involve the AC joint. Under these circumstances, the preferred treatment is non-operative management, and excellent clinical and radiographic outcomes can be expected [11, 22, 34, 35]. Critical to the stability of a distal clavicle fracture is the integrity of the coracoclavicular (CC) ligament. When intact, the CC ligament provides stability to the medial fracture fragment [35]. However, when compromised, concomitant CC ligament injury leads to displacement of the medial fracture fragment [35]. In this scenario

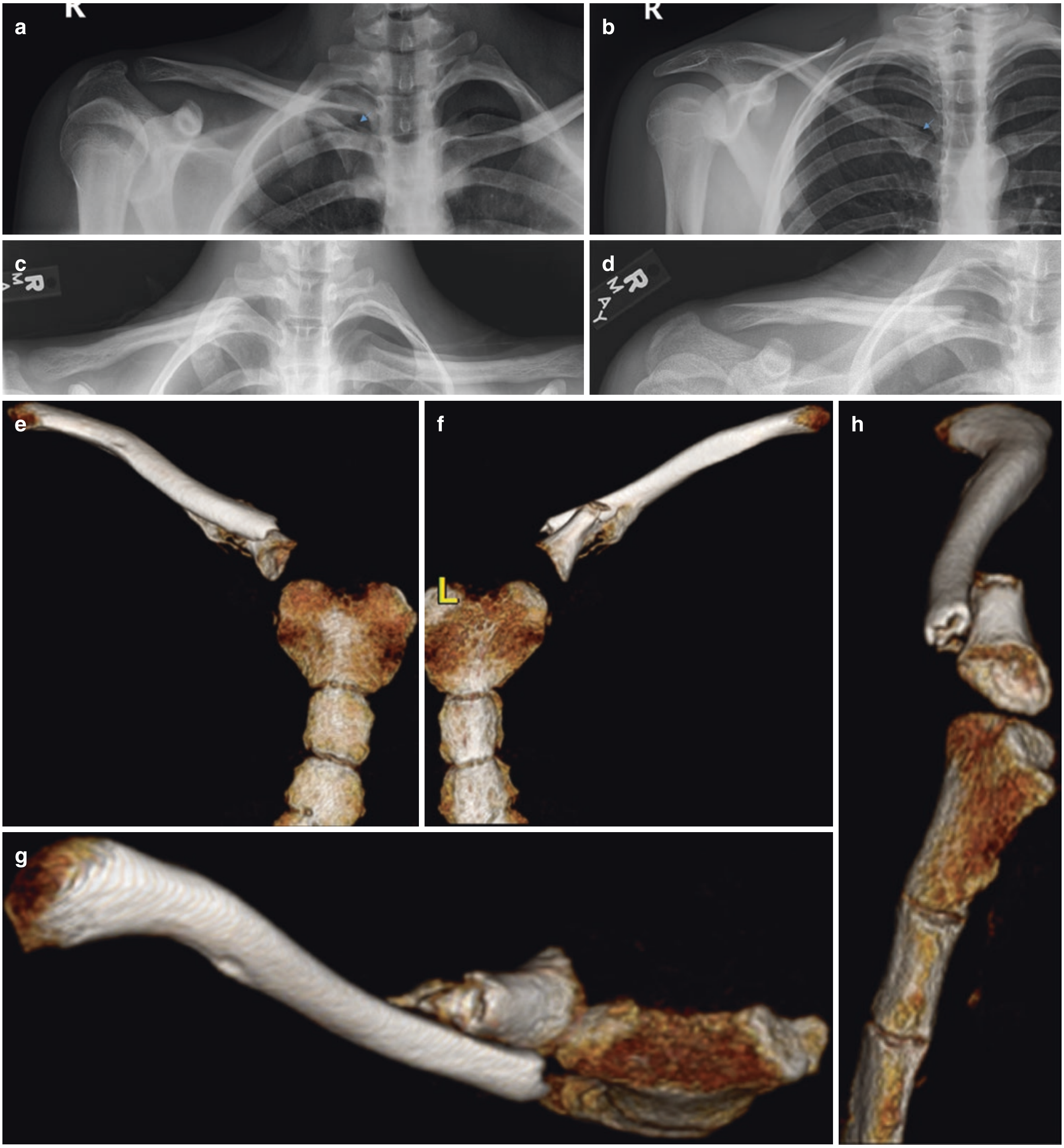


Fig. 12.7 A right displaced medial clavicle fracture (arrows) in an 18-year-old male as a result of a collision with another player while playing basketball (**a**, **b**). Plain radiography (**c**, **d**) and 3D CT images (**e**–**h**) a month later revealed mild callus formation with the extend of the displacement

(Neer Type 2 distal clavicle fracture), nonunion rates can be as high as 30% [10, 11, 34, 35]. Surgical treatment recommendations (Table 12.1) in the setting of displaced, distal third clavicle fractures are based on stability of the fracture segments, displacement of the fracture fragments, and patient age [11, 22, 35].

Follow-Up Care

For non-operative treatment of clavicle fractures, follow-up should be every 2–3 weeks to assess clinical healing (e.g., pain, function). Immobilization with a sling or figure-of-8 brace should be for about 2–4 weeks or until pain allows the

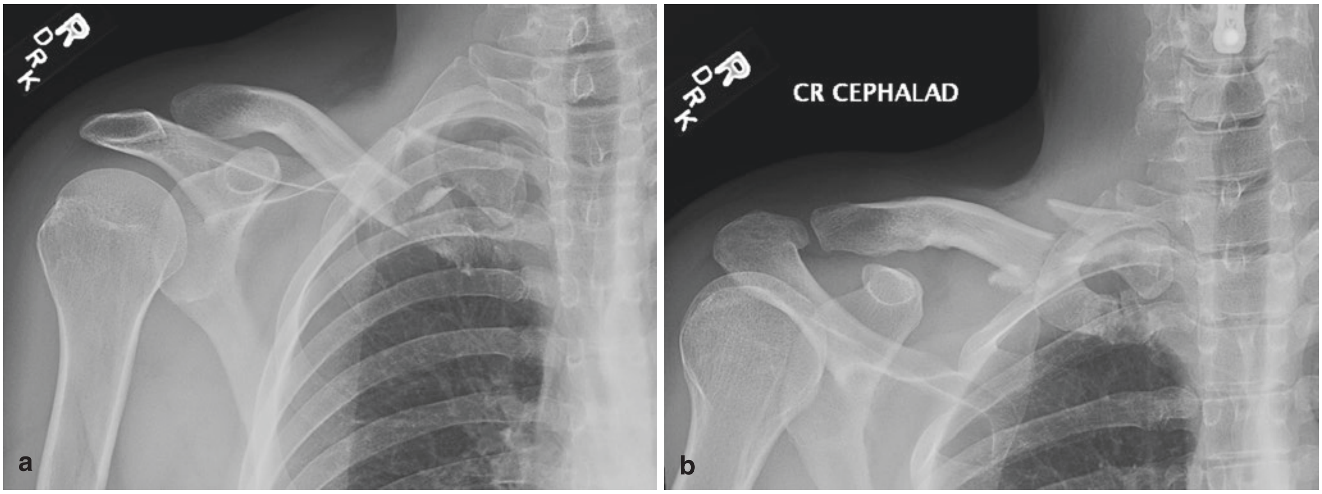


Fig. 12.8 A comminuted and displaced right medial clavicle fracture (**a**, **b**) in a 36-year-old male as a result of a mountain bike injury

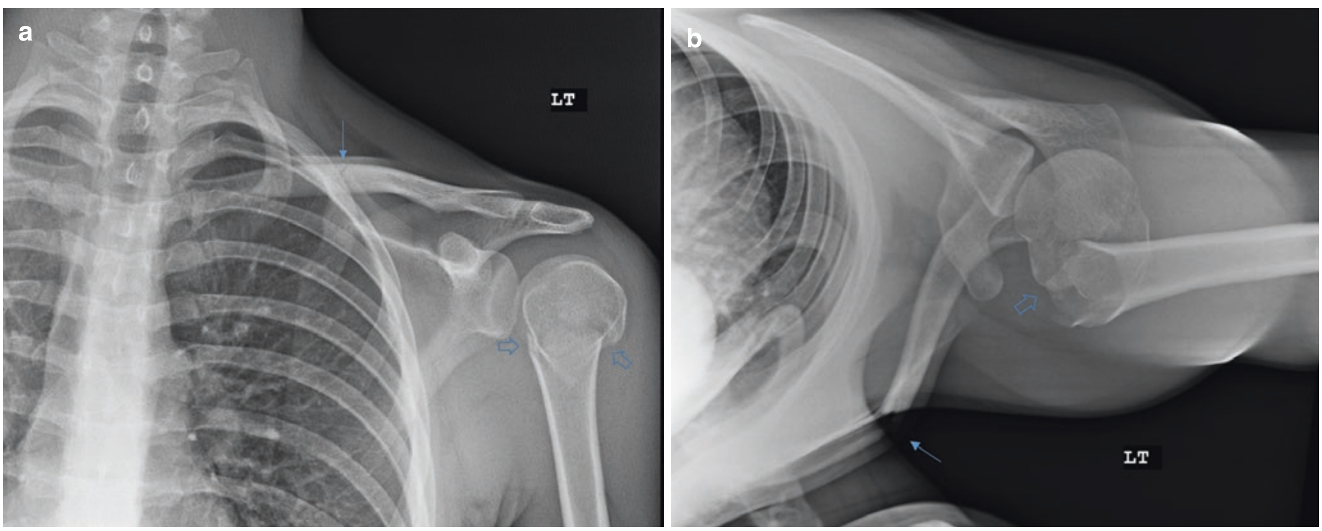


Fig. 12.9 Left non-displaced mid-clavicle (arrows) and severely displaced humerus neck (open arrows) fractures in an 18-year-old male as a result of a snowboarding injury (**a**, **b**)

Fig. 12.10 A distal left clavicle fracture (Type IIA) in a 37-year-old male as a result of a ski injury

Fig. 12.11 A distal left clavicle fracture (Type IIB) in a 61-year-old male as a result of a direct fall on his shoulder

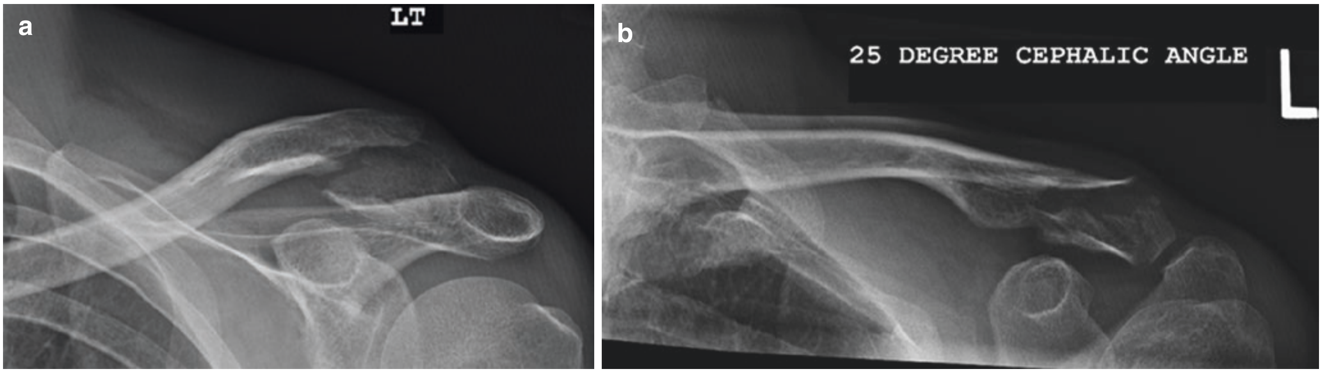


Fig. 12.12 A distal left clavicle fracture (Type III) in a 40-year-old male as a result of a fall (**a**, **b**)

Table 12.1 Management guidelines for clavicle fractures [11, 13, 16, 17, 22, 27, 28, 32, 33]

Fracture category	Proximal (medial)	Middle	Distal (lateral)
Length of immobilization (sling)	2–6 weeks	2–8 weeks	2–8 weeks
Indications for urgent orthopedic referral	Skin tenting, open fractures, neurovascular compromise, polytrauma, floating shoulder	Open fractures, fracture with posterior SC dislocation, neurovascular compromise, polytrauma	Open fractures, fractures with irreducible GHJ dislocation, neurovascular compromise, polytrauma
Indications for operative management discussion	Comminuted fractures, >100% displacement, displaced intra-articular fractures, infection	Comminuted fractures, >100% displacement, shortening >2 cm, vertical fragment, infection	Comminuted fractures, displaced unstable (Neer II or V) fractures, infection
Follow-up	Every 2–3 weeks	Every 2–3 weeks	Every 2–3 weeks
Healing time	6–8 weeks	6–10 weeks	6–8 weeks
Return to sports	2–4 months	2–4 months	2–4 months

beginning of gentle active range of motion exercises [16, 27, 28]. The expected time course for healing of clavicle fractures is 6–8 weeks [6, 16]. Repeat radiographs are performed only after full clinical healing. There is no consensus on the duration of immobilization or rehabilitation protocol following a clavicle fracture [16, 18, 20, 23, 27]. However, athletes should be allowed to start passive range of motions (<90° in abduction and forward flexion) as soon as they can tolerate it. Strengthening can be started at 4–6 weeks [16, 20, 23, 27, 28].

Postoperatively, athletes can be placed in a sling for 1–4 weeks with immediate restrictions including no elevation of the arm >90° in any plane for the first 2–4 weeks, no lifting objects >2 kg for the first 4–6 weeks, and avoiding repetitive reaching for the first 4–6 weeks [11, 22, 28, 36, 37]. Icing the shoulder 3–5 times a day in 15 minutes' intervals can help control swelling and inflammation. Rehabilitation program varies based on the type of treatment (e.g., non-operative, plate fixation, or intramedullary nailing), surgeon's preference, and type of sports [11, 22, 28]. Physical therapy can be started as early as 1 week after the surgery [11, 22, 26, 28]. This can include pendulum exercises, triceps extensions with a resistance band, isometric exercises with the arm at the side, and stationary bike as tolerated. During the second phase of rehab (2–6 weeks postoperative), patients can undergo isometric scapular proprioceptive neuromuscular facilitation stretching as well as working on gentle exercises to improve range of motion and strive for progressive gains of active motion up to a goal of 90° of shoulder forward flexion and abduction [11, 22, 28]. During the 6–8 weeks of postoperative period, athletes can start working on mid-range of motion, as well as active and light resistance exercises through 75% of the patient's range of motion [11, 22, 26, 28]. Athletes usually demonstrate full active shoulder range of motion at 8–12 weeks postoperatively [11, 22, 26, 28]. At 12 weeks postoperatively, athletes can focus on progressive strengthening and should be able to return to non-contact sports [11, 22, 26, 28]. This should be determined by the physical therapist through functional testing specific to the injury and sport.

Return to Sports

There is limited literature on the return to sport following clavicle fractures [24, 33, 38]. Surgical treatment of clavicle fractures in adult athletes seems to lead to a faster return to sport [11, 24, 26, 38]. In general, athletes should be able to start a return to sports protocol as soon as symptoms allow [26, 32]. Athletes should be asymptomatic with normal range of motion and strength to be able to return to sports. Depending on the type of fracture, athletes' age, treatment, and specific sports, most athletes are able to return to sports in 2–4 months [11, 24, 26, 32, 33, 38].

Complications

Complications and outcomes from non-operative care include nonunion (Fig. 12.13), malunion, cosmetics (deformity), and functional deficits [5, 12–14, 16, 21]. Risk of nonunion is higher with significant clavicle shortening (>2 cm), fracture comminution, significant fracture displacement, female, and older age [16, 28]. Complications with operative management are dependent on the chosen method of fixation. Complications associated with plate fixation occur up to 23% of the time and include deep infection, plate breakage, nonunion, symptomatic hardware, and re-fracture following plate removal [28, 39]. In addition, there is concern for a greater risk to underlying neurovascular structures with use of a plate [28, 31, 39, 40]. If hardware removal is considered, complication and reoperation rates may be as high as 43% and 25%, respectively [28, 39]. Anatomically contoured clavicle plates may reduce the need for hardware removal [36, 41].

Following intramedullary nailing of the clavicle, the reported complications (~9%) include implant breakage, skin breakdown, and temporary brachial plexus palsy [28, 42, 43]. There are very little reports of complications with medial clavicle fractures. It seems that the nonunion is rare. There is an increased risk with complete fracture displacement [22]. Complications from surgery are primarily anecdotal, as surgical fixation of these fractures are uncommon [30].

Pediatric Considerations

Most pediatric clavicle fractures are non-displaced or mildly displaced (Figs. 12.14 and 12.15). Torus fractures are extremely rare [44]. In pediatric patients, almost all middle and distal (lateral) third clavicle fractures can be treated non-operatively due to their inherent remodeling capability [11, 45–47]. As there is a lack of high-quality randomized control trial studies, most recommendations are based on level B evidence (at most) [46, 48]. In general, it seems that both operative and non-operative management of displaced middle-third clavicular fractures (Fig. 12.16) have good functional outcomes [46]. Nonunion is extremely rare in pediatric population with clavicle fractures regardless of the operative of

Fig. 12.13 Left chronic mid-clavicle nonunion in a middle aged man. Other than a non-tender deformity, he had normal range of motion and strength years after his original mid-clavicle fracture

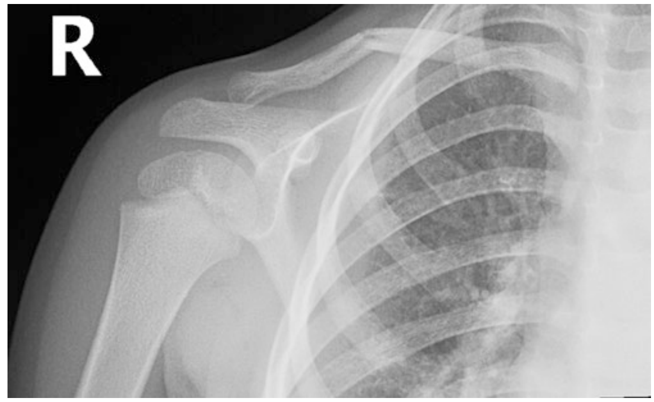


Fig. 12.14 A mildly angulated right mid-clavicle fracture in a 3-year-old boy as a result of a ski injury

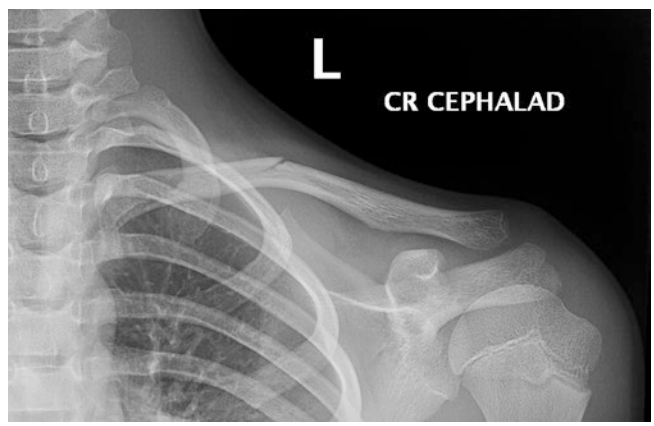


Fig. 12.15 A mildly angulated left mid-clavicle fracture in a 14 year-old boy as a result of a ski injury

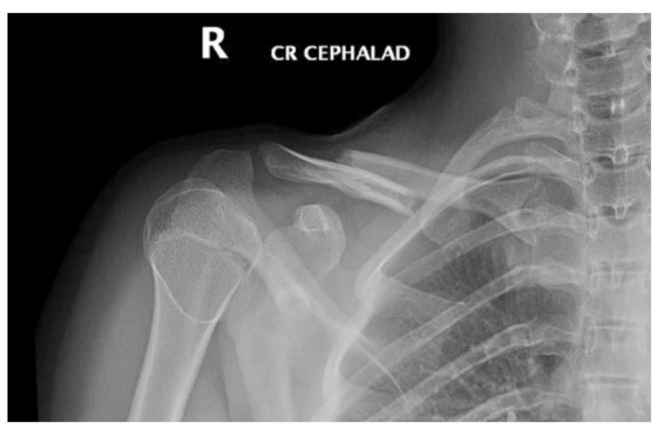


Fig. 12.16 Right displaced mid-clavicle fracture in a 16-year-old male as a result of a fall

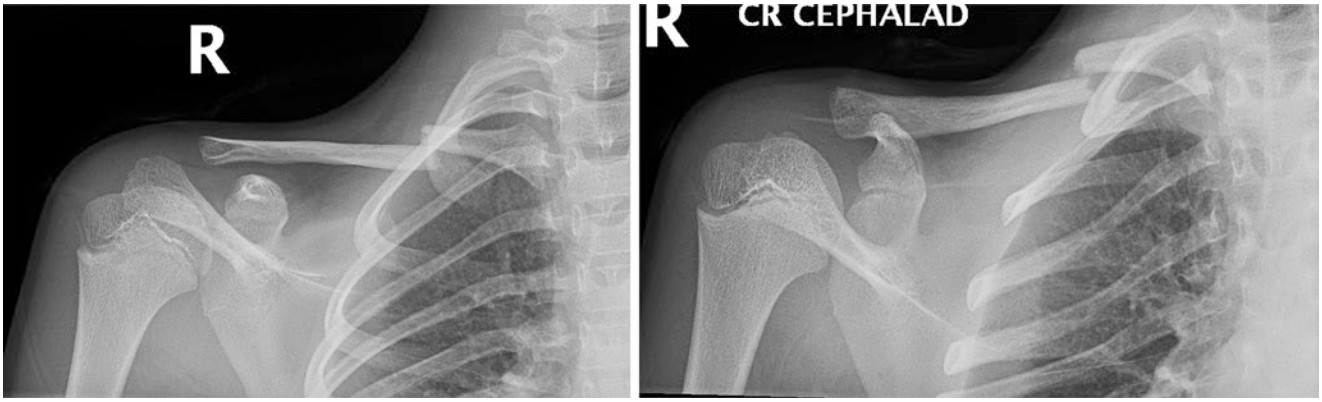


Fig. 12.17 A displaced right medial clavicle fracture in an 11-year-old boy as a result of a ski injury

non-operative treatment [45–48]. Adolescents with significant clavicle fracture displacement should be referred to sports medicine for a thorough discussion on benefits and risks of operative versus non-operative management [46, 47, 49]. Most injuries of the lateral aspect of the clavicle involve physeal separation, since the AC joint and CC ligaments are more robust than the physis [11]. These physeal injuries can be further delineated using CT imaging, but the majority demonstrate significant potential for healing and remodeling with non-operative care [49]. Most fractures of the medial clavicle in children and adolescents are physeal fractures. The medial epiphysis does not ossify until 20 years of age and rarely fuses before age 25. As with middle and distal clavicle fractures, many medial clavicle fractures (Fig. 12.17) can be treated non-operatively due to their high remodeling potential.

Sternoclavicular (SC) Joint Injuries

Mechanism of Injury in Sports

Traumatic injuries to the SC joint can occur by direct or indirect forces, the latter being the more common of the two. Although the majority of these traumatic injuries occur from motor vehicle collisions (>80%), contact and collision sports can pose risk to injury to this joint [50, 51]. Such high-risk sports include American football and rugby. A direct force to the anteromedial clavicle with the arm held in adduction and flexion can cause a posterior displacement of the medial clavicle within its articulation with the SC joint [50, 51]. A common sports scenario for this is when an American football player is at the bottom of a “pile on” after a tackle. Anterior displacement is far more common than the posterior displacement and is generally from an indirect force applied to the anterolateral clavicle while the shoulder is rolled backward and held in an abducted position [50, 51]. Both injuries can occur while being tackled or from a fall onto the shoulder in American football, rugby, martial arts, or in collisions in ice hockey or basketball. Protective shoulder pads in American football reduces this risk.

Epidemiology

Sternoclavicular joint injuries are very uncommon, especially in sports-related shoulder girdle injuries [50]. It only counts for <1% of dislocations in the body [52]. This is likely due to the fact that although there is inherent bony instability of the joint, the strength and integrity are provided by the strong surrounding ligaments, particularly the capsular ligament [50, 51]. As previously stated, anterior injuries are more common, accounting for approximately 95% of these injuries, and posterior injuries account for the remaining 5% [50, 51, 53, 54].

Sprain Classification

Classification of sternoclavicular joint injuries is based upon the degree of injury to the surrounding ligaments and displacement of the articulation of the medial clavicle to the SC joint (Fig. 12.1). Type I injuries are mild sprains in which the SC joint is stable and the surrounding ligaments are intact [50, 51, 53]. Type II injuries are moderate sprains with subluxation of the medial clavicle in relation to the SC joint, and there is partial disruption of the surrounding ligaments. The joint may sublux with manual stress [50, 51]. Type III injuries are severe sprains and gross dislocation of the medial clavicle within the SC joint [50, 51, 53, 54]. The capsule and surrounding ligaments are completely torn.

Clinical Presentation

Athletes with injuries to the SC joint may have deformity, swelling, tenderness, pain, and bruising at the site of the SC joint (Figs. 12.18 and 12.19). They will commonly hold the

affected arm adducted and to the side. A palpable step-off and a ballottement may be felt at the medial clavicle [50]. It is important for the examining physician to examine the athlete unclothed, with all protective padding off, and to compare the affected SC joint with the normal SC joint. In addition, athletes with posterior dislocations may have impingement of posterior mediastinal structures and may complain of dyspnea, dysphagia, or have findings such as tachypnea and venous congestion in the ipsilateral arm and neck [50, 51, 53, 55]. They may also complain of numbness and tingling which may suggest compression of the brachial plexus. Anterior injuries may exhibit prominence of the medial clavicle. The examining physician must have a high index of suspicion for posterior SC joint injuries due to their high risk for injury to the posterior vascular structures and may need more immediate referral for evaluation. In severe cases, athletes may have underlying shock or pneumothorax.

Fig. 12.18 Left anterior SC joint injury (Type II) in a 40-year-old female as a result of a ski injury

Diagnosis

Initial diagnostic imaging includes a standard AP radiograph of the bilateral sternoclavicular joints. In addition to the AP view, an oblique view of the SC joint (serendipity view) may help in diagnosis of SC joint injuries (Fig. 12.19) [50, 51, 53]. This view is obtained by directing the X-ray beam 40° cephalad while the patient is supine centered at the SC joint. It is helpful to radiograph both SC joints to allow for comparison of any subtle abnormalities seen with such injuries. However, since plain radiographs can miss many SC joint

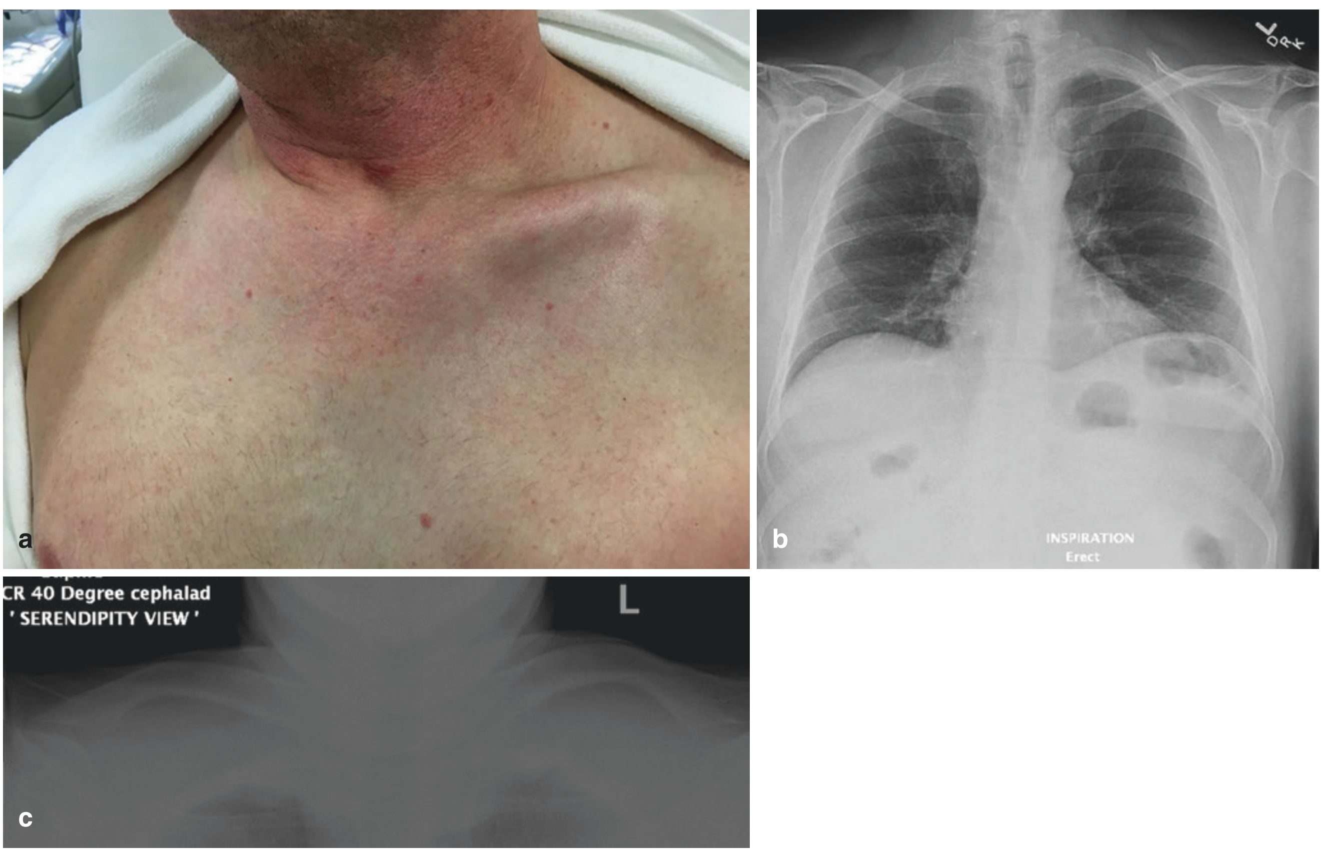


Fig. 12.19 Protruded left clavicle (anterior SC joint injury Type II) in a 54-year-old male as a result of a ski injury (**a**). Chest X-ray (**b**) was unremarkable; however, mild asymmetry is visible on serendipity view (**c**)

and medial clavicle injuries, along with assessing injuries to the sensitive mediastinal structures, most recommend obtaining a CT scan particularly in cases suspecting to have a posterior subluxation and dislocation [50, 51, 53]. Intravenous contrast is recommended if there is suspicion for vascular injury. The scan should include both SC joints again for comparison.

Initial Management

Type I and Type II anterior and posterior SC joint injuries can be treated non-operatively, given there are no concerns for other underlying injuries [50]. Athletes can be treated in a sling or figure-of-8 brace for comfort, along with the use of ice and analgesics for pain. Type III anterior injuries are also treated non-operatively [50, 51, 53]. An attempt to closely reduce the anterior dislocation can be performed by skilled sports medicine physicians [50]. For a Type III anterior SC joint injury, closed reduction is performed first by placing the athlete supine with a roll between the shoulder blades. Gentle pressure is then applied to the anteromedial clavicle until reduction has been achieved. Unfortunately, anterior dislocations tend to be unstable after reduction and will dislocate once pressure is released [50, 51, 53]. If reduction is maintained, then the athlete should be placed in a sling or a figure-of-8 brace for 4–6 weeks to promote healing of the surrounding ligaments and capsule. Due to a potential risk of damaging vital organs, athletes with suspected Type III posterior injuries should be immediately referred a skilled health care facility with access to CT angiogram and orthopedic surgery [50, 51, 53]. No reduction attempts should be allowed on the field. Closed reduction of a Type III posterior dislocation is performed in the operating room with a cardiothoracic surgeon available should there be complications from vascular injury. While under general anesthesia, the athlete is placed supine with a roll in between both shoulder blades. Traction is then placed on an abducted arm while slowly extending it. If this does not reduce the dislocation, a towel clamp can be used to grasp the medial clavicle and pull it anteriorly. Once reduced, in comparison to anterior dislocations, the joint is typically stable. The athlete is then placed in a sling or figure-of-8 brace to wear for 4–6 weeks.

Indications for Orthopedic Referral

All Type III posterior SC joint injuries require urgent transport and referral to a skilled health care facility for thorough evaluation. This is due to the high risk of injuries to the posterior mediastinal structures. Type III anterior SC joint injuries can be managed by primary care or orthopedic sports medicine physicians. With regard to operative treatment of anterior and posterior Type III SC joint injuries, most authors recommend closed treatment, as there have been a number of complications associated with open reduction and stabilization procedures [50, 51, 53, 56]. Thus, the risks outweigh the benefits.

Follow-Up Care

With Type I and Type II SC joint injuries treated non-operatively, follow-up can be 2–4 weeks after injury to ensure clinical healing, strength, and range of motion of the affected shoulder. For Type III dislocations that require closed reduction, follow-up after 4–6 weeks of immobilization is recommended to begin a rehabilitation program [50].

Return to Sports

There is no evidence to suggest when an athlete can return to sport from a Type I or II SC joint injury [50]. Most athletes can return to sport safely when their pain is well controlled, have normal range of motion, and protective strength of the affected shoulder [50]. Formal physical therapy may help accelerate return to play in athletes after an SC joint injury. Athletes with a Type II SC joint injury may need 4–6 weeks off from sport to allow adequate healing of the capsule and surrounding ligaments to prevent long-term instability and symptoms. Athletes with a Type III SC joint injury may need a minimum of 4–6 weeks of rest and immobilization prior to starting rehabilitation [50, 57].

Complications

Type I and II SC joint injuries are well tolerated and typically have no long-term complications. Painful instability and cosmetic deformities of the joint may occur with Type II SC joint injuries [50]. Most complications occur with Type III posterior SC joint injuries. Injuries to the mediastinal structures, including vascular injury, can occur. Injuries to the trachea, brachial plexus, and esophagus are uncommon but can occur [50, 51, 53]. Complications of surgical management include loss of reduction, postoperative infection, and posttraumatic arthritis [50–53, 55, 56]. Migration of hardware into heart and surrounding blood vessels has been reported in the literature [58].

Pediatric Considerations

Since the medial clavicular epiphysis does not ossify until 20 years of age and rarely fuses before 25, most injuries to the medial clavicle and SC joint are physeal fractures in chil-

dren and adolescents. These fractures tend to heal remarkably well non-operatively due to their high remodeling potential. However, true dislocations, whether anterior or posterior, can occur in this age group, and the standard reduction techniques are used [59]. Open reduction and internal fixation is rarely performed in physeal fractures, except for an irreducible posteriorly displaced fracture with symptoms of mediastinal compression. After ORIF, a figure-of-8 brace is used for 4 weeks [59].

Acromioclavicular (AC) Joint Injuries

AC joint injuries (also known as "separation" and "dislocation") are common among athletes, particularly in contact sports.

Mechanism of Injury in Sports

AC joint injuries can be caused by direct or indirect forces. The most common cause is a fall or direct impact to the anterior, superior edge of the acromion with the arm in an adducted position [60–62]. As a result, the acromion is driven downward and medially, and a cascade of failure of the stabilizing structures ensues. The first structures to fail are the AC ligaments, followed by the coracoclavicular (CC) ligaments (conoid preceding trapezoid), and with more severe trauma the fascial and muscular attachments of the deltoid and trapezius to the clavicle can be compromised [60–62]. It is no surprise that sports where there is a high frequency of contact also have the highest frequency of AC joint injuries [60, 62]. In fact, in the contact athlete, it is the most common shoulder injury. Sports that put players at the highest risk for this injury pattern are football, ice hockey, rugby, and wrestling [63, 64]. Direct player-to-player contact accounted for 72% of AC joint injuries versus 27% of injuries sustained during contact with the ground in a study of NCAA men's football players [65].

Epidemiology

Especially in the young and athletic, AC joint injuries are one of the most common causes for shoulder pain. They account for 8% of all joint dislocations, with an overall incidence of 9 per 1000 person-years in younger athletes as demonstrated in a recent longitudinal cohort study performed by the United States Military Academy [64]. Numerous studies have shown that the highest risk sports for this injury pattern are those in which contact commonly occurs, which includes rugby, wrestling, hockey, judo, lacrosse, and football [64, 66–69]. In a recent review of the NFL Injury Surveillance System, 29% of shoulder injuries were due to AC joint injuries, with quarterbacks followed by special teams players being the most prone to injury [70]. These injuries most frequently occur in the third decade of life, and males have a reported five times greater risk of injury than females [64]. Level of play and practice versus competition also factor into AC injury. More injuries are sustained in intercollegiate compared to intramural athletes [64]. Players sustain injuries more frequently in-season and in competitive play compared to practice as shown by a review of the NCAA Injury Surveillance System of men's football in 2004–2009 [65].

Sprain Classification

The most widely accepted classification system has been developed by Rockwood and colleagues (Fig. 12.20) [71]. This is a modification of the original work by Tossy et al. [72]. This classification scheme (Fig. 12.20) is based upon the extent of damage and thus the degree of injury to the AC, CC ligaments, and deltotrapezial musculature by physical examination and imaging [62, 73–75]. Type I injuries are sprains or partial tears of the AC ligament without evidence of radiographic or clinical instability. Type II injuries are the most common AC joint injury diagnosed in young athletes [64]. This injury type results from complete tear of the AC ligaments with sprain, but not complete disruption of the CC ligaments. While vertical stability is maintained, horizontal instability occurs and can be visualized as widening of the AC joint space on radiographs. Together Type I and II injuries make up 96% of AC joint injuries in the NCAA men's football population [65]. Type III injuries are due to complete disruption of the AC and CC ligaments, resulting in both horizontal and vertical instability. Radiographically they are defined as a CC distance increase between 25% and 100% compared to that of the contralateral side. A posterior dislocation of the distal clavicle through the trapezial fascia characterizes Type IV injuries. An axillary radiograph will demonstrate the posterior displacement of the clavicle with respect to the acromion. Type V injuries are defined as greater than 100% increase in the CC space relative to the contralateral side. In addition to the AC and CC ligaments, the deltotrapezial fascia is detached. Type VI injuries are rare, the largest published series involving only three individuals [76]. The clavicle in this type is either lodged beneath the coracoid and conjoint (short head of biceps and the coracobrachialis) tendon or in a subacromial location.

Clinical Presentation

When first evaluating a patient for a potential AC joint injury, information gathering begins with getting a thorough history

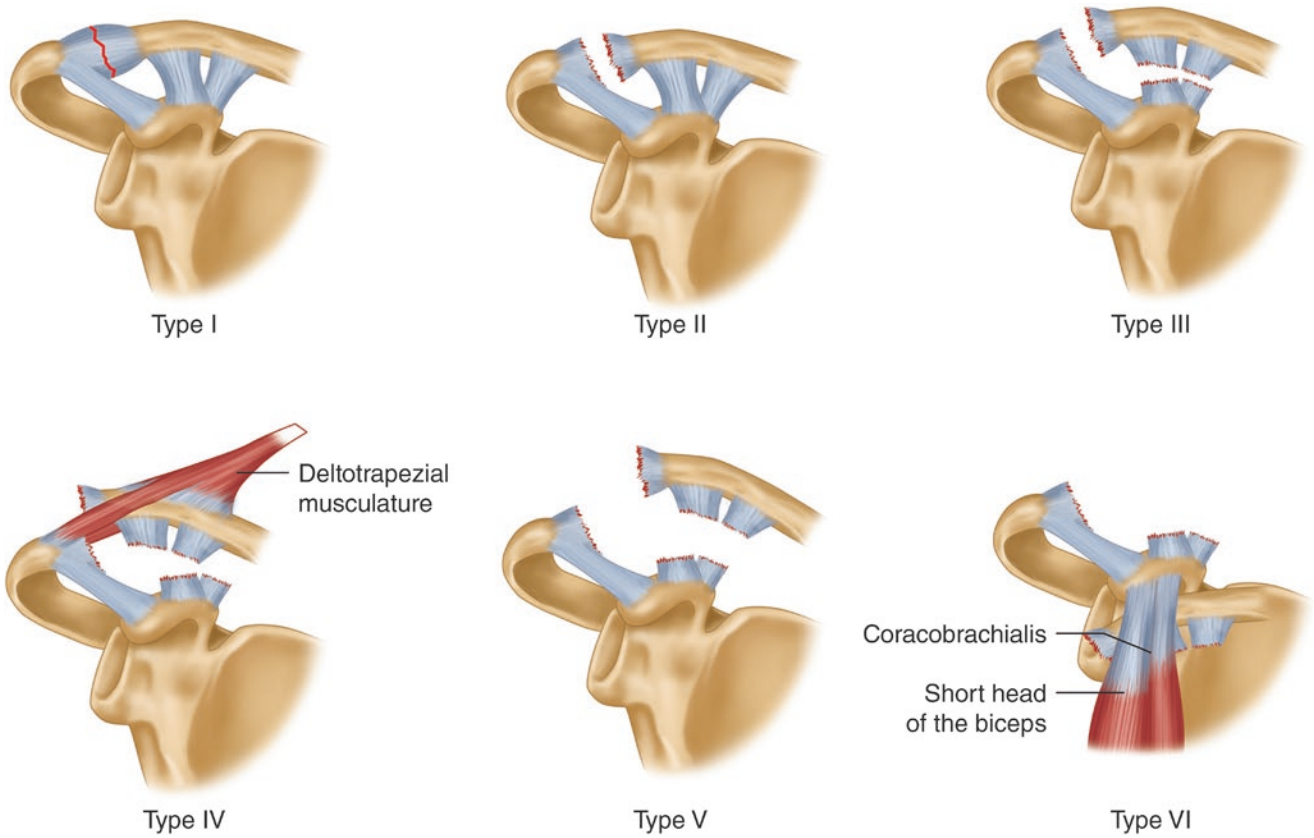


Fig. 12.20 Rockwood classification of the AC joint injury (X). Type I sprain of the AC ligament (joint capsule); Type II AC joint is vertically displaced (<25%), AC ligament is ruptured, and CC ligament is intact or sprained; Type III AC joint is vertically dislocated (25–100%), AC and CC ligaments are ruptured, and deltotrapezial musculature is typically intact; Type IV AC joint is posteriorly dislocated through trapezius muscle, AC and CC ligaments are ruptured, and deltotrapezial musculature is detached; Type V AC joint is dislocated (100–300%) with rupture of deltotrapezial facia, AC and CC ligaments are ruptured, and deltotrapezial musculature is detached; Type VI AC joint is dislocated with clavicle positioned in subacromial or subcoracoid, AC and CC ligaments are ruptured, and deltotrapezial musculature is detached

of the event leading to the athlete's shoulder pain and symptoms afterward [60, 62, 73, 74]. Once a careful history has been obtained, it is important to do a thorough upper extremity examination, including a cervical, glenohumeral (GH), and neurovascular examination of the upper extremities in addition to assessment of the AC joint and clavicle. Along with asymmetry (Fig. 12.21), tenderness at the AC and potentially CC ligaments, and increased mobility or instability of the joint, pain may be produced with taking the patient's arm into a flexed and adducted position. Asymmetry at the AC joint, skin abrasion, swelling, and ecchymosis over the distal clavicle may be seen on inspection [60, 62, 74]. It is important to ask the athletes to relax and drop their arms to accurately evaluate the step deformity and asymmetry (Figs. 12.21 and 12.22). In Type I injuries, athletes may have tenderness to palpation over the AC joint, but this is absent over the CC ligaments. However, in Type II and higher, tenderness over the CC area may also be present. In Type III injuries, dislocations are often displaced superiorly but are reducible on examination. The athlete should be instructed to shrug their shoulders [74]. The deformity in Type III AC joint injury typically is reduced by shrugging as the deltotrapezial fascia is intact [74]. If the shrugging did not reduce the deformity, most likely this is a Type V injury [74]. The distal clavicle can be ballotable and reducible by pressing (inferior direction) the medial aspect of the clavicle. Tenting of the skin in the posterior superior aspect of the shoulder is rare and should raise suspicion for a Type IV injury that has punctured through the trapezius muscle or possibility of an associated fracture [60, 62, 74]. Type V injuries may clinically present with a significant drooping deformity of the shoulder and the clavicle is non-reducible as it has pierced through the fascia [60, 62, 74]. Provocative tests such as the cross-arm adduction and active compression (e.g., O'Brien) tests usually aggravate the AC joint pain [60, 62, 74, 77].

Diagnosis

AP and axillary plain radiographs are the initial imaging recommended for confirming the diagnosis of an AC joint injury [60, 62, 74, 78]. An AP view will identify the amount of verti-

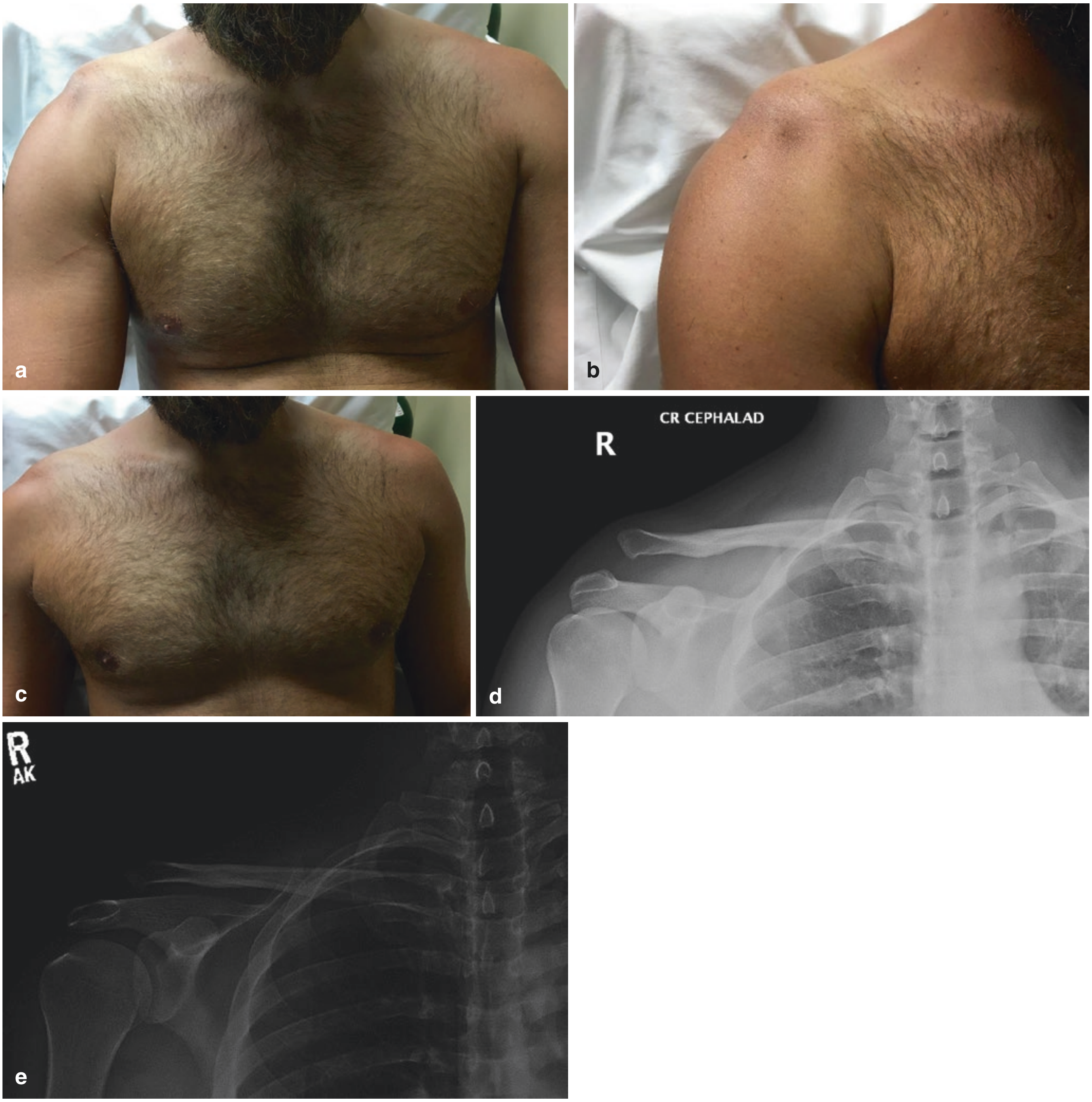


Fig. 12.21 Right Type V AC joint injury in a 32-year-old male as a result of a snowboarding injury (**a**, **b**). The deformity diminished by shrugging the shoulder (**c**). Plain radiography confirmed the diagnosis (**d**, **e**)

cal displacement, and the axillary view is key in demonstrating displacement in the horizontal plane (Figs. 12.23, 12.24, 12.25, 12.26, and 12.27). The axillary view is particularly key in demonstrating the posterior displacement of the clavicle in Type IV injuries (Fig. 12.28). The addition of a Zanca view (AP with 10°–15° of cephalic beam tilt) (Figs. 12.29, 12.30, and 12.31) further optimizes evaluation as it improves visualization of the AC joint by removing the superimposition of the acromion on the distal clavicle and takes the scapula out of the field of view [60, 62, 74]. A wide plate should ideally be utilized for the AP and Zanca views in order to visualize the contralateral shoulder for direct comparison on a single cassette [60, 62, 74, 75, 78]. This is important as up to 50% subluxation of the distal clavicle can be seen in normal shoulders [75, 78, 79]. The normal AC width is between 1 and 3 mm and decreases with increasing age [62]. The measurement of the CC distance is best achieved by comparing with the contralateral side to determine displacement [62, 73–75].

Fig. 12.22 Left Type V AC joint injury in a 40-year-old male as a result of a mountain bike injury (**a**). The deformity diminished by shrugging the shoulder (**b**). Plain radiography confirmed the diagnosis (**c**)

Athletes should have both arms relax on the side of the body without any shrugging as this may understate the degree of the injury [60, 62, 73, 74]. Stress views of the AC joint, which involve the patient holding weight in each arm while radiographs are taken may provide additional information, but it is rarely performed due to patient discomfort [60, 62, 73–75]. While not standard of care at this point, there is growing popularity in obtaining MRI and ultrasound as aids in diagnosing injuries of the AC joint [60, 62, 73, 74]. MRI has approximately 50% concordance with radiographic AC joint injury classification, allows for a more complete evaluation of the extent of soft-tissue injury, and will depict any concomitant injuries such as SLAP tears, which in high-grade type IV AC joint injuries may occur in up to 15% of cases [60, 62, 73, 74, 80]. Ultrasound can be helpful when radiographs are equivocal as a dynamic evaluation can occur, whereby abnormal micromotion can be detected [81].

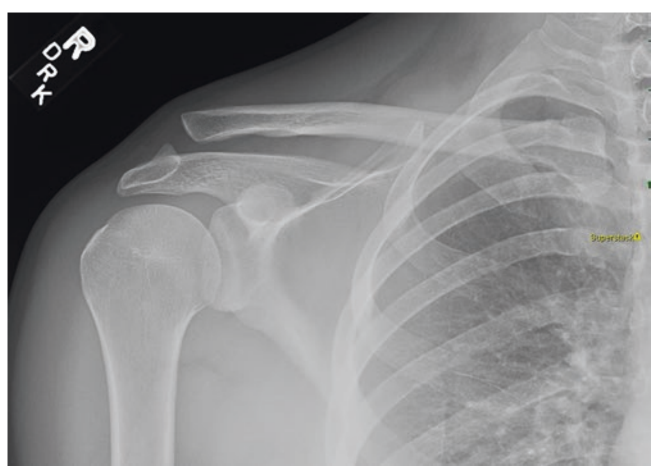


Fig. 12.23 A right Type II AC joint injury in a 25-year-old male as a result of a fall

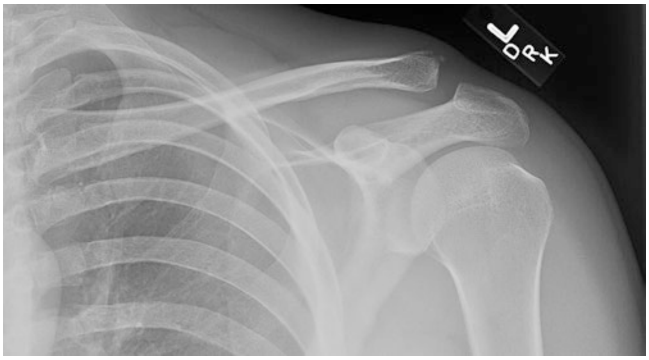


Fig. 12.24 A left Type II AC joint injury with an intra-articular avulsion fracture of the distal clavicle in a 25-year-old male as a result of a snowboarding injury

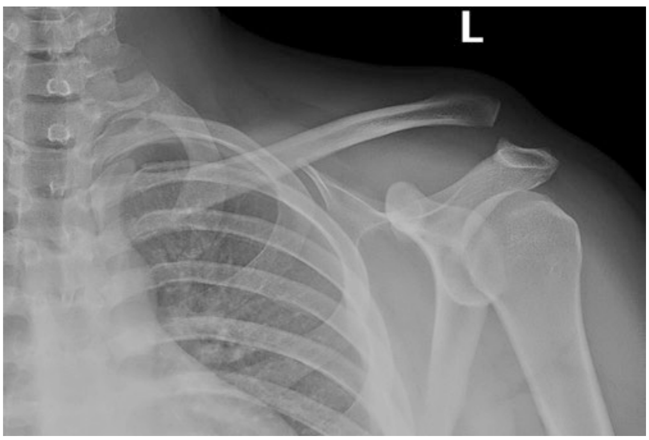


Fig. 12.25 A left Type III AC joint injury in a 23-year-old male as a result of a fall

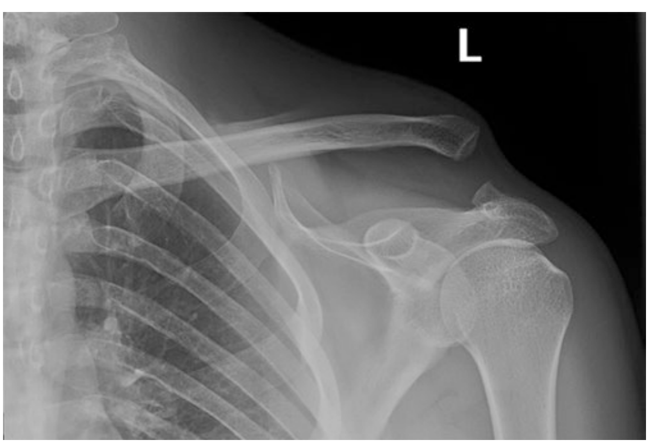


Fig. 12.26 A left Type V AC joint injury in a 26-year-old male as a result of a mountain bike fall

Fig. 12.27 A right Type V AC joint injury in a 40-year-old male as a result of a snowboarding injury

Initial Management

When an AC joint injury is suspected on the sidelines, cervical and glenohumeral pathology should also be considered and ruled out. Physical examination should be performed by the team physician. If the player's pain remains focal to the area around the AC joint, there is no skin tenting, and the joints (AC and GH) are stable on palpation in the vertical and horizontal plains, then the player can be placed in a sling to take forces off of the AC joint. In cases of suspected neurovascular compromise or fractures, the athlete should be transferred to an urgent care for further evaluation and imaging. If an AC injury continues to be suspected, then confirmation with radiographic imaging as described above can be obtained. The patient with an AC injury should be placed into sling with the recommendations of icing and NSAIDs for pain control.

Indications for Orthopedic Referral

Urgent orthopedic consultation is indicated in patients with skin tenting, open fracture, or neurovascular compromise. All athletes with Type IV, V, or VI AC injuries should be referred to orthopedic surgery [60, 73, 74, 82, 83]. While acute Type I and Type II injuries do well with non-operative management focusing on pain control, sling immobilization, early shoulder range of motion, and physical therapy, there remains a lot of controversy as to the best management of Type III injuries [83]. All athletes with Type III AC joint injuries should be referred to sports medicine (primary care or orthopedics) to allow an opportunity to evaluate and discuss the best treatment plan for the individual athletes [62, 73, 74, 84–86]. There is limited evidence, based on high-quality RCTs, to suggest that patients with Type III AC joint injury do better with surgical intervention compared with non-operative management [73, 74, 84]. Some clinicians advocate for early surgical intervention in those with high functional demands such as overhead athletes and laborers; however, multiple studies have demonstrated equivalent strength in the AC injured versus non-injured arm and over-

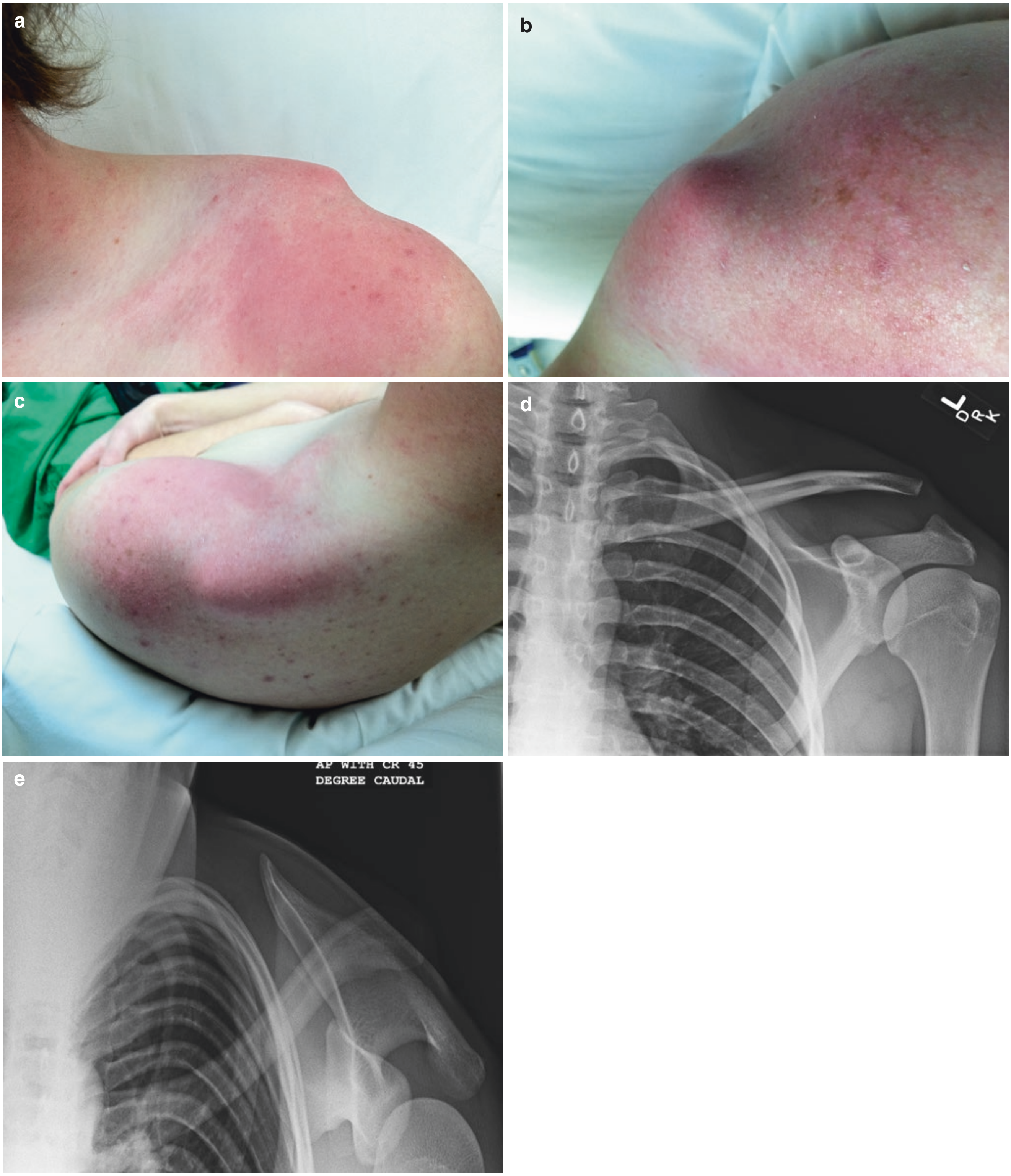


Fig. 12.28 A Type IV AC joint injury in an 18-year-old male as a result of a fall while snowboarding (**a**–**c**). AP (**d**) and AP with 45° caudal (**e**) plain radiography confirmed the diagnosis

all good-to-excellent results in those that are treated non-operatively [60, 62, 73, 74, 83–88]. Sport played, level of play, functional demands, and in-season versus out-of-season injury all need to be considered in determining the best treatment modality for a Type III injury in athletes. For those with a Type I, II, or conservatively treated Type III injuries that

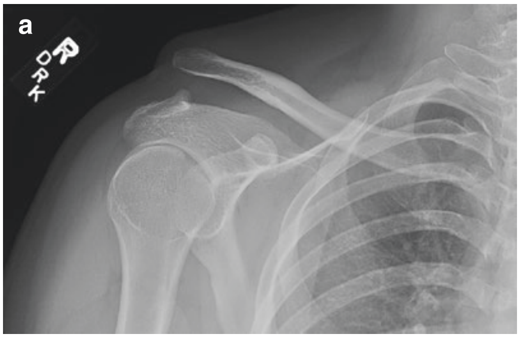


Fig. 12.29 A right Type IV AC joint injury in a 27-year-old male as a result of a mountain bike fall (**a**, **b**)

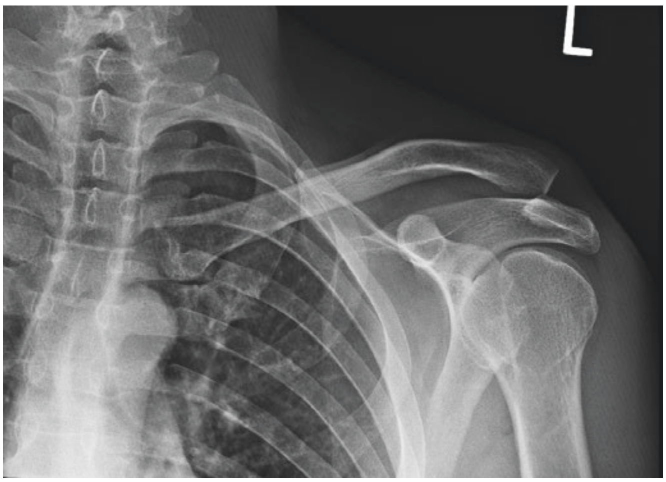


Fig. 12.30 A left Type IV AC joint injury in a 37-year-old male as a result of a mountain bike injury (**a**, **b**)

fail non-operative management and continue to have persisting pain, scapular dysfunction, or weakness, referral to an orthopedic surgeon should be made. Studies have shown that even those with type III injuries surgically treated after failing non-operative management can expect the same outcome as those successfully treated non-operatively [89, 90].

Follow-Up Care

Athletes with Type I, Type II, and initially non-operatively treated Type III injuries should stop using a sling when they no longer have pain with the arm at the side and during self-care activities [91]. Enrollment into physical therapy should be initiated immediately to promote mobility and range of motion [73, 74, 91]. Gradual return to unrestricted activity usually occurs within 2 weeks after Type I and II injuries. A follow-up visit with treating physician should thus occur at about 2 weeks from the athlete's injury for clearance to return to play.

For managing Type III injuries non-operatively, a graduated rehabilitation program is encouraged [92]. Physical therapy protocol can be divided into four progressive phases: (1) pain control with immediate protected range of motion and isometric exercises to prevent atrophy, (2) isotonic strengthening exercises and proprioceptive control, (3) unrestricted functional participation with focus on increasing strength and endurance, and (4) return to activity with sports-specific drills [91, 92]. Evaluation by a clinician should occur in this injury pattern every couple of weeks to ensure that progressive improvement is made [73, 74, 91, 92]. If no improvement or functional decline occurs after a 6–12 week trial of rehabilitation, revisiting surgical intervention is recommended.

Postoperative follow-ups are typically arranged by treating orthopedic surgeon. Usually, sutures are removed and radiographic images are taken 7–14 days after surgery. A period of immobilization in a sling and initiation into a physical therapy protocol is usually begun immediately. The amount of time in the sling is dependent upon the surgical technique used and based upon the surgeon's preference [73, 74, 91, 92]. As there are over 60 different described techniques for surgical stabilization of AC joint injuries, no standard postoperative protocol has been established. At least 6 weeks in a sling is often described in expert opinion with immediate initiation of pendulum exercises [73, 74, 91, 92]. Restoration of range of

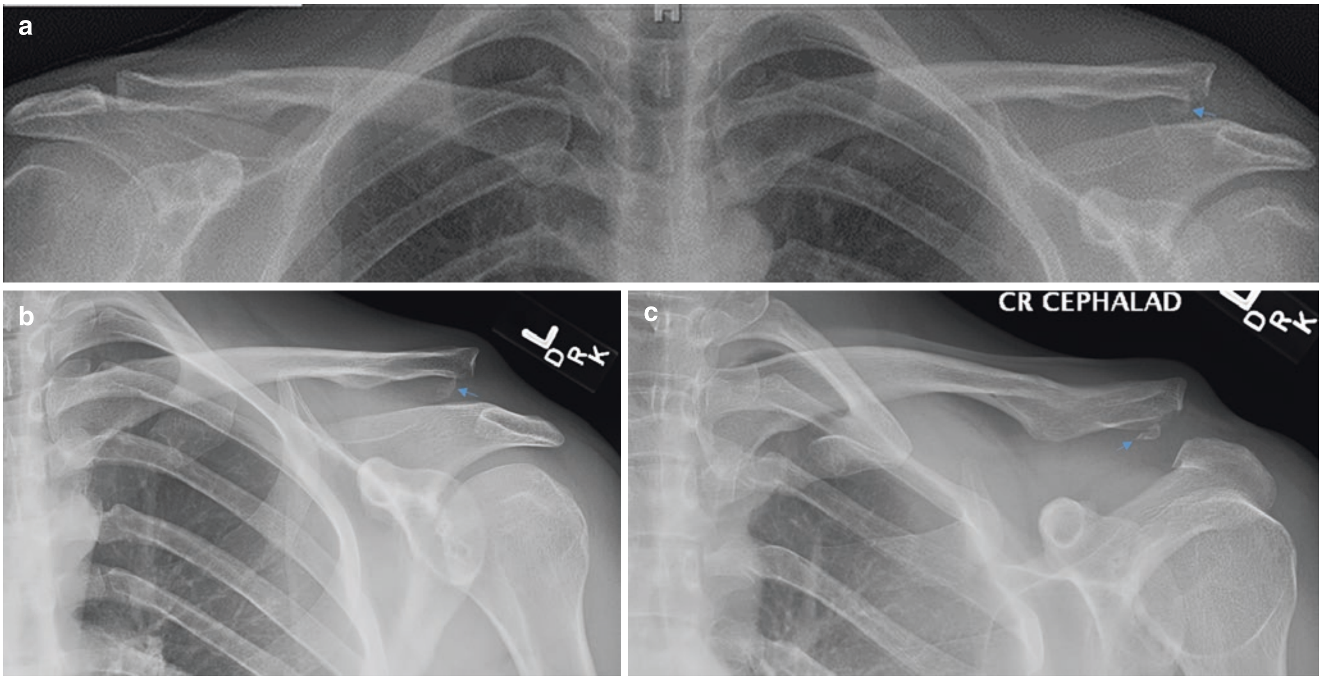


Fig. 12.31 A Type III AC joint injury and Type III distal left clavicle fracture (arrows) in a 32-year-old male as a result of a mountain bike injury (**a**–**c**)

Table 12.2 Management guidelines for AC joint injuries [60, 62, 73, 74, 82–86, 88, 91, 93–95]

AC joint injury category	Grade I	Grade II	Grade III	Grade IV	Grade V	Grade VI
Length of immobilization (sling)	1 week	1–2 weeks	2–4 weeks	2–6 weeks	2–6 weeks	2–6 weeks
Indications for operative management discussion orthopedics	No[a]	No[a]	Yes[a]	Yes	Yes	Yes
Follow-up	In 2–3 weeks	In 2–4 weeks	Every 2–3 weeks	Every 2–4 weeks	Every 2–4 weeks	Every 2–4 weeks
Healing time	2–3 weeks	2–6 weeks	4–12 weeks	8–12 weeks	8–12 weeks	8–12 weeks
Return to sports	1–2 weeks	2–4 weeks	1–3 months	1–5 months	1–5 months	1–5 months

[a]Referral to a primary care sports medicine physician is acceptable

motion and scapular exercises are gradually worked into the athlete's program followed by more progressive strengthening starting around 3 months postoperatively [73, 74, 91]. The frequency of physician follow-up, radiographic imaging is again surgeon dependent but usually tapers as the athlete progresses through the rehabilitation protocol. Table 12.2 summarizes the guidelines for management of different types of AC joint injuries.

Return to Sports

Epidemiological studies of AC injuries in collegiate athletes and NFL football players have shown on average time loss of about 11 days in low Type I and II AC joint injuries [64, 65]. In the collegiate population average time lost for Type III or higher injuries was 64 days compared to 32 days in professional football players [64, 65]. This data demonstrates quick functional recovery for lesser AC joint injuries. Undoubtedly the demands and importance of a quick return-to-play are higher in the professional athlete compared to a high school, collegiate, or recreational athlete, which is likely why the return to play in the NFL population appears to be half of that in the young athletic population [64, 65]. This importantly depicts that in higher type AC injuries, rehabilitation and return to play are very much dependent on the sport played, level of play, and functional demands of the athlete along with whether and what type of surgical intervention was carried out [60, 64, 65, 73, 74]. An athlete is deemed safe to return to sport once achieving full range of motion, minimal pain or tenderness, satisfactory clinical examination, and demonstration of adequate strength on testing. Generally speaking, most expert opinion indicates return to contact sport can begin at 5–6 months, though progression in strength and endurance can be expected up to 12 months [91, 96].

Fig. 12.32 A right AC joint deformity in an otherwise asymptomatic 58-year-old male as a result of a remote Type V AC joint injury (**a**, **b**)

Complications

In general, rate and severity of complications are higher with operative compared to non-operative management [74]. Complications following a non-operative management include early-onset degenerative joint disease, AC joint instability, chronic pain, cosmetic deformity (Fig. 12.32), and AC joint osteolysis [62, 73, 74]. These complications typically do not cause any clinical and functional deficits [62, 74]. For Type I, II, III, and IV injuries, the failure of non-operative management (persisting pain, scapular dysfunction, and weakness despite an appropriate period of rehabilitation) can result in orthopedic referral for potential surgical intervention [94]. Asymmetry or cosmetic dissatisfaction should not be an indication for surgery as persisting deformity in surgically treated groups ranges between 15% and 80% in clinical series [60, 73, 74, 82, 93]. This is especially true as loss of reduction postoperatively does not correlate with patient symptoms. The overall complication rate after surgery can be as high as 15% [93]. Postoperative complications are infection, neurovascular injury, adhesive capsulitis, implant failure, suture granuloma, hardware irritations, and rarely migration [60, 73, 74, 82, 93]. Complications after surgical intervention are often dependent on the surgical intervention performed. The severity of complications in those treated operatively is variable ranging from loss of reduction without symptoms, to clavicle fracture about a clavicular bone tunnel or near plate fixation, the planned or symptom-mediated removal of a plate if one is used, and infection either superficial or deep [60, 62, 73, 74, 82, 84, 93, 95, 97].

Pediatric Considerations

Injuries to the AC joint in children and adolescents are commonly physeal fractures of the distal clavicle or occult fractures due to the strong ligamentous attachments [44, 98]. The distal clavicular physis ossifies around 18 years of age and closes shortly after ossification. High-grade AC joint injuries in children are very uncommon [98]. Many AC joint injuries, including distal clavicle fractures in children and adolescents, can be treated non-operatively with excellent results. As in adults, Type I to III AC joint injuries can be treated non-operatively (Fig. 12.33). In children with Type IV to VI AC joint injuries, a referral to a pediatric orthopedic surgeon is recommended [98].

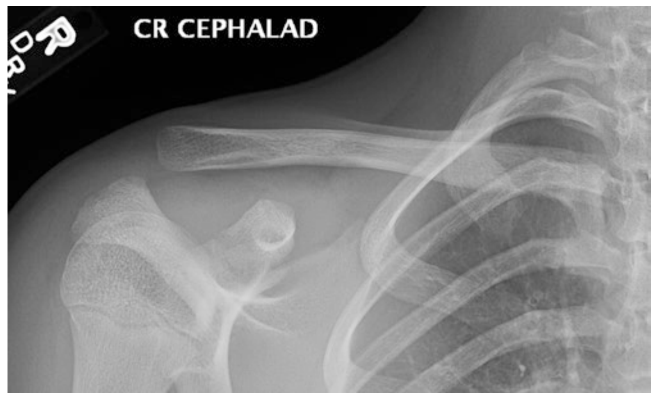


Fig. 12.33 A right Type III AC joint injury in a 14-year-old male as a result of a mountain bike injury

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Scapula

13

Stephen M. Spadafore, Michelle Wolcott, and Darcy Selenke

Key Points

- Because of the high force impact needed, scapular fractures are often associated with other, possibly more serious injuries.
- Scapular fractures are most often treated conservatively and generally have good outcomes.
- Indications for surgery are varied and currently evolving.

Introduction

Scapular fractures represent less than 1% of all fractures and 3–5% of fractures of the shoulder girdle [1]. Its incidence in sports is unknown, but it seems to be more common in high-speed sports such as skiing, snowboarding, cycling, and motor sports [2]. Because scapulae are well protected by the surrounding soft tissue structures and have a relatively high degree of mobility, they usually require high-energy forces to fracture. They occur with concomitant injuries in 80–95% of cases, most commonly to the thorax [3, 4]. Scapular fractures are associated with a 10–15% mortality, usually due to concomitant injuries to the head or chest [4]. Traditionally, scapular fractures have been managed non-operatively with generally good outcomes. However, there is increasing interest in defining clear surgical indications [5].

Anatomy

The scapula is a flat, triangular-shaped bone located on the superior aspect of the posterior thorax. Relevant bony landmarks include the body, neck, spine, glenoid fossa, acromion process, coracoid process, medial border, lateral border, and superior border (Fig. 13.1). The scapula has 17 muscle attachments and 3 joints. It articulates with the humerus at the glenohumeral joint, with the clavicle at the acromioclavicular joint, and with the thorax at the scapulothoracic joint. Normal movement of the upper extremity requires coordination of these many components [6].

Scapular Fracture Classification

There is no consensus system used to classify scapula fractures. Historically, systems classified scapula fractures by what was seen most commonly in clinical practice [5]. More recently, the Arbeitsgemeinschaft für Osteosynthesefragen (AO) Foundation and Orthopaedic Trauma Association (OTA) has developed a comprehensive classification system with the goal to facilitate patient outcome-centered research and clarify indications for surgical treatment [1]. This system divides fractures into three main areas: the body, the glenoid fossa or articular segment, and the processes [1]. This chapter will adhere to this classification system [1, 7, 8].

S. M. Spadafore
Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA
e-mail: stephen.spadafore@cuanschutz.edu

M. Wolcott
Department of Orthopedics, CU Sports Medicine, University of Colorado School of Medicine, Denver, CO, USA
e-mail: michelle.wolcott@cuanschutz.edu

D. Selenke (✉)
Department of Family Medicine, University of Colorado School of Medicine, Denver Health, Winter Park Medical Center, Winter Park, CO, USA
e-mail: Darcy.Selenke@dhha.org

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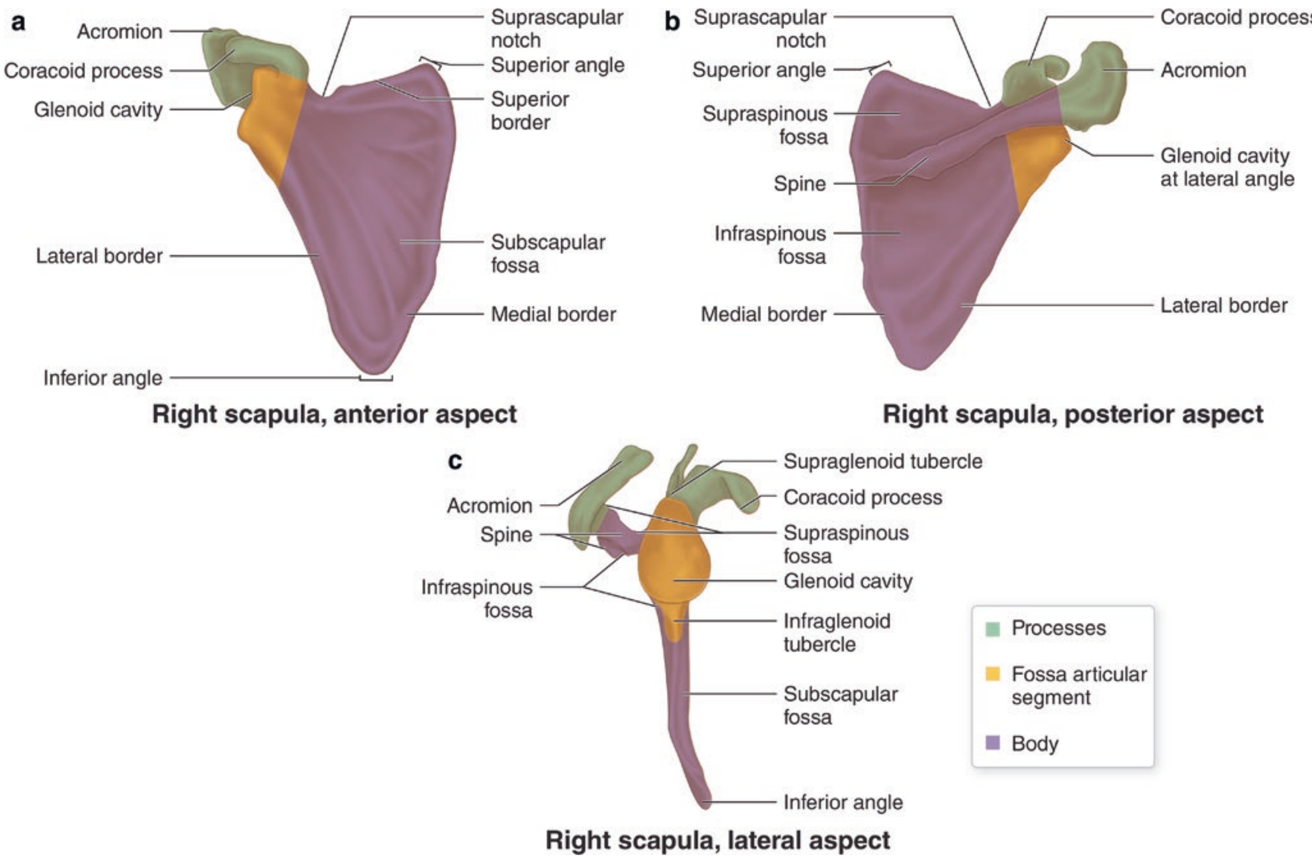

Fig. 13.1 Scapular anatomy. (**a**) Right scapula, anterior aspect. (**b**) Right scapula, posterior aspect. (**c**) Right scapula, lateral aspect

Extra-articular Scapular Body Fractures

Mechanism of Injury in Sports

Scapular body fractures typically result from direct impact high-energy trauma. Common mechanisms include fall from a height or high-speed collision. The impact is usually directly on the upper back [6].

Epidemiology

Scapular body fractures are the most common fracture site, comprising 45–73% of scapular fractures [5, 9].

Classification

The AO/ATO classification system uses the letter "B" to denote scapular body fractures [8]. It then divides scapular body fractures into two types. Fractures that exit the body at two or fewer points are type 1, called B1 [8]. Fractures that exit the body at three or more points are type 2, called B2. They are further qualified by their exit points; lateral border, medial border, superior border, or area lateral to the base of the coracoid [7, 8].

Clinical Presentation

Patients typically present with arm held adducted, localized tenderness to palpation, and resistance of the arm to movement. There may be posterior shoulder flattening if the spine is involved [4].

Diagnosis

Diagnosis is made radiographically (Figs. 13.2 and 13.3). Initial imaging should include X-ray of the scapula in AP, axial view, Grashey view, and lateral views. CT scan can be helpful in further clarifying the extent of the fracture [6].

Initial Management

Because of the high impact required for scapular body fractures, initial management often includes evaluation and treatment of more serious associated injuries (e.g., pneumothorax, pulmonary contusions, cranial lesions, spinal lesions, major vascular injuries). A thorough neurovascular examination of the affected side should also be performed as brachial plexus injuries are often seen with scapular body fractures

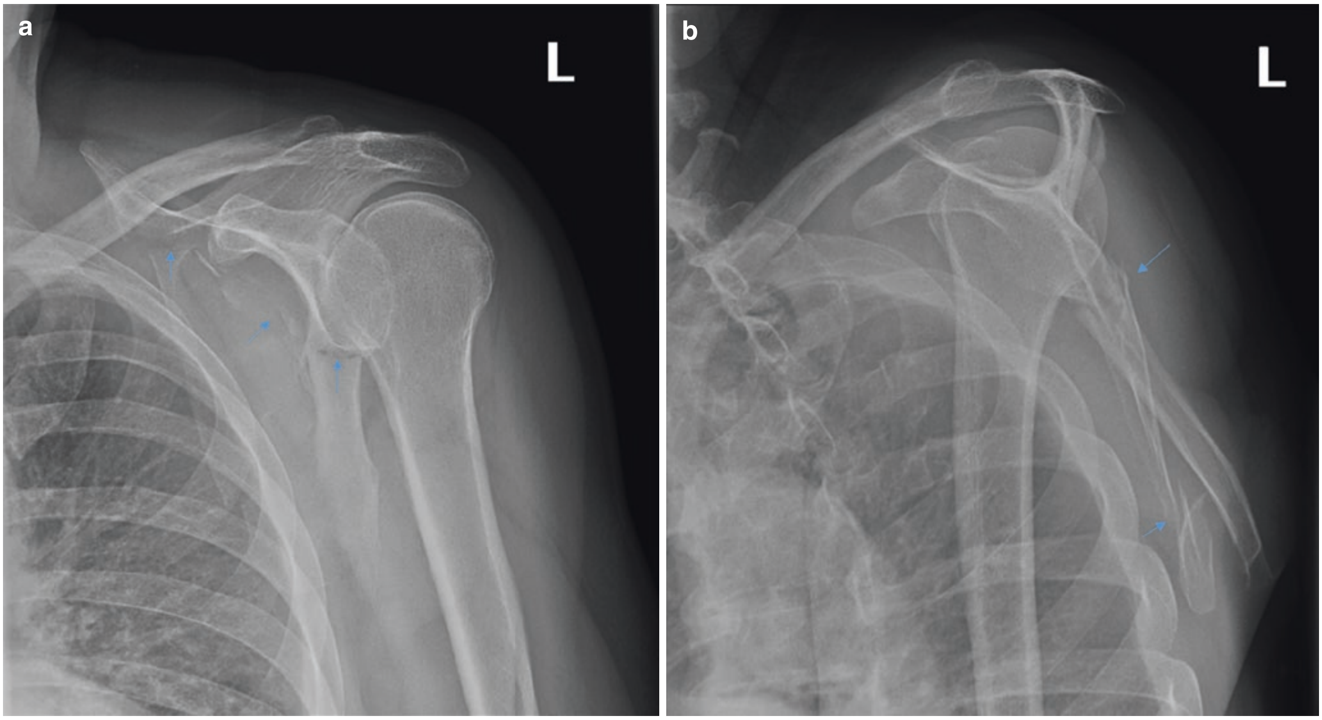

Fig. 13.2 Comminuted left scapular body fracture (arrows) in a 68-year-old male as a result of a fall while skiing (**a**, **b**)

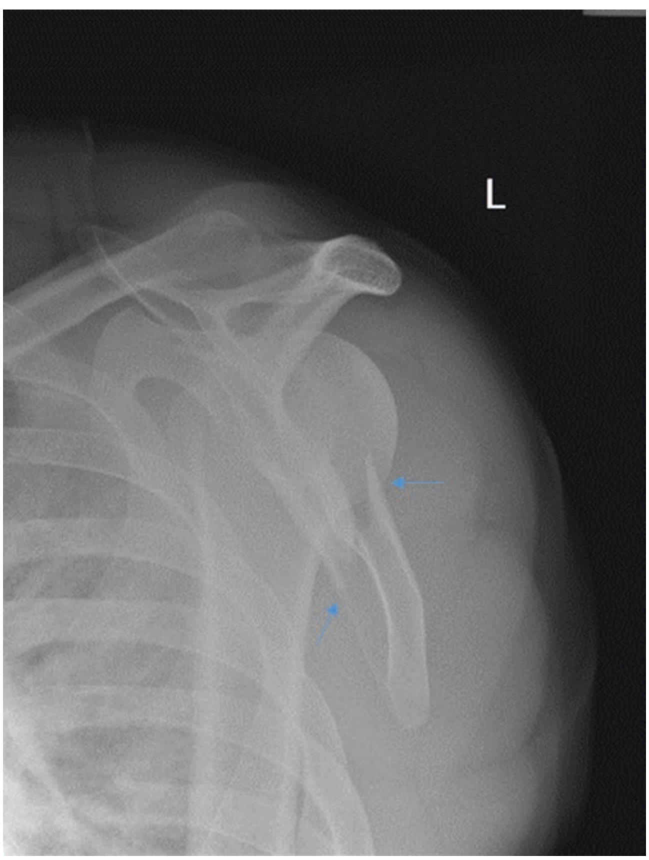

Fig. 13.3 Comminuted and posteriorly displaced left scapular body fracture through the infraspinous (arrows) in a 40-year-old male

[4]. Once otherwise stabilized, initial management includes supporting the arm in a sling for comfort with ice and analgesics for pain control [9].

Indications for Orthopedic Referral

The vast majority of scapular body fractures are treated nonoperatively with overall good outcomes [4, 5, 10]. Multiple authors have suggested guidelines for surgery based on the degree of displacement, though there is a lack of high-quality evidence that would point to consensus recommendations. With regard to the scapular body fractures, displacement is measured in lateral border offset, angulation, and glenopolar angle (GPA). Lateral border offset is measured in the AP or Grashey view from the most lateral aspect of the inferior fragment to the most lateral aspect of the superior fragment. Lateral border offset of 10–20 mm is an indication for orthopedic referral. Angulation is measured in the lateral view as the angle between the inferior fragment and the superior fragment. Angulation of 40–45° is an indication for orthopedic referral. The GPA is measured in the AP or Grashey view as the angle between a line that runs from the most cranial point of the glenoid cavity to the most caudal point of the glenoid cavity and a line that runs from the most cranial point of the glenoid cavity to the most caudal point of the scapular body. A normal GPA is 30–45°. A GPA of 20–22° or less is an indication for orthopedic referral [6].

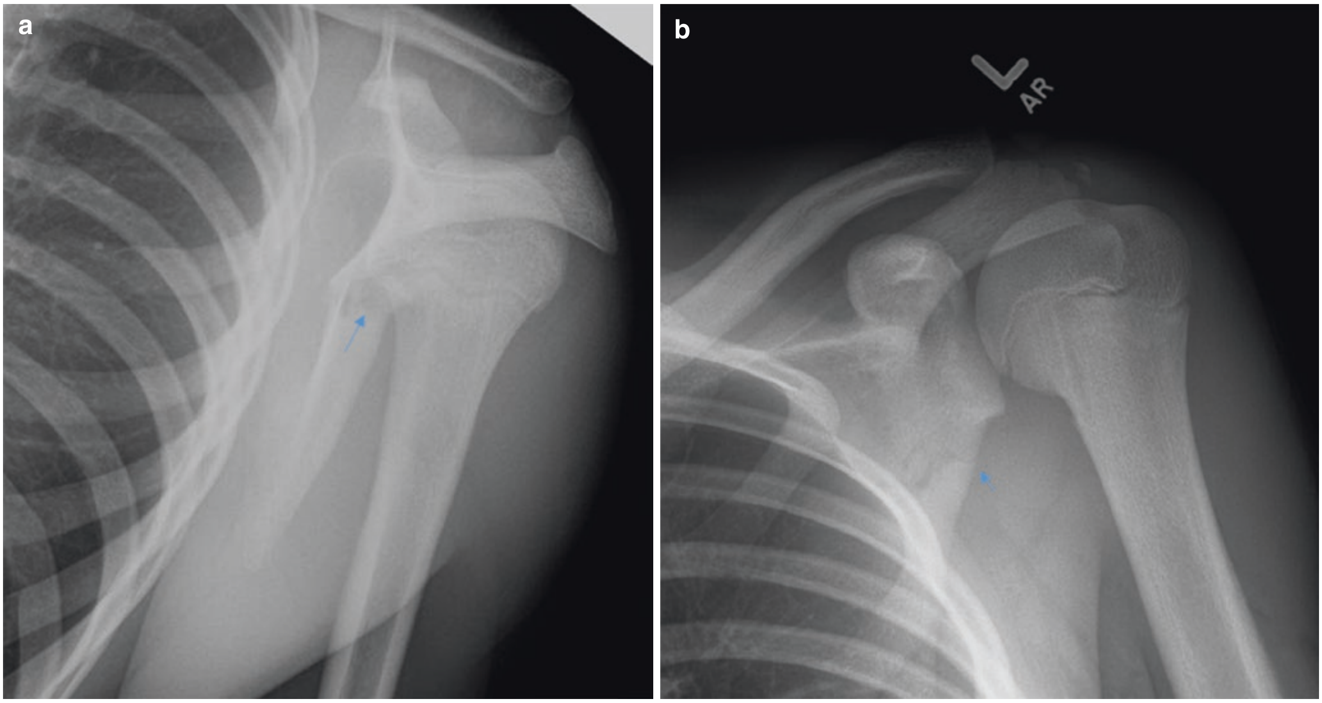

Fig. 13.4 Left scapular body fracture (arrows) in a 14-year-old boy as a result of a football injury (**a**, **b**)

Follow-Up Care

Slings are typically worn for 2–3 weeks. Gentle range of motion exercises are introduced as pain allows, often within the first week. Regular (once or twice) X-rays should be performed for the first 3 weeks to demonstrate presence of healing and absence of progressive displacement. Active range of motion exercises are then started, usually around 4 weeks. At 8 weeks, progressive shoulder strengthening exercises are added [10]. Physical therapy should be continued to maximize range of motion, strength, and functional use of the arm. A full recovery may be expected at 6 months to 1 year [11].

Return to Sports

Non-contact participation may occur when patient is pain free with activity, usually at 6–8 weeks [11]. There is a lack of consensus when patients should return to contact sports or sports that involve heavy use of the shoulder.

Complications

In general, nonunion or malunion is rare, especially in minimally displaced scapular body fractures [4, 10]. Patients often report pseudoparalysis and lack of control of the arm during healing. Progressive deformity of the fractured scapula has also been seen, especially in more unstable fractures [10].

Pediatric Consideration

There are no significant differences in the management of pediatric scapular fractures (Fig. 13.4) and adults' scapular fractures.

Intra-articular Glenoid Fossa Fractures

Mechanism of Injury in Sports

Glenoid fossa fractures most often occur as a result of the humeral head being driven into the fossa by a force applied to the lateral aspect of the shoulder [4]. Glenohumeral dislocation is associated with glenoid rim (e.g., Bankart) fractures, as well [6].

Epidemiology

Glenoid fossa fractures comprise approximately 10% of all scapular fractures [1].

Classification

Glenoid rim and fossa fractures had traditionally been classified according to the Ideberg classification system, which described five commonly seen fracture patterns [4]. The AO/ATO classification system denotes fossa fractures with the letter "F" and divides them into three types [8]. The first type, called F0, is a fracture that runs through the glenoid neck resulting in detachment of the glenoid fossa from the scapular body. It does not involve the articular surface of the glenoid [1]. This type of fracture was previously known as a scapular neck fracture [8]. The second type, called F1, are simple rim, transverse, or oblique fractures through the articular surface with a single fracture line resulting in two articular fragments. The third type, called F2, are multifragmentary fractures through the articular surface with multiple fracture lines and three or more articular fragments. The F1 and F2 fractures are further divided by their location in the four quadrants of the glenoid fossa [1].

Clinical Presentation

As with other types of scapular fractures, patients typically present with arm held adducted, localized tenderness to palpation, and resistance of the arm to movement [4].

Diagnosis

Diagnosis is made radiographically (Fig. 13.5). Initial X-rays should include the four views discussed above.

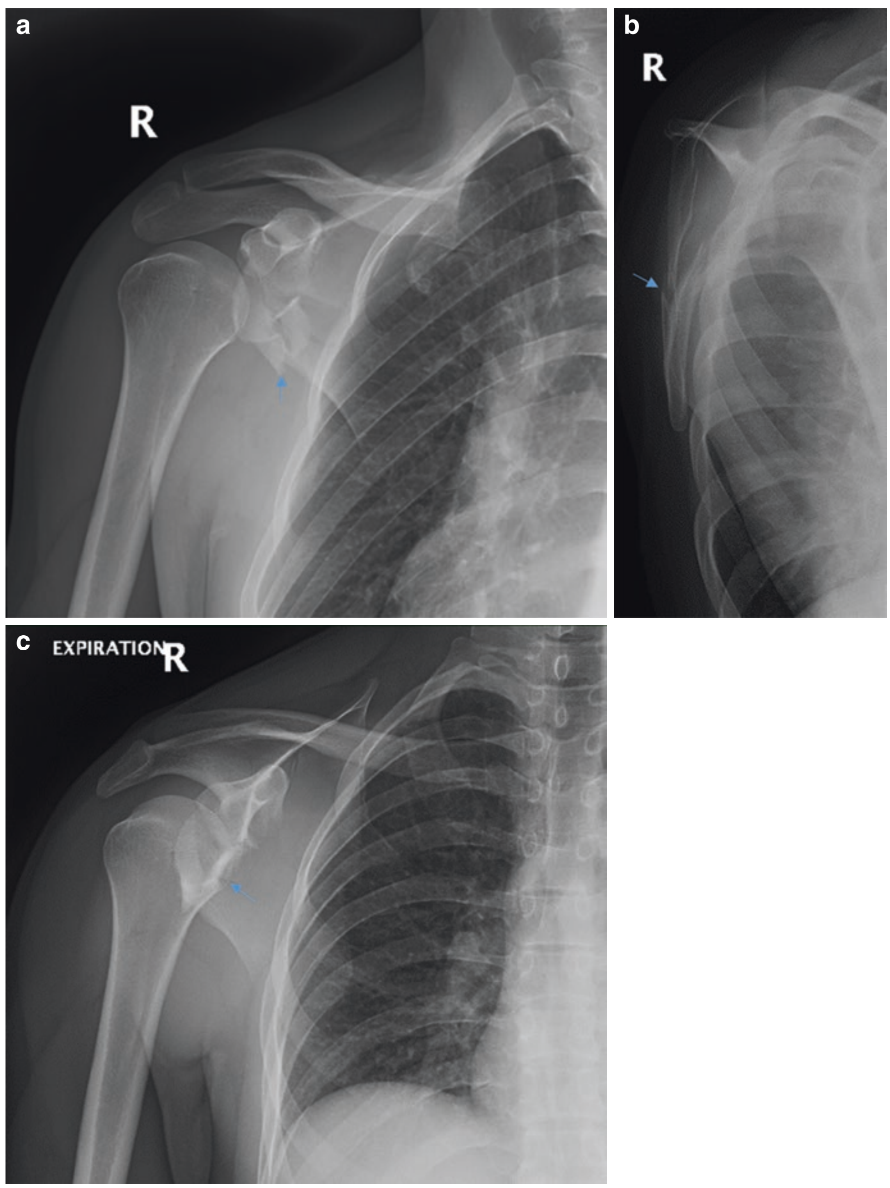

Fig. 13.5 Comminuted right scapular neck fracture (arrows) extended into the glenoid and scapular body in a 33-year-old male (**a**–**c**)

Grashey and axillary views are best for detecting intra-articular fractures. There should be a lower threshold to obtain CT scan if there is concern for intra-articular fracture, particularly if there is incongruity of the glenohumeral joint or obvious bony step-off [6].

Initial Management

As with other scapular fractures, initial management requires evaluation and treatment of more serious concurrent injuries. After that, the arm should be supported in a sling for comfort with ice and analgesics for pain control [9].

Indications for Orthopedic Referral

In contrast to the predominantly non-surgical management of scapular body fractures, 80% of glenoid fossa fractures warrant consideration for surgical management. Because of the function of the glenoid fossa, inadequate reduction of a fracture can result in joint instability and risk for subsequent arthritis [1]. As with many aspects of managing scapula fractures, there are no consensus indications for surgical repair of glenoid fossa fractures [10]. Generally, nondisplaced fractures can be managed non-operatively, and displaced or unstable fractures should undergo ORIF. Much of the literature cites >4 mm displacement of the articular surface as the threshold for surgical repair, though some advocate as little as 2 mm displacement [4, 6]. Other indications for surgical consideration include 25% involvement of the anterior rim, 33% involvement of the posterior rim, fractures associated with glenohumeral instability or recurrent subluxation of the humeral head, open fractures, and fractures extending to the medial border [6, 11].

Follow-Up Care

This is similar to the extra-articular scapular body fractures for non-operative management cases.

Return to Sports

Non-contact participation may occur when patient is pain free with activity, usually at 6–8 weeks [11]. There is a lack of consensus when patients should return to contact sports or sports that involve heavy use of the shoulder.

Complications

Articular surface fractures with persistent joint incongruity are at greater risk for osteoarthritis, instability, chronic pain, and decreased range of motion [6].

Pediatric Considerations

There are no significant pediatric considerations in treatment of scapular fractures.

Scapular Process Fractures

Mechanism

In addition to direct blunt trauma to the shoulder, indirect trauma from anterior glenohumeral dislocation can cause fractures of the coracoid and acromion processes. Avulsion fractures by attached muscles can also occur [6].

Epidemiology

Coracoid process fractures comprise 2–13% of scapular fractures [6]. Acromion process fractures represent 8–16% of scapular fractures [6]. Scapular spine fractures comprise 6–11% of scapular fractures [6].

Classification

The AO/ATO classification system pays much less attention to process fractures compared to scapular body and glenoid fossa fractures. All process fractures are denoted with the letter "A". Coracoid process fractures are A1, acromion process fractures are A2, and scapular spine fractures are A3 [7].

Clinical Presentation

As with other types of scapular fractures, patients typically present with arm held adducted, localized tenderness to palpation, and resistance of the arm to movement [4].

Diagnosis

Diagnosis is made radiographically (Figs. 13.6 and 13.7). The four views discussed previously should identify most process fractures. Additional axillary or oblique views can clarify less obvious acromion and coracoid fractures [6]. If there is concern for a double disruption of the superior shoulder suspensory complex (SSSC), a weight-bearing view should also be obtained [11].

Initial Management

As with other scapular fractures, initial management requires evaluation and treatment of more serious concurrent injuries. After that, the arm should be supported in a sling for comfort with ice and analgesics for pain control [9].

Indications for Orthopedic Referral

Isolated fractures of the coracoid, acromion, or scapular spine are most often treated non-surgically. Surgical indications include fracture displacement greater than 1 cm, interarticular extension of a fragment, open fractures, and chronic irritation or painful nonunion after conservative management. Surgical repair should also be performed for athletes and manual laborers that require frequent use of the extremity [6]. In addition, orthopedic referral should be placed for double disruptions of the SSSC. The SSSC can

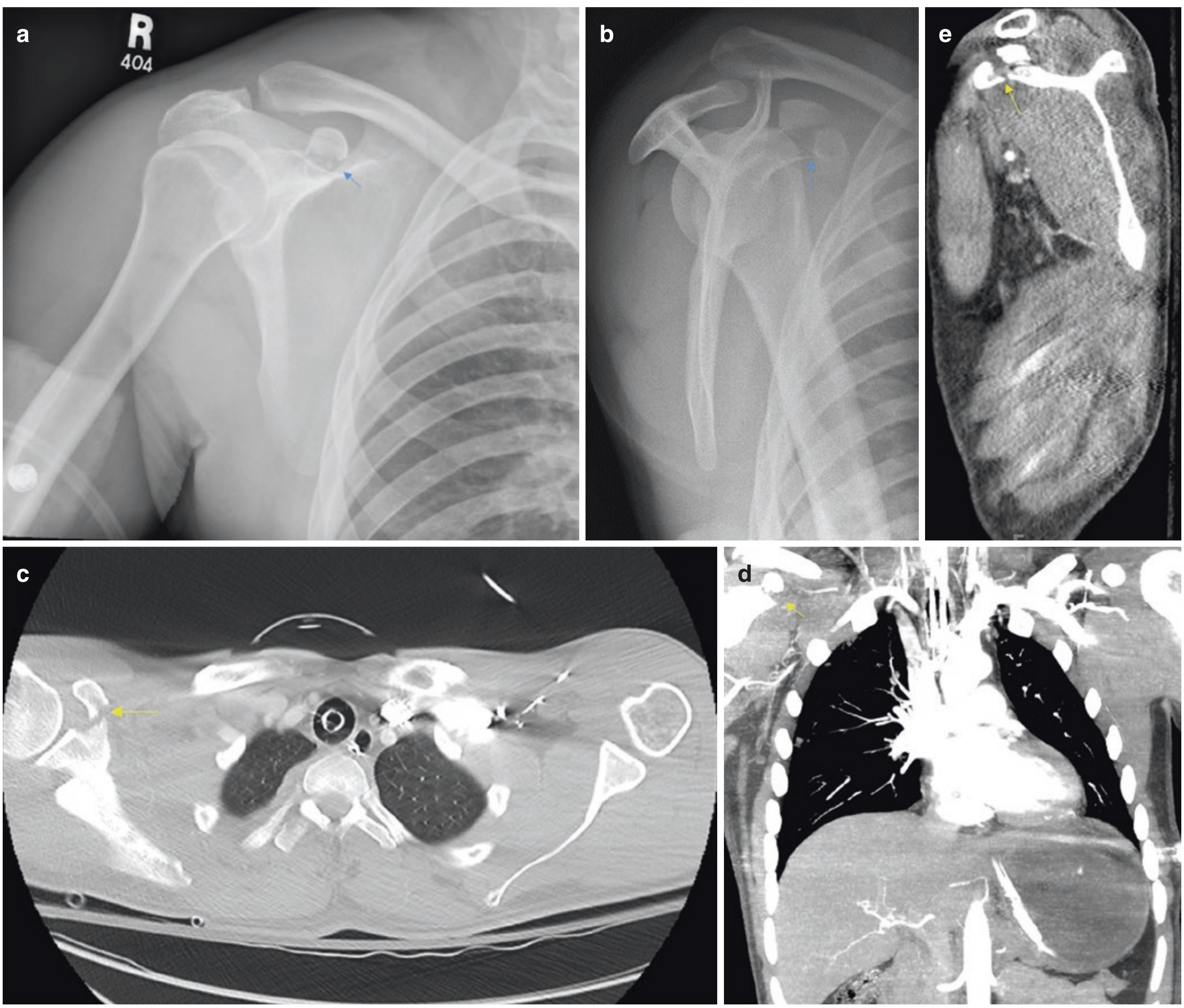

Fig. 13.6 Right coracoid process fracture (arrows) in a 36-year-old male (**a**, **b**). CT scan confirmed the diagnosis (**c**–**e**)

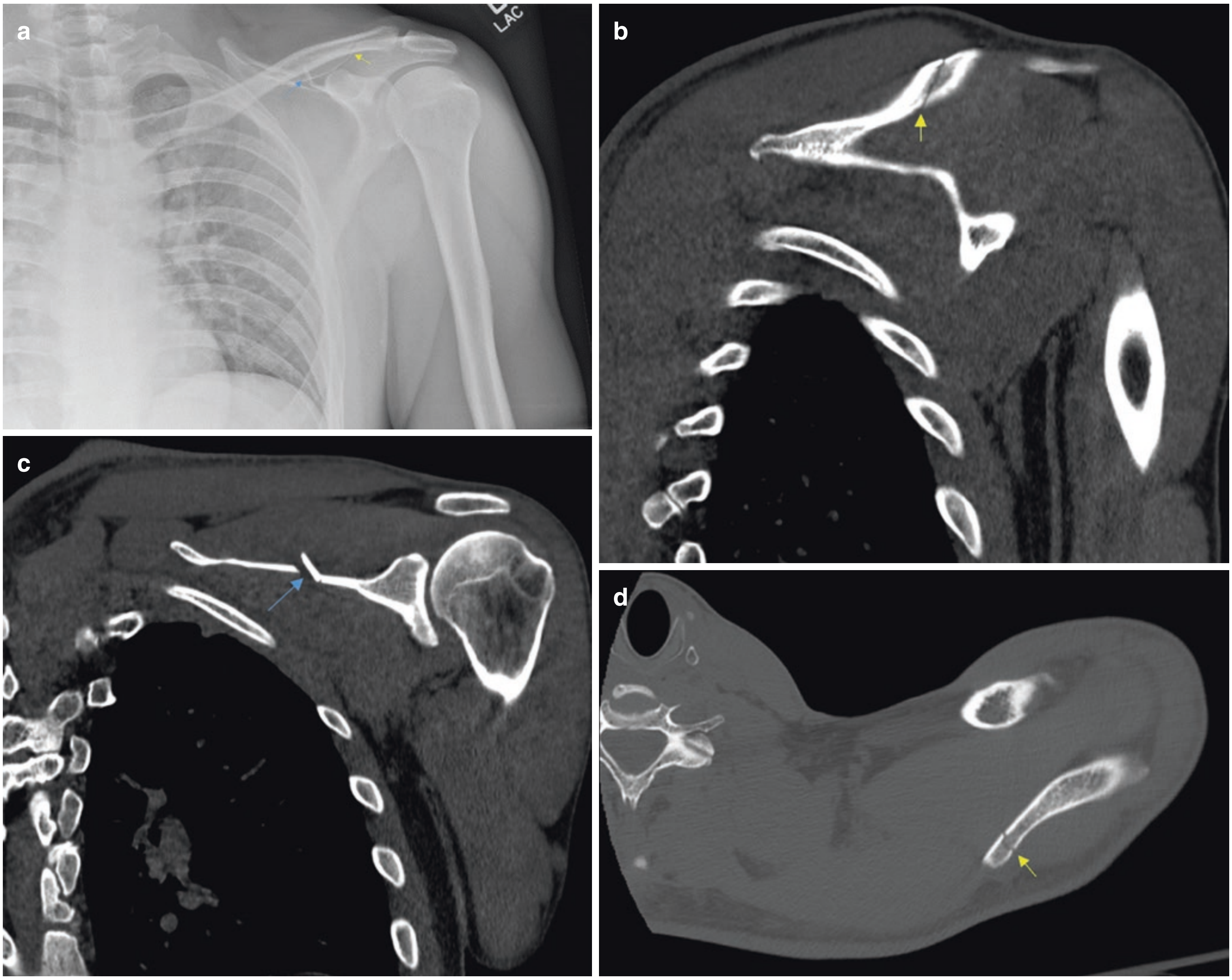

Fig. 13.7 Left superior border scapular fracture (blue arrows) and scapular spine fracture (yellow arrows) in a 43-year-old male (**a**). CT scan confirmed the diagnosis (**b**–**d**)

be conceptualized as a ring held up by two struts, which serves to suspend the arm from shoulder. The ring is comprised of the glenoid process, the coracoid process, the coracoclavicular ligament, the distal clavicle, the AC joint, the acromion process, and the coracoacromial ligament. The clavicle and the scapular body/glenoid neck are the two struts. While single disruptions of this system can be managed conservatively, double disruptions have potential for an unstable joint and adverse long-term outcomes [11]. Though there is some disagreement about the need and type of surgery that is indicated, orthopedic referral is certainly warranted [10].

Follow-Up Care

The follow-up care is similar to the extra-articular scapular body fractures for non-operative management cases.

Return to Sports

This is similar to the extra-articular scapular body fractures.

Complications

Painful nonunion, impingement syndrome, and neurologic compression have been reported in isolated coracoid and/or acromion process fractures [10]. Complications from double lesions of the SSSC include malunion, chronic pain, impingement, rotator cuff dysfunction, and osteoarthritis [6].

Pediatric Considerations

There are no significant pediatric considerations in the treatment of scapular fractures.

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14 Glenohumeral Joint

Karin VanBaak, Stephanie W. Mayer, Matthew J. Kraeutler, and Morteza Khodaee

Key Points

- The glenohumeral joint (GHJ) is the most common joint dislocation seen in emergency departments.
- Anterior GHJ dislocations are far more common than posterior or inferior dislocations.
- Male athletes participating in contact sports are at highest risk for sustaining a GHJ dislocation.
- Most GHJ dislocations are a result of contact injury.
- The main long-term risk of initial GHJ dislocation is recurrent joint instability.
- While many do well with nonoperative management following first-time (primary) shoulder dislocations, some patients require surgical management to prevent recurrent instability.

Introduction

Shoulder injuries are common in sports. Intra-articular fractures (i.e., humerus and glenoid) and soft tissue injuries such as acute tendon ruptures (e.g., long head of the biceps and rotator cuff) are discussed elsewhere in the book. In this chapter we primarily discuss glenohumeral joint (GHJ) dislocations.

Shoulder (GHJ) dislocations are the most frequent dislocation treated in the emergency department [1]. The bony articulation of the joint is the spherical humeral head onto the concave glenoid (Fig. 14.1). Static stabilizers of the joint include the glenoid labrum and glenohumeral ligaments (Fig. 14.1). The rotator cuff, long head of the biceps, deltoid, and scapular stabilizing muscles provide dynamic stability to the joint. This joint anatomy makes it the most mobile joint in the body, and also predisposes it to this type of injury [1, 2]. By definition, GHJ dislocation requires either damage to one or more of these stabilizing structures, or underlying laxity of one or more stabilizing structure. GHJ dislocations can also be classified as primary or secondary, traumatic or atraumatic, and acute or chronic. In general, GHJ dislocations can be divided into anterior, posterior, and inferior.

Anterior Dislocations

Epidemiology

Anterior dislocations are the most common type of GHJ dislocation, accounting for up to 98% of reported dislocations [3, 4]. Various studies have shown first-time anterior GHJ dislocations to occur with an incidence of 0.08–1.69 per 1000 person-years in civilian populations, and as high as 3% per year in military populations [4, 5]. These injuries occur more frequently in males than females [2, 5, 6]. Anterior GHJ dislocations are also most likely to reoccur in this demographic group [7–9]. In fact, age between 21 and 30 years is the only factor found by some authors to be associated with recurrence of dislocation [8]. An estimated 19% of all patients undergoing primary closed reduction for anterior GHJ dislocation later require repeat closed reduction, with an estimated 48%–77% of patients experiencing further dislocations not necessarily requiring closed reduction [7, 9–11].

K. VanBaak
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: karin.vanbaak@cuanschutz.edu;

S. W. Mayer
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: stephanie.mayer@cuanschutz.edu

M. J. Kraeutler
Department of Orthopedics, St. Joseph's University Medical Center, Paterson, NJ, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

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Mechanism of Injury in Sports

Anterior GHJ dislocations are most commonly a result of a force on the arm which displaces the humeral head anteriorly with respect to the glenoid. These injuries most often occur when the arm is in a position of external rotation, abduction, and extension [4, 12]. Among athletes, GHJ dislocations occur most frequently due to player-player contact [6]. Sports in which participants have been shown to have high rates of anterior GHJ instability episodes include American football, rugby, rodeo, wrestling, lacrosse, gymnastics, boxing, and ice hockey [2, 6, 12]. Overall, the rate of GHJ dislocation seems to be higher among collegiate than high school athletes [6]. In addition

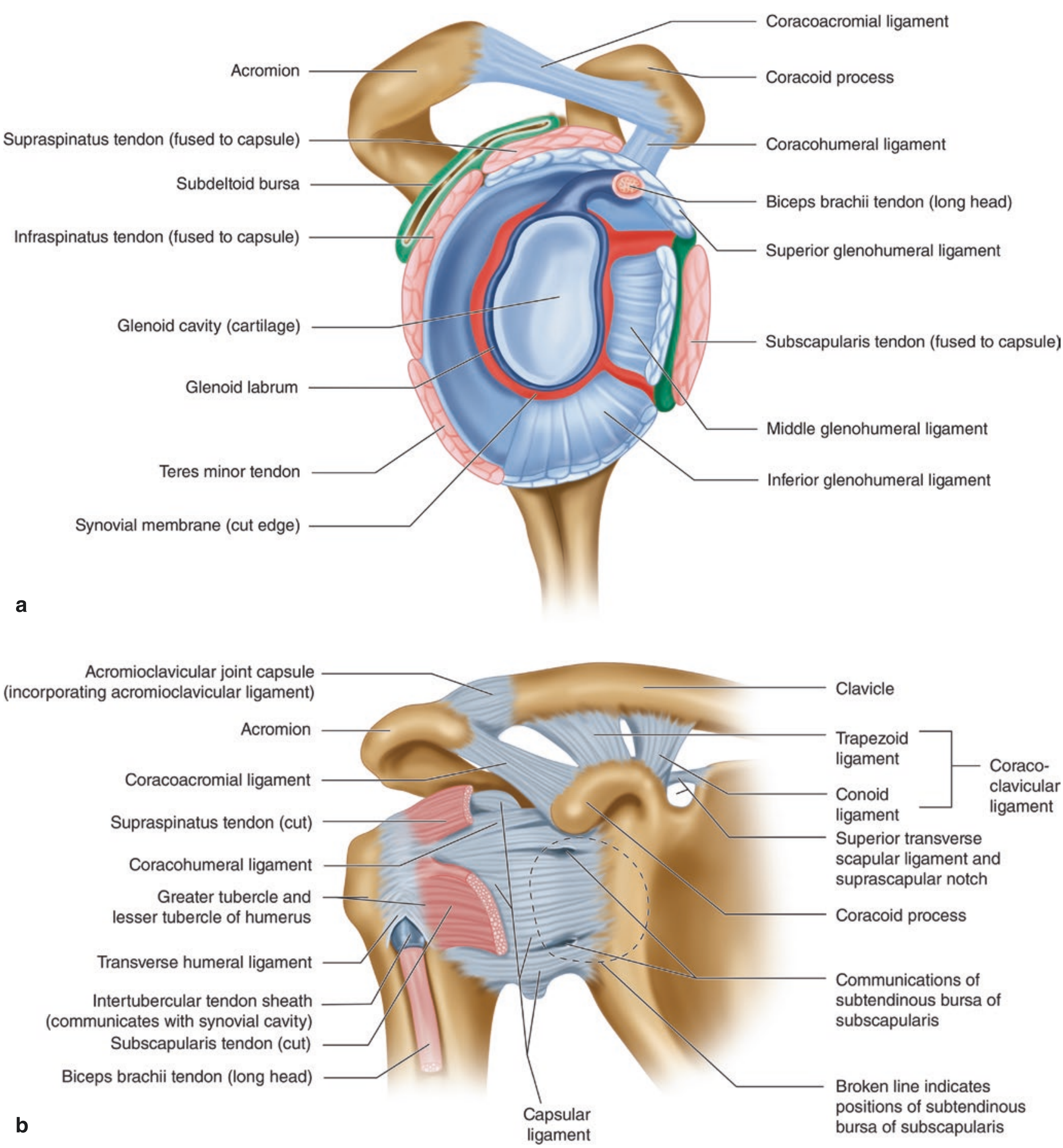

Fig. 14.1 Lateral view of the glenoid (**a**). Anterior view of the GHJ (**b**). Coronal cross-section of the GHJ (**c**)

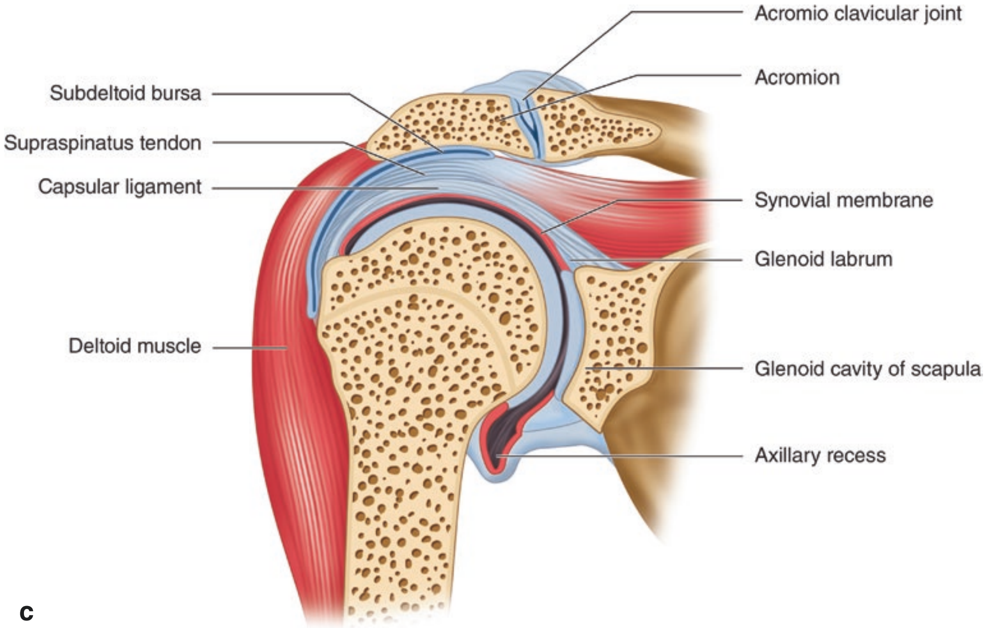

Fig. 14.1 (continued)

to these factors, male sex and various injury-specific factors increase a patient's risk of recurrent anterior shoulder instability [2, 5].

Dislocation Classification

An anterior GHJ dislocation occurs when the head of the humerus displaces anterior to the glenoid fossa. By definition, all anterior GHJ dislocations have different degrees of inferior displacement as well. There is no universal agreement on how to categorize anterior GHJ dislocations. The majority of anterior GHJ dislocations are subcoracoid in nature (Fig. 14.2), with the humeral head being displaced anterior to the glenoid and inferior to the coracoid [4]. True subglenoid (humeral head displaced anterior and inferior to the glenoid without overlap of the superior aspect of the humerus and inferior glenoid rim), subclavicular (humeral head displaced medial to coracoid), and intrathoracic anterior GHJ dislocations are very rare (Fig. 14.2) [1, 4].

Traumatic shoulder dislocations in particular usually result in damage to the normal stabilizing structures of the GHJ. The antero-inferior capsulolabral complex (anterior inferior glenohumeral ligament, anteroinferior labrum, inferior joint capsule) is the main restraint to anterior dislocation in a position of vulnerability, and therefore most often damaged with anterior GHJ dislocation [1, 2, 4, 12]. Detachment of this complex from the rim of the glenoid (Bankart lesion) has been reported in 85%–97% of shoulders after a first-time anterior GHJ dislocation [1, 2, 4]. A "bony Bankart" occurs when a bony fragment from the glenoid rim is detached along with the labral complex (Figs. 14.3 and 14.18) [1, 2, 4, 13]. Less commonly, the inferior glenohumeral ligament can avulse from its attachment to the humeral head. This is referred to as a HAGL (humeral avulsion of the glenohumeral ligament) lesion [2, 4].

A Hill-Sachs lesion (cortical depression of the posterolateral humeral head) is also commonly seen in first-time (primary) anterior GHJ dislocation (Figs. 14.4 and 14.8) [2, 4, 12]. Hill-Sachs lesions are significantly more common in patients with recurrent anterior GHJ dislocations and have been reported in 90% of collegiate athletes with an anterior GHJ dislocation [2, 14].

Rotator cuff tendon tears and greater tuberosity humeral fractures are seen more frequently with increasing patient age as a result of acute anterior GHJ dislocation (Figs. 14.5 and 14.18) [4, 15]. In rare cases, more significant fractures can be associated with GHJ dislocations (Fig. 14.6).

Clinical Presentation

Patients often report feeling the sensation of the shoulder popping or rolling out of the socket during the traumatic event. Following an acute dislocation event, a patient with an anterior GHJ dislocation will usually present with severe pain, muscle spasm, and limited voluntary motion of the shoulder. Individuals with anterior GHJ dislocations often report having the arm abducted and externally rotated during the event. After the acute injury, the arm is usually held in slight abduction (Fig. 14.7) [4]. The humeral head may be palpable anterior to the GHJ, with a defect or sulcus visible

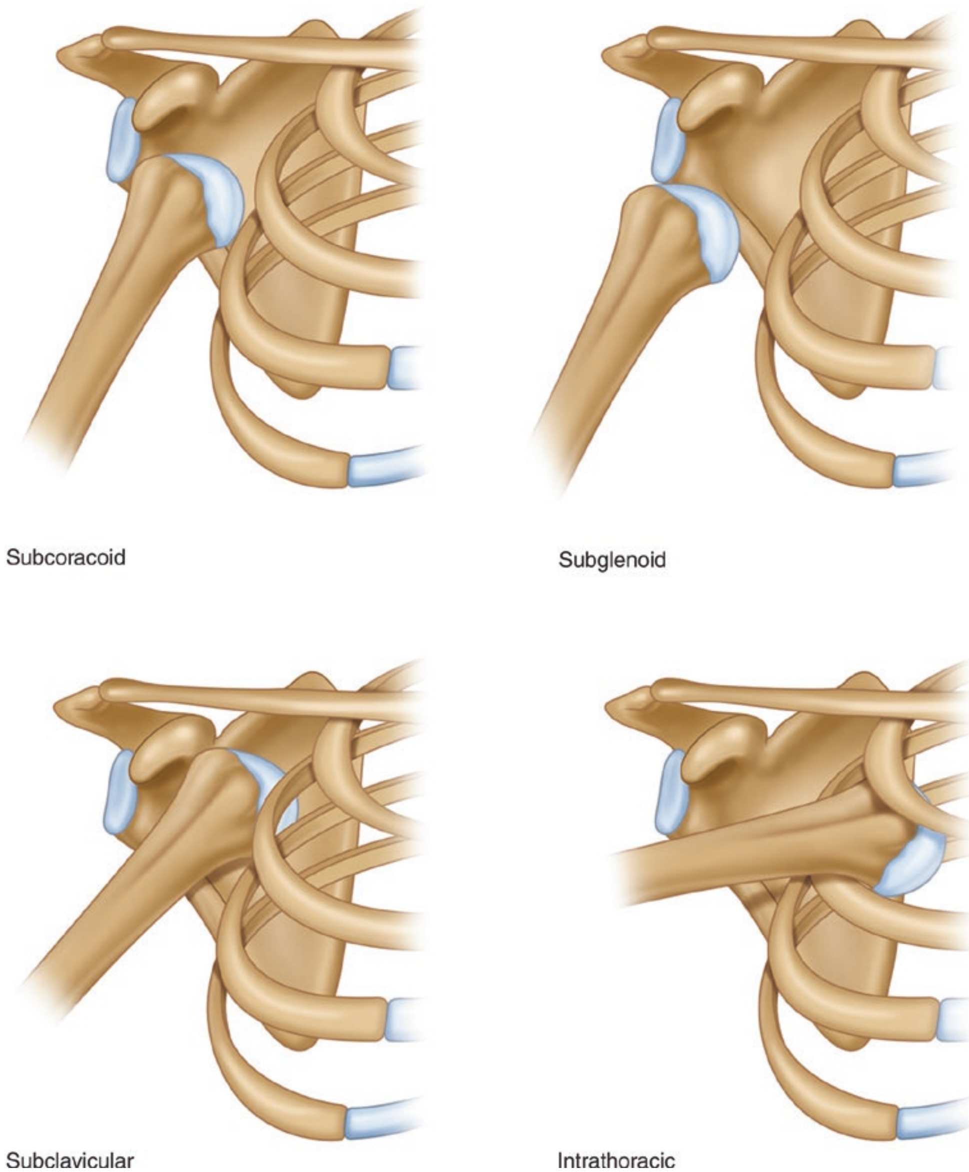

Fig. 14.2 Classification of anterior GHJ dislocations

laterally and posteriorly beneath the acromion (Figs. 14.7, 14.8, and 14.14) [4, 13].

Initial physical examination should include evaluation of neurovascular status. This includes lateral upper arm sensation and distal upper extremity vascular status [13, 16]. Any evidence of axillary artery injury (reduced capillary filling or radial pulse) should prompt further diagnostic evaluation with angiography [3].

If the patient is presenting after a dislocation which has already been reduced or after a subluxation event, evaluation may involve a more detailed physical examination. The apprehension and relocation tests are highly specific for predicting recurrent instability, though they should not be performed in an acute setting [2]. Additionally, these patients should be evaluated for underlying hyperlaxity using application of the Beighton hypermobility criteria (Chap. 9), sulcus sign, and the load and shift test, among others [2, 5].

Diagnosis

Initial diagnosis of an anterior GHJ dislocation is accomplished with a combination of history, physical examination, and plain radiographs. The preferred X-ray series includes an AP, oblique (Grashey), axillary, and scapular Y view. If it is not possible to obtain an axillary view (e.g., due to patient's discomfort), a Velpeau axillary view (Fig. 15.6; Chap. 15) may be obtained [2, 4]. Axillary and scapular Y views can determine the type of GHJ dislocation (Figs. 14.8, 14.18, and 14.21).

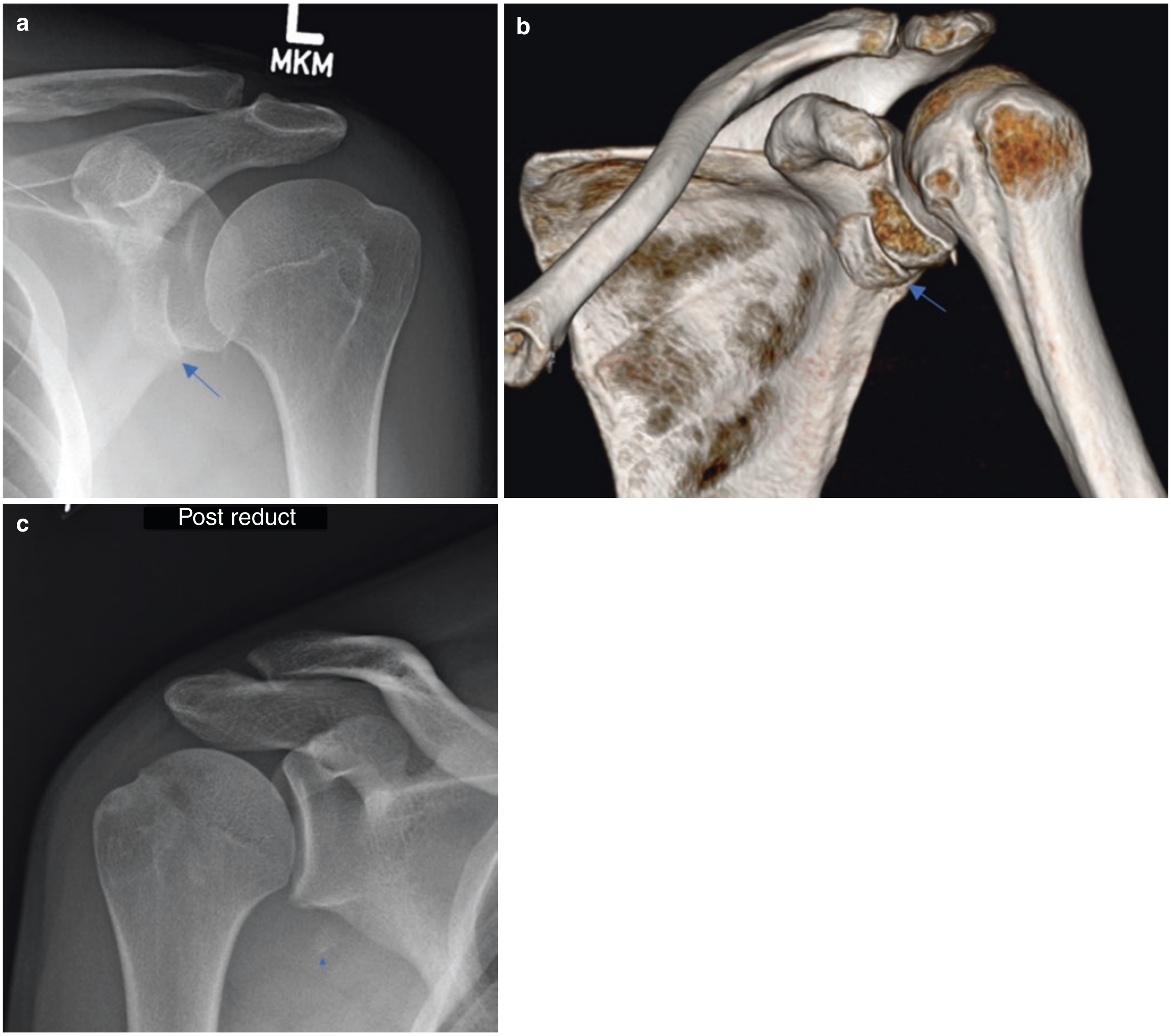

Fig. 14.3 Bony Bankart (arrows) lesion in a 52-year-old male (**a**), (**b**) and in a 23-year-old male (**c**) as a result of anterior GHJ dislocations evident on post-reduction plain radiographs (**a**), (**c**) and 3D CT (**b**)

Following reduction, a second set of radiographs should be obtained, and a more detailed examination completed. This should include neurovascular examination which should particularly focus on the function of the axillary nerve [13]. Patients with an axillary nerve injury may have decreased sensation of the lateral upper arm and decreased motor function of teres minor and deltoid (weakness with shoulder abduction and external rotation) [1].

Following an instability event, an MRI without contrast or an MR arthrogram can be used to evaluate for soft tissue injuries [2]. A CT scan can be performed to evaluate degree of glenoid or proximal humeral bone loss (Figs. 14.4 and 14.6) [2, 13].

Initial Management

The goal of initial management of anterior GHJ dislocation is to successfully reduce the dislocation, confirm or restore normal neurovascular status, and stabilize any fracture present.

One can consider performing a reduction for this type of injury on the sideline or other pre-hospital location. However, given the low rate of neurovascular injury in anterior GHJ dislocation, the patient certainly can be sent to an urgent care or the emergency department depending on clinician level of comfort, experience, and access to imaging and higher level care.

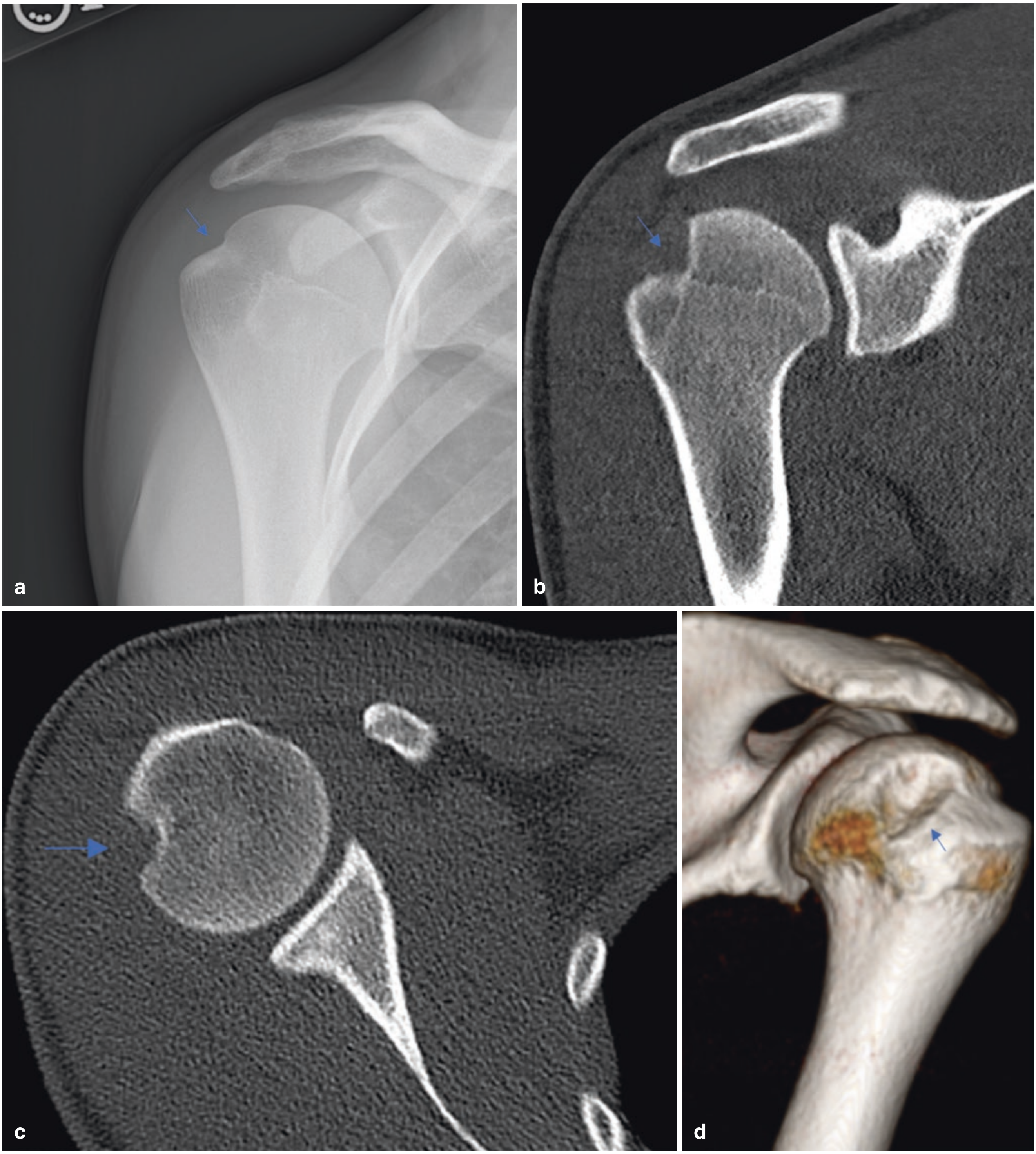

Fig. 14.4 A Hill-Sachs lesion (arrows) in a 23-year-old recreational basketball player as a result of an anterior GHJ dislocation on an AP plain radiograph view (**a**). CT (**b**), (**c**) and 3D CT (**d**) also demonstrate the lesion

Depending on the length of time since the dislocation, multiple analgesic modalities are available for reduction [16, 17]. If the athlete presents within a couple of hours of the anterior GHJ dislocation, or if this is a recurrent dislocation, a reduction attempt without analgesia is a reasonable choice [16–18]. Otherwise, oral (e.g., acetaminophen, NSAIDs, opioids), intranasal (e.g., fentanyl), inhaled (e.g., nitrous oxide), or intra-articular (e.g., lidocaine and bupivacaine) analgesics can be administered before reduction [16, 17]. The reduction attempt should not be delayed as a successful

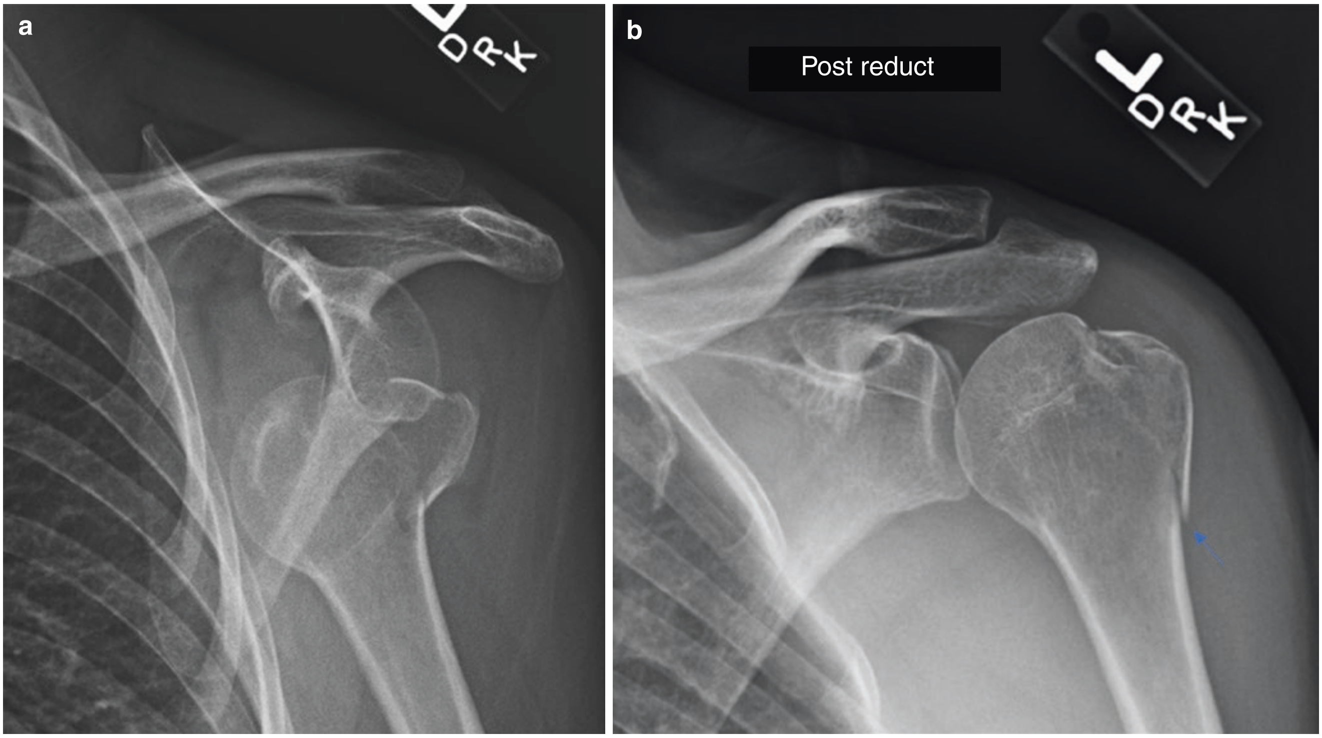

Fig. 14.5 A 52-year-old female with anterior GHJ dislocation and greater tuberosity fracture as a result of a ski injury (**a**). Post-reduction radiograph (**b**) shows the extent of the greater tuberosity fracture (arrow)

reduction is the most effective pain-reducing method in acute cases [16, 17, 19]. Intra-articular analgesic injection may be attempted either in the field or in the office [19]. Intravenous (IV) analgesics (e.g., ketamine, fentanyl, propofol) and conscious sedation may be necessary in cases of difficult reductions and if there are associated injuries (e.g., fractures) or if the athlete presents more than a few hours after dislocation (Chap. 5) [16, 17]. Unless access to radiography is going to significantly delay the reduction, plain radiography should be obtained before reduction to confirm the diagnosis and evaluate concurrent bony injuries [16, 17].

A myriad of reduction techniques have been described for anterior GHJ dislocations, with the optimal often depending on the clinical situation. There is an exhaustive list of GHJ reduction techniques, [17, 18, 20, 21] most of which have similar maneuvers. As an essential biomechanical rule, we would recommend techniques which stabilize the glenoid. It is prudent for clinicians to be comfortable with more than one technique. In cases of difficult reduction, good anesthesia (e.g., conscious sedation) and the use of multiple assistants are essential. In this section we only review a few of these techniques.

The *Stimson* technique is performed with the patient in a prone position, with the affected arm hanging over the side of the bed (Fig. 14.9). The patient's torso is stabilized, and a weight (usually 3–7 kg) attached to the wrist. This provides sustained downward traction, and within 15–30 minutes, the peri-humeral muscles will relax and the shoulder will reduce itself, with occasional clinician facilitation [16, 17, 20, 22].

The *Chair* technique is performed with the patient sitting in a chair with the affected arm hanging over the backrest (Fig. 14.10). Downward traction is applied to the arm until the reduction occurs [16, 18, 20]. To protect axillary neurovascular structures, padding of the axilla is recommended.

The *Kocher* technique can be performed in a seated or supine position. The maneuver starts with the elbow in flexion and arm in adduction (Fig. 14.11). Then, the shoulder should be externally rotated until resistance is felt. At this point the shoulder should be flexed and adducted as far as possible and, finally, the shoulder internally rotated [20, 22]. Many modifications to the Kocher technique have been described including the use of more forward flexion than originally described [17].

The *Milch* technique is performed with the patient sitting or supine (Fig. 14.12). The clinician starts with the arm in 10°–20° of forward flexion and continues to elevate the arm, applying traction but stabilizing the humeral head in position, until the arm is overhead [17, 20, 22]. The clinician will then attempt reduction by applying gentle, direct manipulation of the humeral head, pushing it over the rim of the glenoid. If this is not successful, the clinician may subsequently bring the arm down through a full lateral arc until reduction occurs.

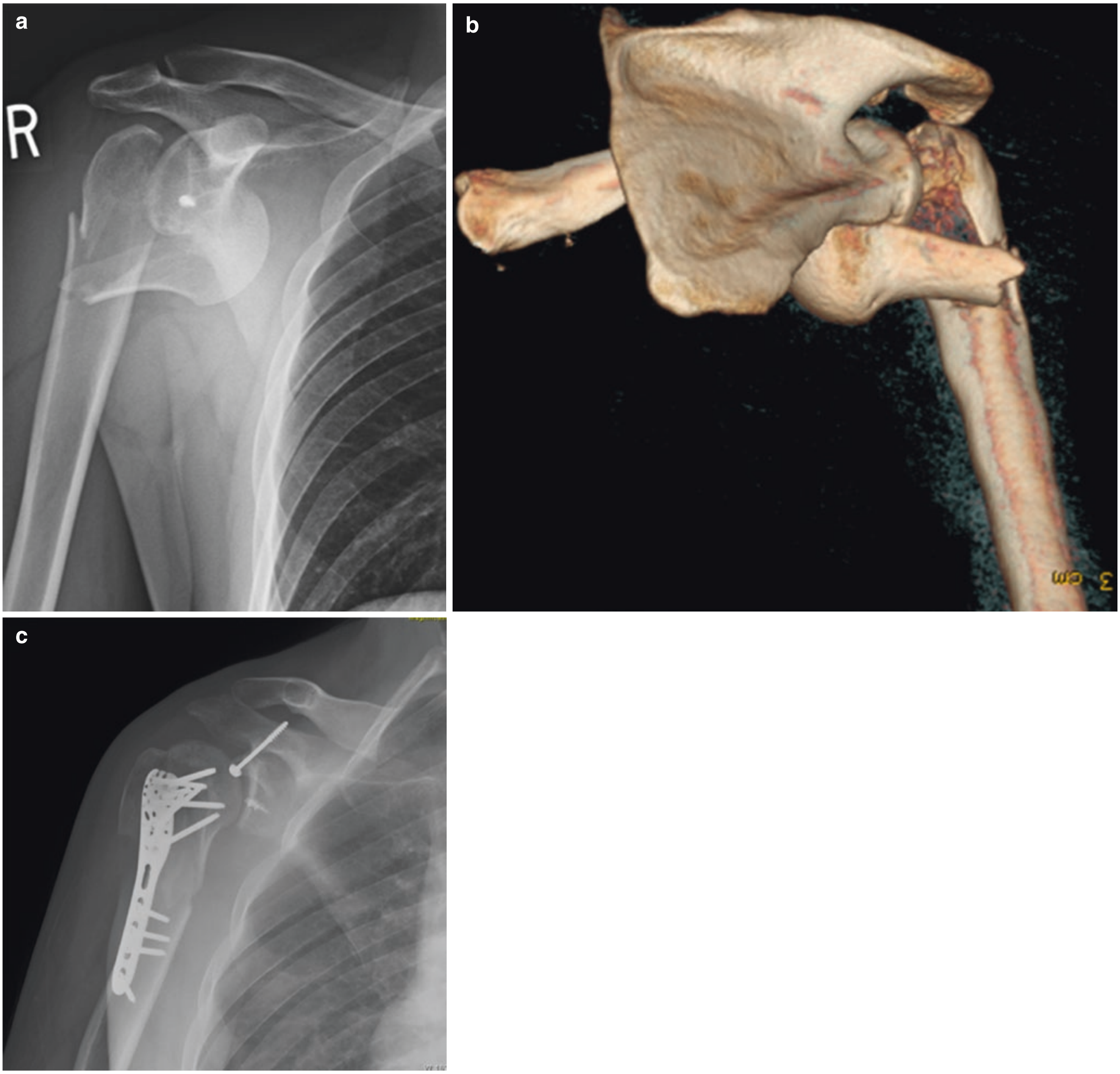

Fig. 14.6 A 33-year-old male with anterior GHJ dislocation and proximal humerus fracture as a result of a ski injury (**a**). 3D CT revealed the extent of the fracture (**b**). Post-operative radiograph (**c**) shows fixation of the proximal humerus and glenoid fractures

In the *scapular manipulation* technique, the glenoid is repositioned, rather than the humeral head. This can be performed with the patient prone, sitting, or supine (Fig. 14.13). This technique usually requires two clinicians. One will place traction on the humerus while lifting (forward flexion) the arm into 90° of flexion. Simultaneously, the other clinician manipulates the scapula, rotating the inferior aspect of the scapula upward and/or medially and rotating the superior aspect of the scapula laterally. The *modified scapular manipulation* (*stabilization*) technique is usually performed in a seated position (Figs. 14.13d and 14.14b). One clinician stabilizes the lateral border of the scapula, while another clinician stabilizes the anterior shoulder with one hand and applies traction with another hand [17, 20, 22, 23]. One meta-analysis showed this technique to be the most successful (97%), fastest (mean 1.75 minutes to reduction), and least painful technique examined [21].

In the *traction-countertraction* method, the patient is positioned supine (Fig. 14.15). One assistant has a towel or sheet around the patient's chest and applies countertraction. Simultaneously, the clinician grasps the hand (Fig. 14.15a) or elbow flexed at 90° (Fig. 14.15b). As the physician leans

Fig. 14.7 A patient with anterior GHJ dislocation keeping his arm in abducted position. A deformity (sulcus sign) is also present

back, traction is applied to the forearm with the shoulder in 45° of abduction [17, 18, 20]. A modification of this technique has also been described (Fig. 14.16).

The *Boss-Holzach-Matter* technique describes a maneuver that the patients perform largely themselves [20, 22]. Starting in an upright seated position, the patient wraps their forearms around the ipsilateral knee flexed at 90° (Fig. 14.17). The head of the bed is lowered, and the patient is instructed to lean back, hyperextend the neck, and shrug the shoulders anteriorly.

If a GHJ dislocation is encountered in the field, definitive indications to send for higher level of care include inability to reduce either due to injury or patient factors, and poor neurovascular status before or after reduction.

Post-reduction radiographs should be obtained to confirm the reduction and to evaluate associated bony pathologies [16, 17]. Following reduction, patients are typically immobilized in a sling for comfort with the arm in a position of adduction and internal rotation. Though some have traditionally advocated for prolonged immobilization in either internal or external rotation to aid in soft tissue healing, recent studies show that early range of motion and even accelerated rehabilitation are safe and assist with early return to sports [2, 3, 13, 24].

Indications for Orthopedic Referral

Surgical intervention is typically not necessary for an anterior GHJ dislocation. In the case of difficult reduction, closed reduction may require use of IV analgesia, conscious sedation, or general anesthesia. In the case of neurologic or vascular compromise, closed reduction remains the fastest solution. In the case of concomitant fracture, closed reduction has still been shown to be safe and effective [25]. Patients with irreducible anterior GHJ dislocations should be immediately referred to a hospital with open reduction capabilities. The utility of surgical intervention for patients following primary GHJ dislocation is an area of controversy. It is standard practice to treat many first-time dislocators nonoperatively [2, 26]. However, there have been some military and civilian studies which have shown favorable outcomes regarding risk reduction for recurrent shoulder instability with primary repair [5]. Based on the algorithm proposed by Owens et al. in 2012, indications for surgical repair include a large bony Bankart lesion, osseous defect of the glenoid or humeral head >25%, recurrent instability, timing near the end of the athlete's competitive season, and inability to regain full sport-specific function with nonoperative care [2, 26]. Due to high rates of recurrent instability, surgical repair is typically indicated in young athletes with labral lesions as well as patients with recurrent instability [12]. For this reason, patients 30 years of age and older likely do not require orthopedic referral following a primary anterior GHJ dislocation unless significant bony pathology is evident on imaging. If surgical treatment is pursued, the question of arthroscopic versus open treatment with or without bony augmentation needs to be considered, but is beyond the scope of this discussion [2, 27].

Follow-Up Care

The goal of follow-up management of a shoulder dislocation should be to restore full, painless shoulder function and reduce the risk of recurrent instability [2, 4]. If initial surgical management is not indicated, the patient should undergo an initial period of immobilization as described above. Patients may then undergo rehabilitation to aid in restoring range of motion, strength, stabilization, and function [2, 4]. Following arthroscopic anterior shoulder stabilization, patients are kept in a sling with a small abduction pillow for about 6 weeks, with physical therapy starting 1–2 weeks after surgery. Patients are allowed to return to play at 18–24 weeks based on range of motion, strength, and sport-specific ability.

Return to Sports

Following a primary anterior GHJ dislocation, nonoperative management of an in-season injury can allow a quick return to play in as little as 7–21 days [26]. However, this may place the athlete at an increased risk for recurrence or further instability, especially if that athlete is a throwing or overhead athlete. In order to return to any sport or physical activity, a patient should have achieved full range of motion, strength,

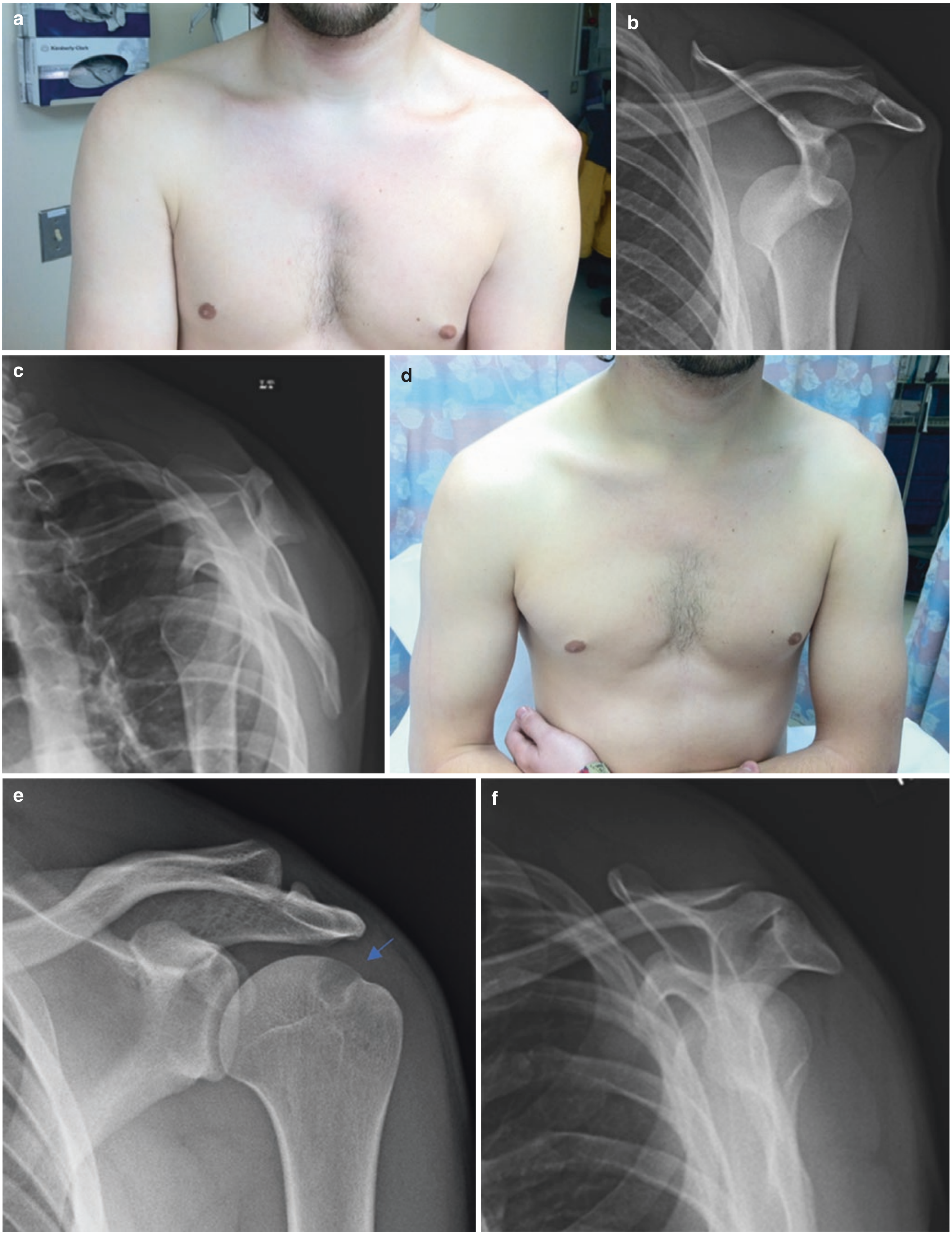

Fig. 14.8 A 23-year-old male with a left shoulder deformity (sulcus sign) as a result of a fall while skiing (**a**). AP (**b**) and scapular Y (**c**) views confirmed the diagnosis of an anterior GHJ dislocation. Post-reduction, the sulcus sign is resolved (**d**). Post-reduction AP (**e**) and scapular Y (**f**) views show a successful reduction and a Hill-Sachs lesion (arrow)

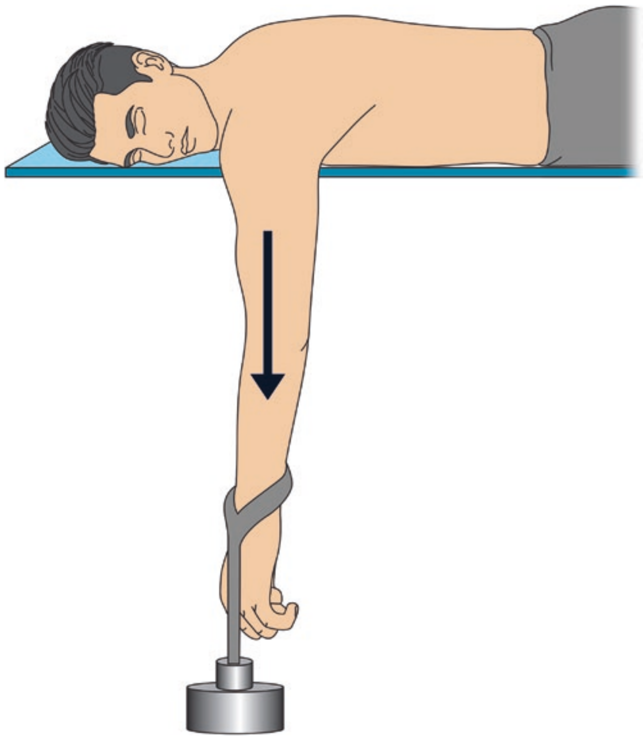

Fig. 14.9 Stimson maneuver

and confidence in their sport-specific shoulder function [2, 4, 13, 26]. Once they have reached this goal, team physicians will often allow athletes to return to sport with a stabilizing shoulder brace, though there is no evidence to show that these prevent recurrent instability [12, 13]. A prospective observational cohort study of National Collegiate Athletic Association (NCAA) athletes, the US Military Academy, US Naval Academy, and US Air Force Academy showed that following an anterior shoulder instability event, 73% of athletes were able to return to play in-season [28]. Mean time loss from competition was less than 1 week. However, in this population, rates of recurrent instability have ranged from 37% to 90% [2, 13, 28]. Based on this data, proposed contraindications for return to sport in-season include failure of nonoperative treatment, large/engaging Hill-Sachs lesion, glenoid bone loss greater than 20%, previous failed stabilization procedure, and HAGL lesion [28]. Based on the results of several studies, a proposed return to play algorithm advocates a period of brief immobilization followed by accelerated rehabilitation for 2–3 weeks with return to sport in a brace once the athlete achieves full symmetric painless range of motion and strength with ability to perform sport-specific skills without reservation [2, 13, 26]. A return to sport time frame of approximately 3 weeks is typically considered

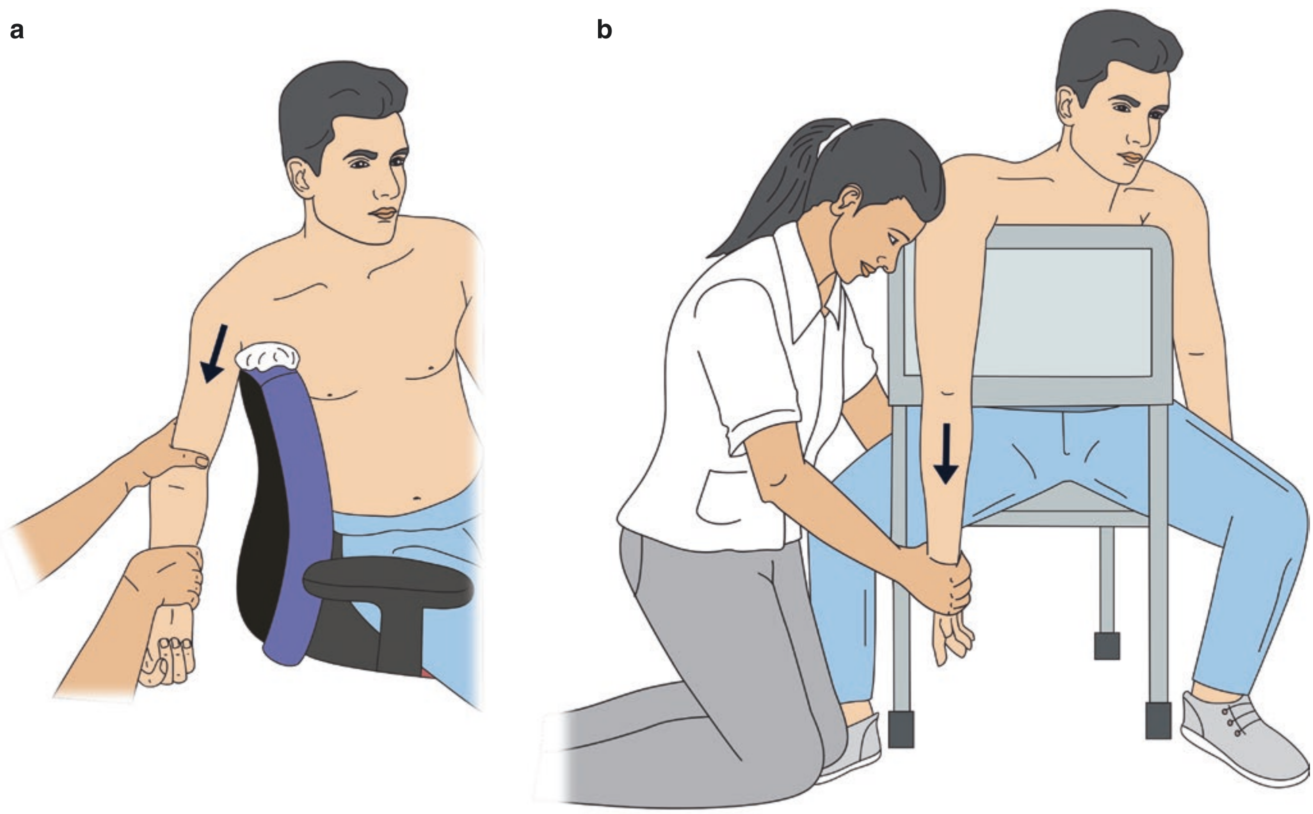

Fig. 14.10 Chair technique. Downward traction can be applied when patient sits sideways (**a**) or backwards (**b**) on a chair with elbow in flexion or extension

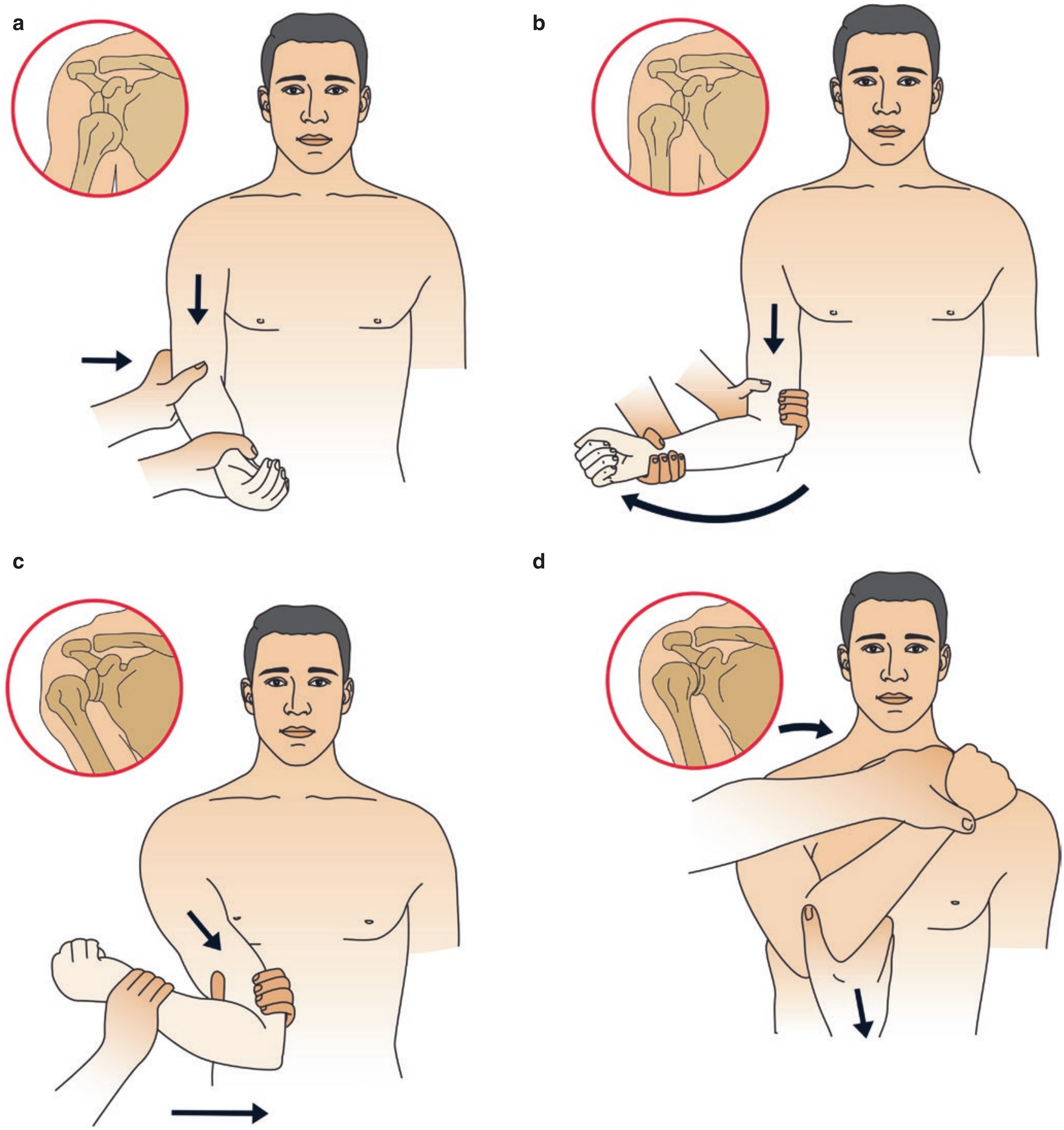

Fig. 14.11 Kocher technique. Use gentle inferior traction throughout the maneuver. Flex the elbow and adduct the arm against the body (**a**). Externally rotated the shoulder until you feel resistance (**b**). Then flex and adduct the shoulder as far as possible (**c**). Internally rotate the shoulder (**d**)

reasonable [13]. Return to sport after surgical repair is considered separately. This time frame typically ranges from 4 to 6 months postoperatively, with patients progressing through phases of strict sling immobilization, followed by progressive strengthening and range of motion and finally sport-specific function [13]. With improved arthroscopic surgical techniques and accelerated rehabilitation protocols, time loss from competition after a primary anterior GHJ dislocation has been reduced [29], with a rate of successful return to contact sports of 90% [13].

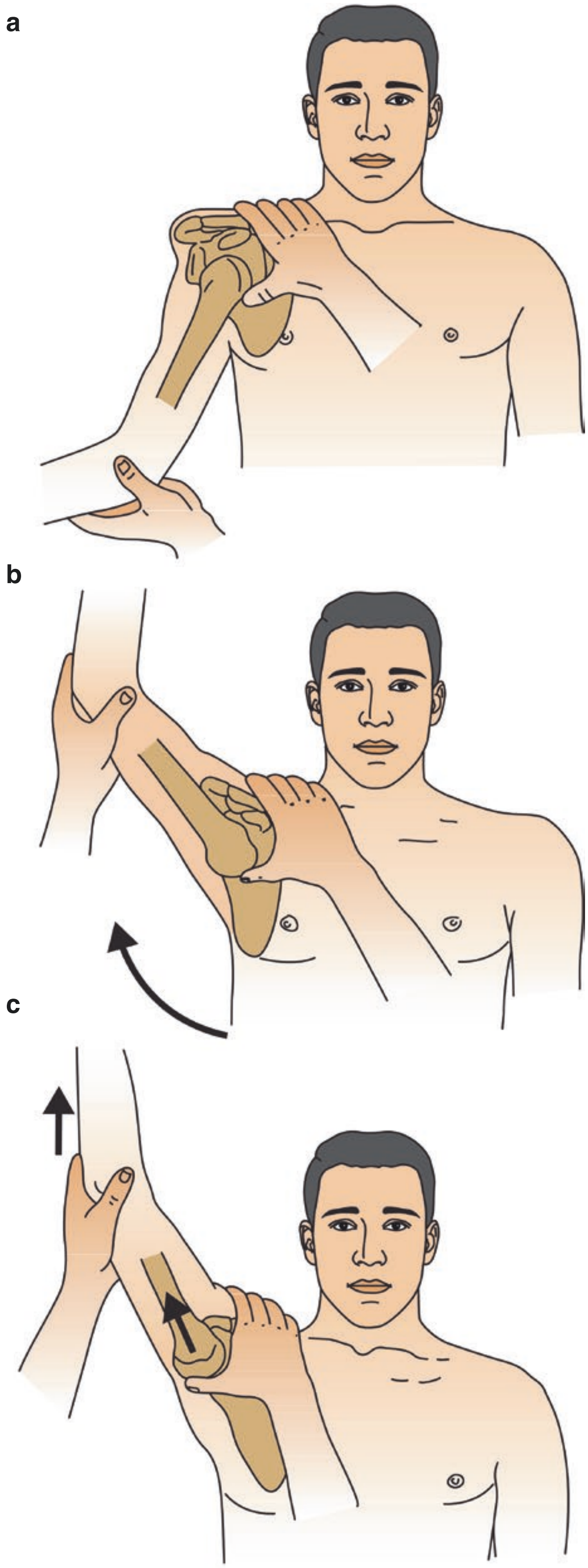

Fig. 14.12 Milch technique. Start with the arm in slight abduction and forward flexion (**a**). Abduct and forward flex the arm until reaching overhead position (**b**). Apply gentle longitudinal traction to the arm with direct upward pressure to the humeral head (**c**)

Complications

Acute complications of anterior GHJ dislocation include fracture, neurologic, and vascular injury.

Fractures occur in about 30% of anterior GHJ dislocations [22]. The most common fracture associated with an acute anterior GHJ dislocation is a Hill-Sachs lesion on the postero-superior humeral head, followed by a bony Bankart lesion (Figs. 14.3 and 14.18), both of which are described above [22, 25]. Other possible fractures include avulsion of the greater tuberosity (Figs. 14.5 and 14.18), coracoid fracture, and humeral shaft fracture which is typically associated with significant trauma [22]. Vascular injury is thought to be uncommon, but it is more common in the elderly [1, 4]. The neurovascular structures most commonly affected are the axillary nerve and axillary artery [1, 4]. Other potential complications include injury to the brachial plexus as well as venous injuries which can result in delayed subclavian vein thrombosis. Neurologic injury is more common than vascular injury, with one study showing a rate of 45% following shoulder dislocation with a concomitant humeral neck fracture [1].

The most common chronic complication from primary anterior GHJ dislocation is recurrent instability. Rates of recurrent instability range widely depending on risk factors, but have been reported as high as 92% in young athletes [12]. Risk factors for recurrent instability include younger age at initial injury, male gender, large Hill-Sachs lesion, significant glenoid rim fracture, greater tuberosity fracture with persistent displacement, contact sport participation, atraumatic dislocations, HAGL lesion, and bone loss greater than 10%–20% [2, 4]. Recurrent instability can also increase the likelihood of arthritic changes developing over time, and result in progressive glenoid bone loss [2, 4, 12, 13].

Posterior Dislocations

Epidemiology

Posterior dislocations account for less than 2% of all GHJ dislocations [30, 31]. A majority of posterior GHJ dislocations are due to nonathletic causes (e.g., seizures, electrocutions), and predominantly in middle-aged men [30–33].

Mechanism of Injury in Sports

A typical mechanism for this injury is forceful adduction with internal rotation and flexion of the shoulder or a direct blow to the anterior shoulder [31]. Posterior GHJ dislocation is usually seen in high-speed sports such as skiing and motor sports [30, 31, 33, 34].

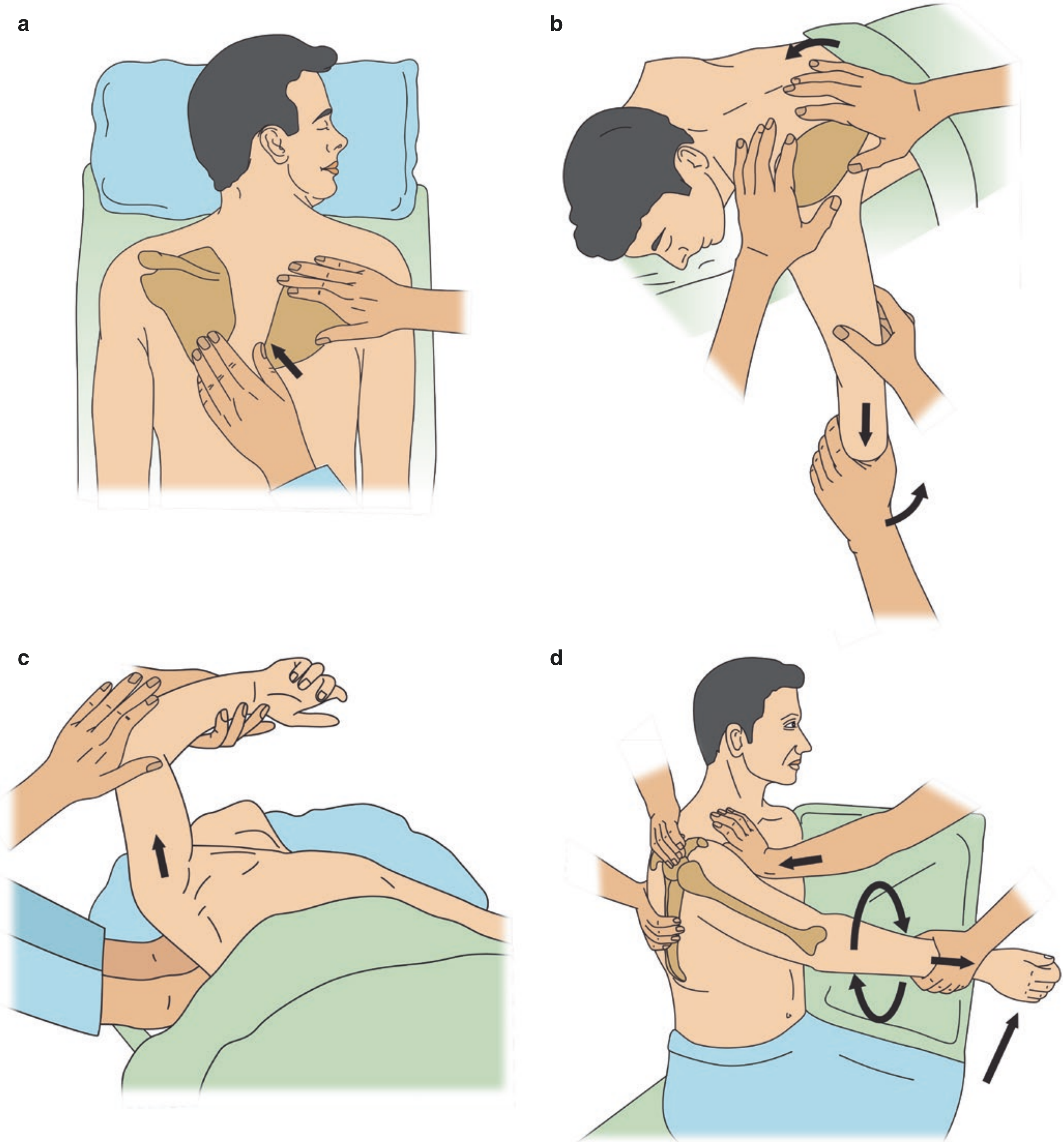

Fig. 14.13 Scapular manipulation technique. Push the inferior tip of the scapula medially while patient is prone (**a**). At the same time, a downward traction with internal and external rotation motion can be applied (**b**). The same maneuver can be performed in supine position (**c**). Modified scapular manipulation (stabilization) technique (**d**). While patient is seated, the scapular lateral border is stabilized by one clinician while the second clinician starts passive forward flexion and applies forward traction with one arm and stabilizes the shoulder from forward movement with another hand. Gentle internal and external rotation movement can be applied as well

Fig. 14.14 Left anterior GHJ dislocation with obvious deformity (**a**). Reduction was achieved, using a modified scapular manipulation (stabilization) technique (**b**). Post-reduction, the deformity has resolved (**c**)

Dislocation Classification

Posterior GHJ dislocations should be classified based on chronicity and associated injuries such as fractures (e.g., greater tuberosity, Hill-Sachs, glenoid, and clavicle) and soft tissue injuries (e.g., rotator cuff tear and labral tear) [33, 35].

Clinical Presentation

As patients may not be able to provide adequate history, a high level of suspicion should be maintained in a patient with recent trauma and inability to perform full shoulder range of motion. On observation, athletes may present with a dimple or flattening of the anterior shoulder and posterior shoulder prominence [31, 34, 35]. However, the deformity may be very subtle (Fig. 14.19). Patients usually have limited external rotation of the shoulder and hold the arm in a "sling" position [31, 34]. Partial forward flexion and abduction (up to 90°) may be preserved [32]. Neurovascular examination should be performed before and after reduction. However, the rate of neurovascular injuries is lower compared to anterior GHJ dislocations [35].

Diagnosis

Plain radiography should be performed on all athletes with suspected posterior GHJ dislocations. Standard shoulder views including AP, oblique (Grashey), and scapular Y are important initial views (Fig. 14.20). Four out of five patients with posterior GHJ dislocations are missed due to not obtaining an axillary view due to patient's discomfort [30, 31]. On the AP view, the humeral head may demonstrate the "lightbulb sign" (Fig. 14.20) [30, 31]. If the diagnosis of a posterior GHJ dislocation is inconclusive, an axillary (or at least a Velpeau axillary) view should be obtained (Fig. 14.21) [30, 31]. A portable ultrasound in expert hands is a useful modality to diagnose posterior GHJ on the sideline or in cases with inconclusive plain radiographs [36].

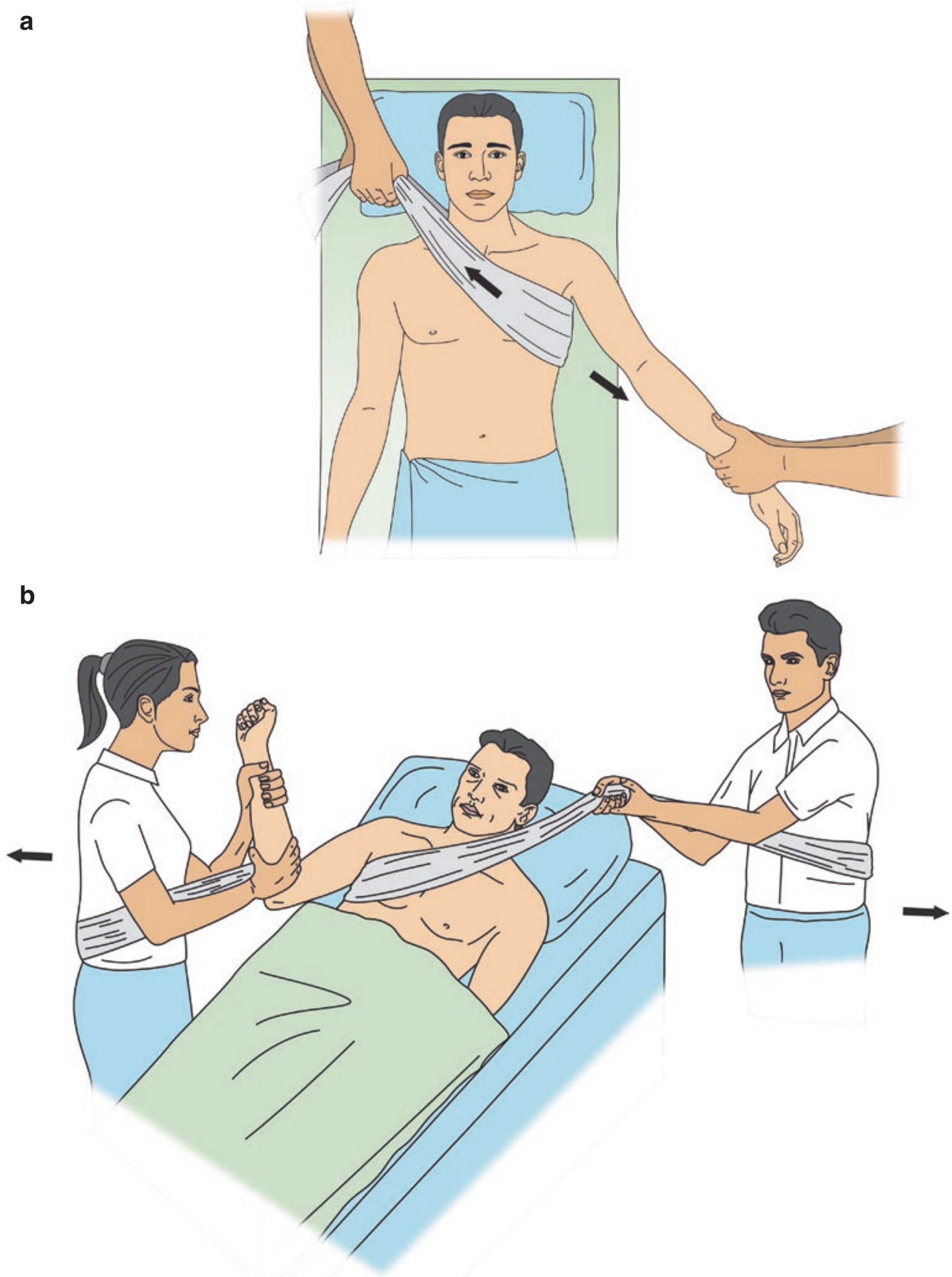

Fig. 14.15 Traction-countertraction technique. This can be performed with grabbing the hand (**a**), or using a sheet when the elbow is flexed (**b**)

Initial Management

Current literature mainly focuses on atraumatic, subacute or chronic dislocations due to delay in diagnosis. For these cases, conscious sedation or general anesthesia is recommended for reductions [30, 31]. There is limited literature on acute and traumatic posterior GHJ dislocations [30–33, 37]. It seems that acute traumatic posterior GHJ dislocations can be managed similarly to anterior dislocations [31]. However, due to high rates of associated bony and soft tissue injuries, particular attention should be given to these potential injuries [30, 31, 33]. In addition to applying direct pressure to the posterior humeral head, a combination of longitudinal traction, forward flexion, and internal and external rotation of the shoulder is recommended for reduction [30, 31, 34, 35, 38].

Indications for Orthopedic Referral

Patients with irreducible posterior GHJ dislocations should be immediately referred to a hospital with open reduction capabilities. Due to the high rate of associated bony and soft

tissue injuries, most athletes with posterior GHJ dislocations should be referred to an orthopedic surgeon within a week of closed reduction [30–32]. All athletes with chronic posterior GHJ dislocation should also be referred to an orthopedic surgeon as soon as possible [30–32, 37].

Follow-Up Care

If a patient is to be managed nonoperatively, follow-up within 1–2 weeks is recommended. Otherwise, follow-up care is similar to anterior GHJ dislocation.

Return to Sports

Return to sports following posterior GHJ dislocation is similar to that for anterior GHJ dislocation.

Complications

Missed or delayed diagnosis of a posterior GHJ dislocation is a preventable event which can have significant consequences such as chronic pain, muscle atrophy, and neurovascular compromise [30–32, 37]. Individuals with delayed diagnosis usually require complex shoulder surgeries, which may eventually result in functional disability [30–33, 37].

Inferior Dislocations

Epidemiology

Inferior dislocations, also called luxatio erecta, are the most rare type of dislocation of the glenohumeral joint, accounting for about 0.5% of dislocations [39, 40]. The mean age at presentation is 31–40 years old, although it has been reported in patients 3 months to 80 years of age [41, 42, 43]. Males account for 80% of patients with inferior GHJ dislocations [39, 43, 44].

Mechanism of Injury in Sports

Most inferior dislocations are a result of one of two mechanisms. A direct axial load on an abducted arm can force the humeral head inferiorly with respect to the glenoid. Forceful hyperabduction on an already partially abducted arm can lever the humeral neck against the acromion and force the head inferiorly. Both of these mechanisms generally occur during a fall, as the patient attempts to catch him/herself with an outstretched arm [39, 42]. Although motor vehicle accidents or falls from a height are the most commonly associated mechanism, inferior dislocations associated with sports have also been reported [39, 44]. Three out of six cases of inferior GHJ dislocation reported by Mallon et al. were due to a hyperabduction force while playing football [44]. Groh et al. reported 16 cases of inferior dislocations, of which 6 were associated with sports including basketball, skiing, biking, mountain climbing, and bull riding [39]. Sports involving contact or the potential for high speed falls or falls from a height place athletes at highest risk for this injury. Medical personnel covering contact sports such as football, basketball, lacrosse, and hockey, or extreme sports such as skiing, BMX or mountain biking, mountain climbing, motocross, and skateboarding may come across this injury. Unilateral injury is most common, though there are several case reports of bilateral luxatio erecta [39, 42–48].

Dislocation Classification

Luxatio erecta is a unique type of inferior GHJ dislocation. However, some patients with a "true" subglenoid anterior GHJ dislocation can be placed in inferior GHJ dislocation category as well. These patients do no present with extreme arm abduction as seen in patients with luxatio erecta.

Clinical Presentation

The clinical presentation of an inferior GHJ dislocation is distinct. The athlete typically presents with significant pain with the affected arm locked in a hyperabducted position above the head, usually with the elbow flexed. The humeral head may be palpable or visible in the axillary fossa, or in some cases anterior to the fossa along the chest wall. Neurovascular injury is relatively common with inferior GHJ dislocation. Approximately 60% of inferior dislocations are accompanied by a neurologic injury, most commonly to the axillary nerve [39, 44]. Ulnar, median, and radial neuropraxiae have also been reported [39, 44]. These neurologic injuries generally resolve, but the length of recovery varies widely between 1 day and 1 year [39, 44]. True arterial or venous injury is less common than neurologic injury, reported in 3.3% of cases [39, 48–51]. Arterial injury is more commonly reported in patients over age 50, likely due to underlying atherosclerosis [51]. Arterial compression was noted in 7 of 18 shoulders prior to reduction in one series, and these patients presented with abnormal radial and/or ulnar artery pulses [39]. All of these returned to normal following reduction without further sequelae. The incidence of vascular injury is higher when neurologic injury is present, and a high index of suspicion should be held when initially examining these athletes. Concomitant

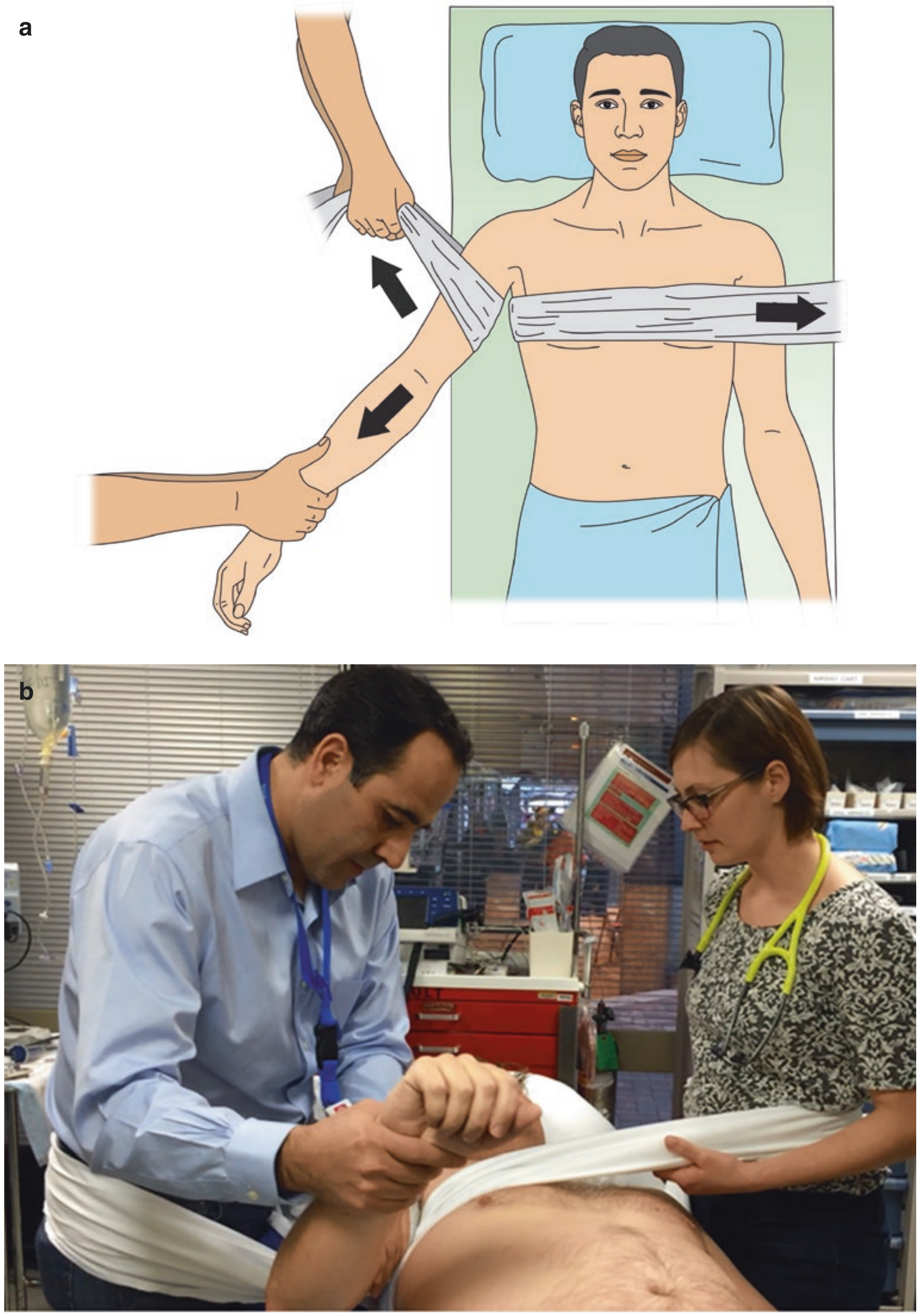

Fig. 14.16 Modified traction-countertraction technique. One person stabilizes the torso and another person applies lateral traction to the proximal arm (**a**), (**b**). Gentle internal and external rotation can be applied as well

musculoskeletal injuries are present in 80% of patients with inferior GHJ dislocations [39, 44]. Fractures about the shoulder such as the humeral head, greater and lesser tuberosity, acromion, coracoid, and clavicle are most common. Individuals with inferior GHJ dislocation should be evaluated for fractures of the spine, and more distal fractures of the upper extremity such as humeral shaft, radius, ulna, or hand, given the common mechanism of a high energy sports injury or fall. Soft tissue injuries about the shoulder are also frequent. The joint capsule and inferior labrum are nearly universally torn. The rotator cuff is commonly torn in older patients. It is thought that a tuberosity fracture is protective of a rotator cuff tear but occurs through the same mechanism. In younger patients, acromioclavicular joint dislocation (separation) may accompany the inferior GHJ dislocation.

Fig. 14.17 The Boss-Holzach-Matter method

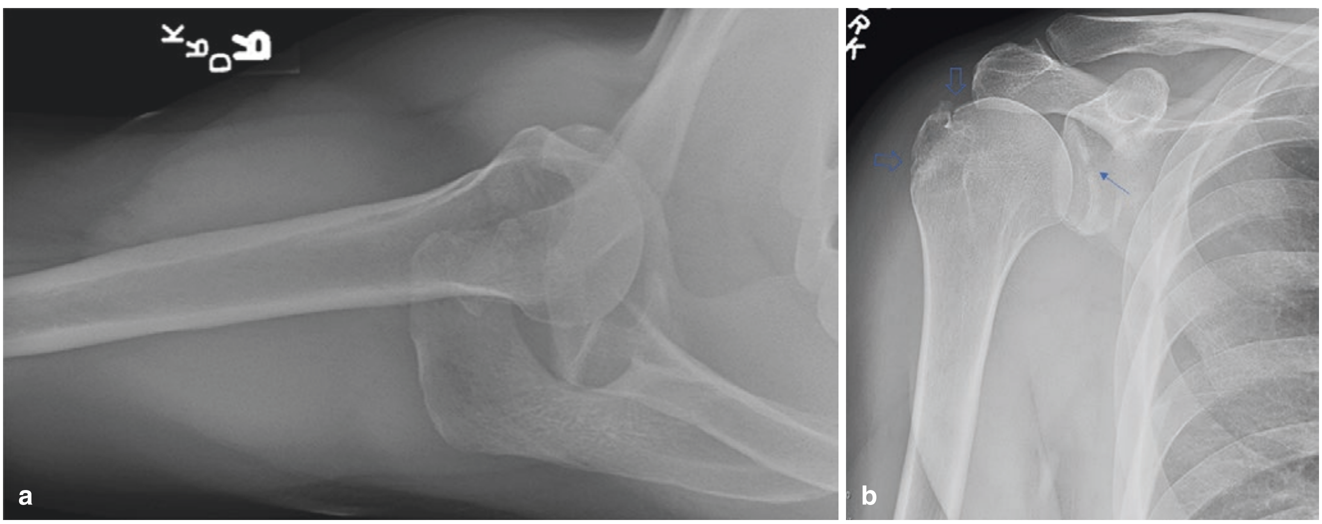

Fig. 14.18 Anterior GHJ dislocation in a 44-year-old male as a result of a ski injury evident on axillary view (**a**). Post-reduction AP confirmed the glenoid (arrow) and greater tuberosity (open arrows) fractures (**b**)

Diagnosis

Diagnosis of luxatio erecta is generally made prior to imaging as the clinical presentation is pathognomonic, though plain radiography is an important part of the initial workup (Fig. 14.22). AP, scapular Y, and axillary views comprise the standard series. Due to the already abducted position of the arm, the axillary view can be obtained in most cases. The location of the humeral head and any other fractures about the shoulder can be visualized, which can guide the reduction maneuver and further imaging. The same series of plain radiography should be obtained following reduction. CT or MRI may be indicated for evaluation of associated fractures or soft tissue injury. Due to the frequency of associated neurovascular injuries, a detailed neurological examination of the axillary, median, radial, and ulnar nerves, palpation of radial and ulnar pulses, and examination of capillary refill should be performed before and after reduction.

Initial Management

Sideline reduction is only recommended if the provider feels confident with the diagnosis and comfortable with reduction techniques. Given the higher potential for neurovascular complications, early reduction is advised. However, the decision on sideline reduction attempts should be based on many factors including the accessibility to plain radiography and distance from an emergency

Fig. 14.19 A 40-year-old male with right posterior GHJ dislocation keeping his arm in adducted position. A subtle deformity (sulcus sign) is also present

department. Reduction of an inferior GHJ dislocation requires adequate analgesia and often sedation. If a reduction attempt is performed on the field, injection of intra-articular lidocaine may provide some analgesia and aid in reduction in this setting. However, injection should not be performed using an axillary approach due to potential risk of damaging the axillary nerve. The classic reduction maneuver is performed with the patient in a supine position. Traction is applied longitudinally in line with the abducted affected arm while a second person applies countertraction. Slight overabduction can be applied to the affected arm to lever the humeral head back onto the glenoid, and when this occurs, gentle adduction down to a neutral position places the shoulder at rest [52].

An alternative reduction technique involves a two-step maneuver that initially converts the inferior GHJ dislocation to an anterior GHJ dislocation [53]. The operator stands at the head of the patient facing caudad and uses the arm closest to the patient to push on the lateral aspect of the humeral shaft to direct the humeral head anteriorly and uses the hand farthest from the patient to pull the limb at the medial epicondyle with a superiorly directed force [53]. The arm should be able to be adducted close to the body at this time. The second step of the reduction can be performed using any of the previously described techniques for an anterior GHJ dislocation.

Following reduction, the limb should be placed into a sling in a resting, adducted position for comfort. Assessment of the neurovascular status of the limb is critical both before and after reduction. Plain radiography is recommended following reduction to ensure complete reduction and to evaluate for associated fractures.

Indications for Orthopedic Referral

Cases of inferior GHJ dislocation which are not reducible by closed means should be referred for orthopedic evaluation in the emergency department as this may warrant urgent surgical intervention for reduction. Several structures have been reported to impede a closed reduction including the capsule, fracture fragments, and the axillary nerve [54, 55]. Following successful closed reduction in cases without vascular complications, referral to an orthopedic surgeon is recommended within 1 week. Consultation with a vascular surgeon is indicated emergently if any residual abnormality of vascular status persists following reduction or with any sign of evolving hematoma in the axillary fossa. Advanced imaging may be obtained with CT for evaluation of tuberosity, acromial, or coracoid fractures, and MRI for evaluation of the labrum, capsule, rotator cuff tendons, and biceps tendon. Findings on advanced imaging can help to direct rehabilitation guidelines and may indicate the need for surgical intervention. Cases of recurrent instability should be referred for orthopedic surgical consultation.

Follow-Up Care

Follow-up care and rehabilitation after this injury will depend largely on the nature of the associated injuries such as neurovascular structures or fractures. In uncomplicated cases, patients are treated with sling immobilization for 3–4 weeks. Gentle passive range of motion exercises may be started under the direction of a physical therapist as soon as 1 week after closed reduction, followed by a progression through active range of motion and strengthening over a 1- to 6-month time frame.

Return to Sports

As with other GHJ dislocations, return to sports depends on the return of full range of motion and strength without pain. Given the higher energy mechanism of inferior dislocations

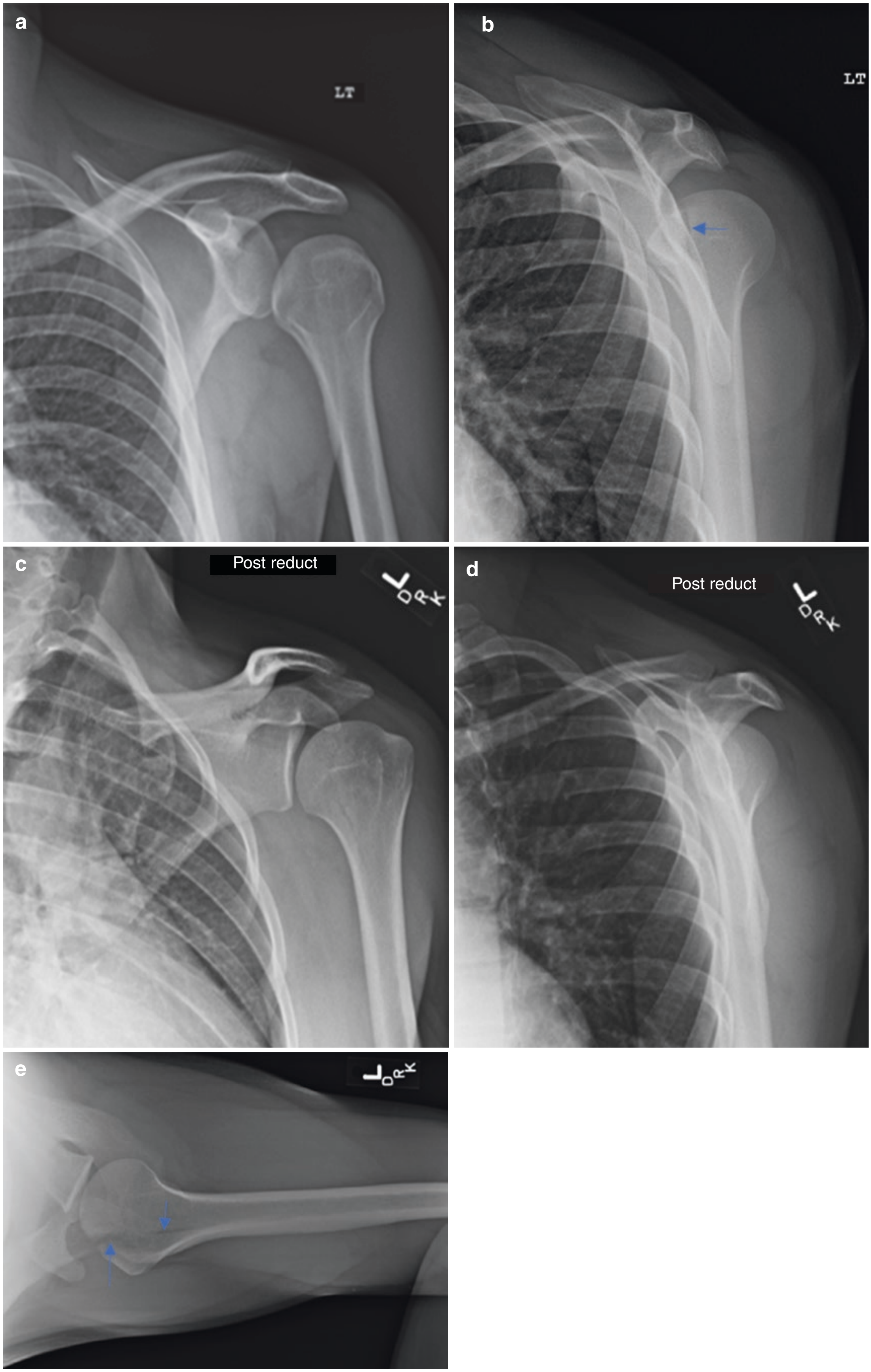

Fig. 14.20 Posterior GHJ dislocation in a 29-year-old male as a result of a ski injury. AP view (**a**) shows "light-bulb" appearance. Scapular Y view (**b**) shows posterior displacement of the humeral head and greater tuberosity fracture (arrow). Post-reduction Grashey (**c**) and scapular Y views (**d**) confirmed the reduction. Post-reduction axillary view (**e**) demonstrates improved visualization of the fracture (arrows)

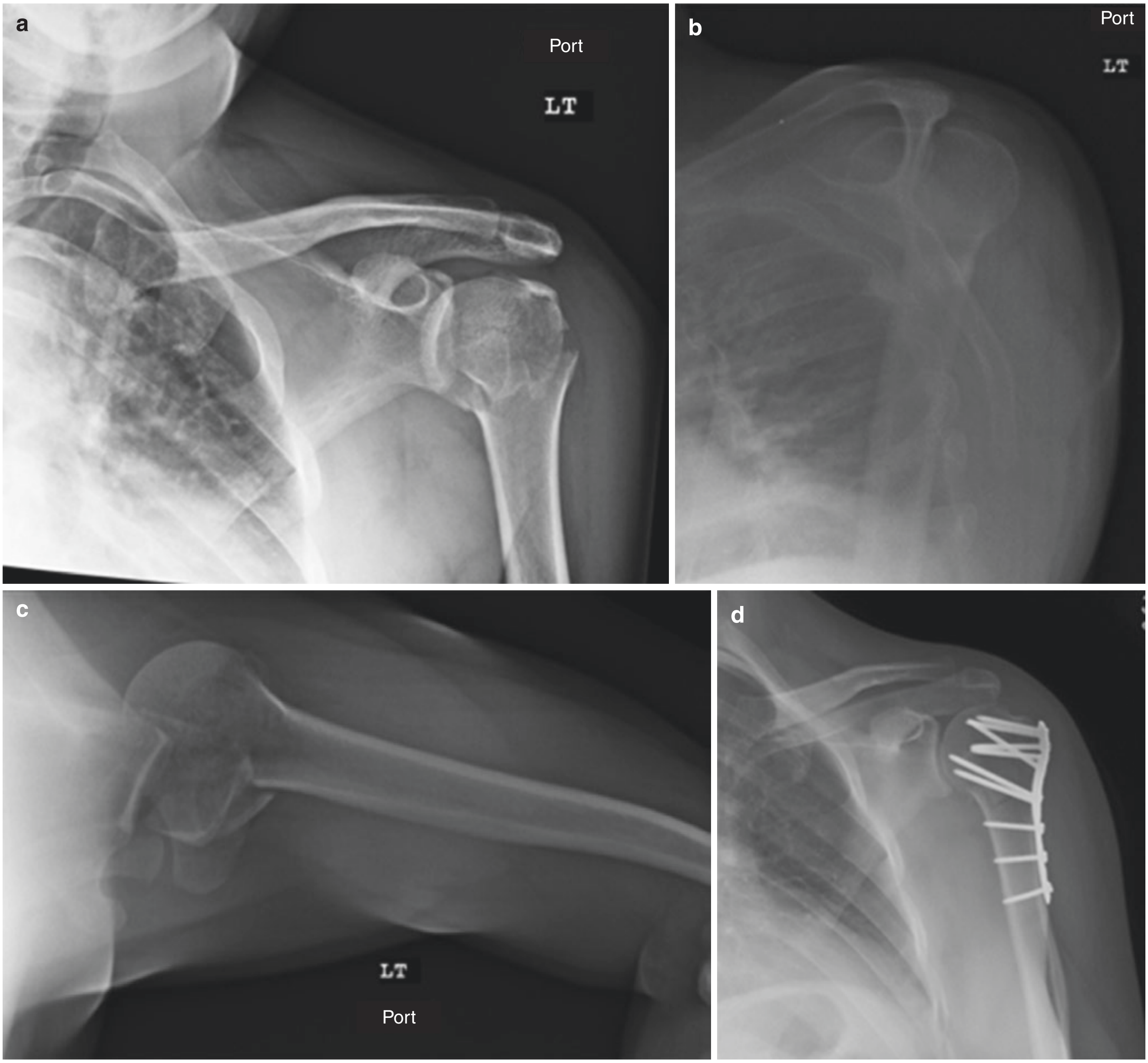

Fig. 14.21 Posterior GHJ dislocation in a 39-year-old male as a result of a ski injury. AP view (**a**) shows dislocation and comminuted humeral head fracture. Scapular Y (**b**) and axillary (**c**) views confirm the diagnosis. Post-operative AP view (**d**) demonstrates good alignment

compared to most anterior or posterior GHJ dislocations, the increased surrounding tissue trauma may portend a longer recovery. Patient factors such as age, sport and position played, and pre-injury conditioning also play a role [29, 56]. In cases of associated neurologic injury, the time needed for recovery of function is widely variable. Return to sports is not recommended until full recovery of nerve function. Fractures of the humeral tuberosity, clavicle, or acromion will generally be protected for 6–8 weeks prior to the strengthening phase of rehabilitation. The variability in injury severity and patient factors leads to the need for an individualized return to sport rehabilitation plan for each athlete [29, 56].

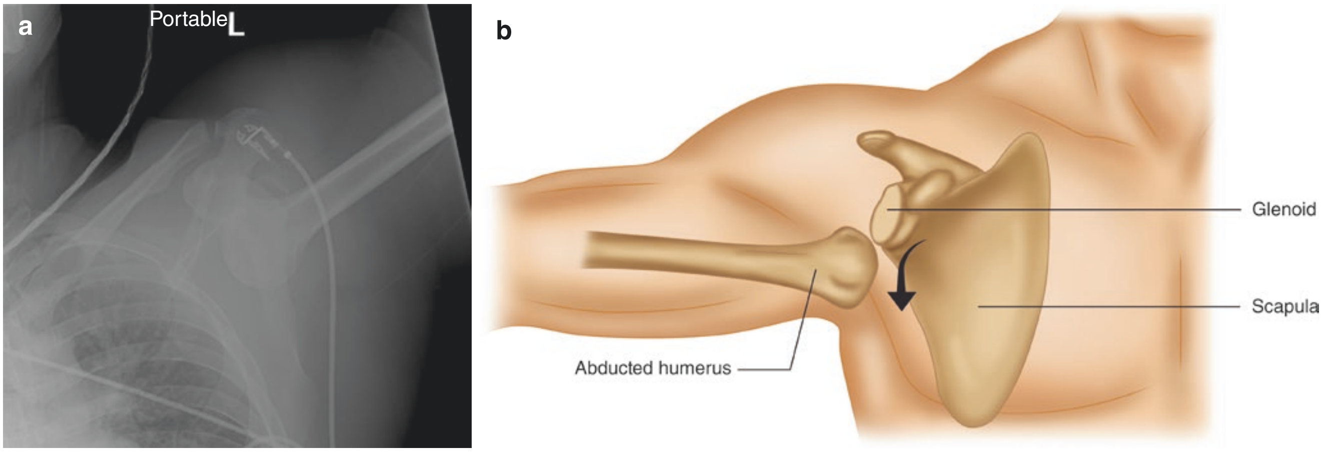

Fig. 14.22 Luxatio erecta in a 30-year-old man as a result of an ejection from a car while unrestrained during a motor vehicle collision (**a**). The illustration shows the relationship between the humeral head and the glenoid (**b**)

Complications

Complications of inferior GHJ dislocations are similar to the other types of GHJ dislocations. However, the rates of neurovascular injuries, associated fractures, and irreducible dislocations are higher.

Pediatric Considerations

Traumatic GHJ instability in patients younger than 20 years of age accounts for about 20% of all traumatic dislocations, with 2% occurring in patients younger than 10 years of age [57]. Anterior instability is much more common and comprises 90%–95% of dislocations, and only a few cases of posterior and inferior instability in skeletally immature patients have been reported [39, 53, 58]. The mechanism of injury is generally the same as adult patients, although in young patients, a lower energy mechanism may cause a dislocation due to ligamentous laxity. Initial evaluation should follow the same principles as outlined above, and presentation of a dislocated GHJ will be similar to adult patients. In skeletally immature patients, a proximal humeral physeal fracture can occur rather than a true dislocation, and the displaced humeral metaphysis can mimic the clinical presentation of a GHJ dislocation [59]. Following initial evaluation, if possible, plain radiography should be obtained, followed by closed reduction (Fig. 14.23). In a very young patient, special consideration should be made for radiographic imaging prior to reduction, as a concomitant proximal humeral fracture may be present requiring an alternative reduction maneuver (Figs. 14.23 and 14.24). Imaging of the skeletally immature patient can be difficult to interpret, as the physes of the proximal humerus, glenoid, acromion, and coracoid may be misinterpreted as a fracture (Fig. 14.24). Knowledge of the order of appearance and fusion of physes in these areas is important for appropriate diagnosis. Contralateral images can be used to differentiate normal physes from a fracture. MRI or CT may be useful adjuncts if the diagnosis of an associated fracture is in question. Labral and capsular injury can also be evaluated with MRI, although in many cases of shoulder dislocation in patients younger than 13 years, no labral injury will be present due to ligamentous laxity. Initial treatment following closed reduction of an uncomplicated shoulder dislocation begins with 2–4 weeks of sling immobilization followed by physical therapy for restoration of range of motion and then strengthening. Following a successful reduction, outpatient referral to an orthopedic surgeon is appropriate within 1–2 weeks of the injury. Referral to an orthopedic surgeon is indicated in cases of recurrent instability in all ages. Recent studies have shown variable success rates with nonoperative treatment following anterior GHJ dislocation in patients younger than 18. Recurrence rates between 0% and 53% are reported for patients younger than 14 years of age, whereas recurrence of up to 60%–100% are noted in patients 14–17 years old [60–66]. Generally, nonoperative treatment is recommended for patients younger than 14 given the low probability of recurrence. In patients 14–17 years of age, an initial discussion regarding the high recurrence rate and possibility of early surgical stabilization is frequently undertaken.

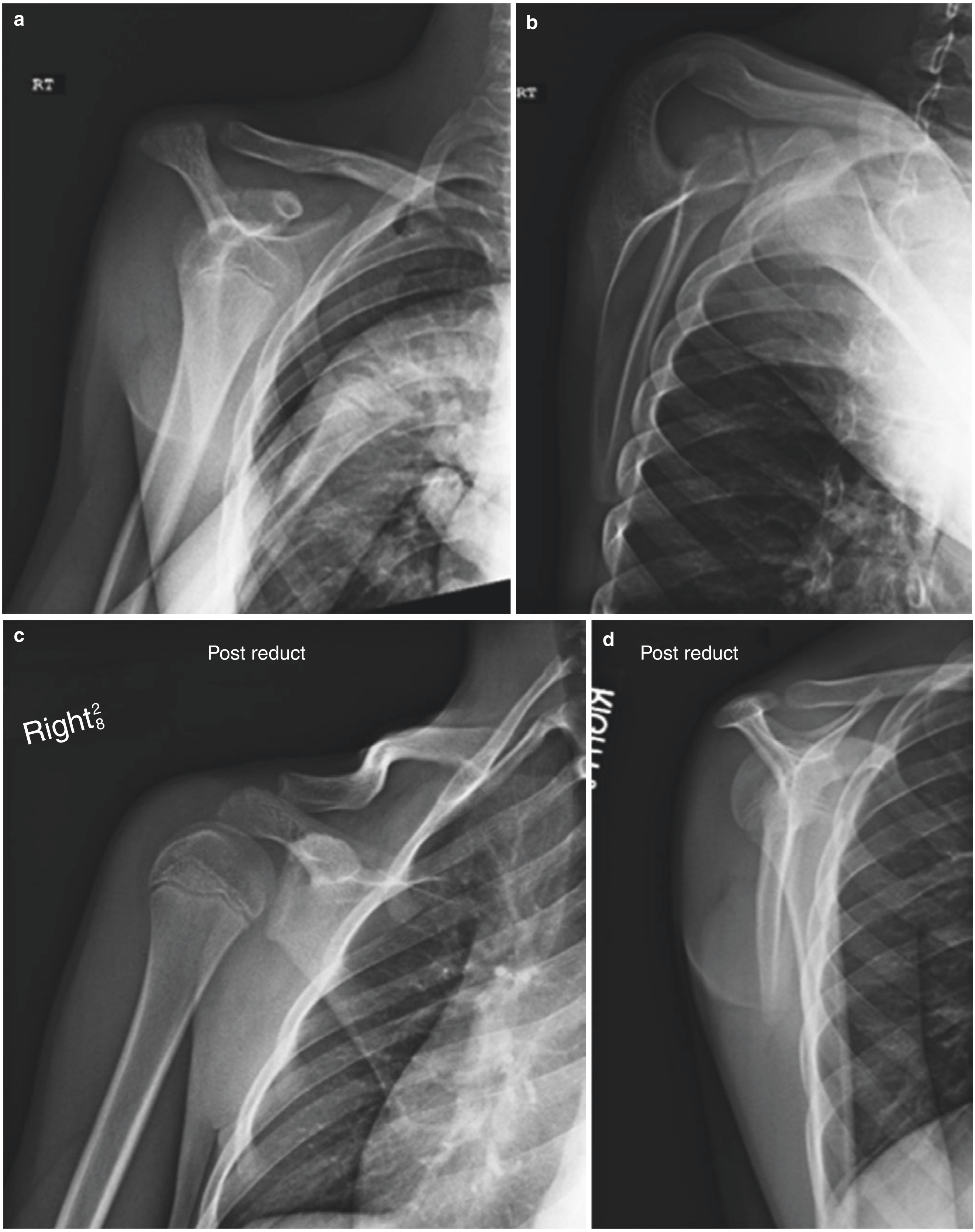

Fig. 14.23 Anterior GHJ dislocation in a 14-year-old boy as a result of a fall while skiing (**a**), (**b**). The post-reduction views (**c**), (**d**) confirm successful reduction with no associated fractures

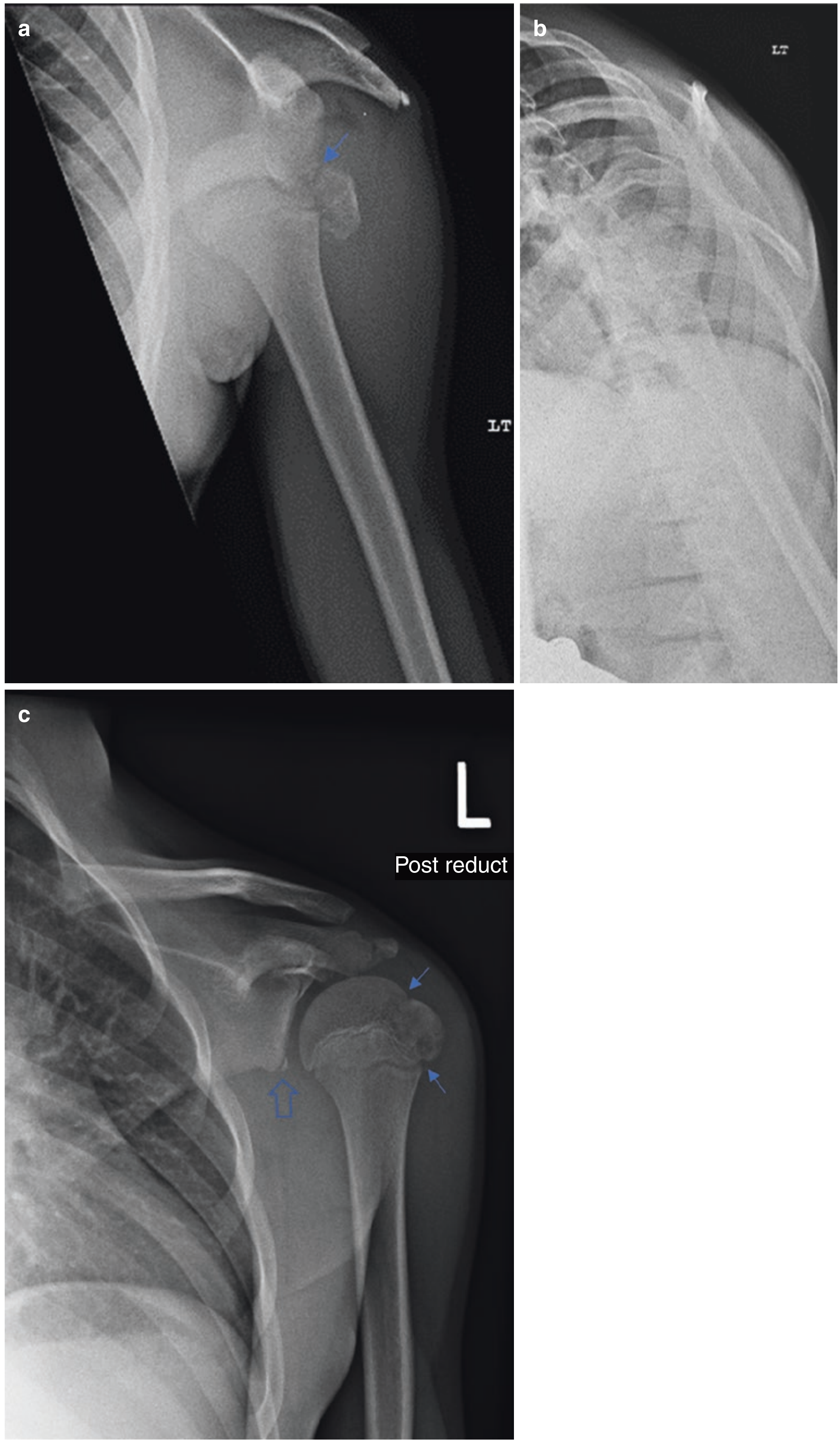

Fig. 14.24 Anterior GHJ dislocation with greater tuberosity fracture (arrow) in a 13-year-old boy as a result of a fall while snowboarding (**a**), (**b**). The post-reduction Grashey view (**c**) confirms the greater tuberosity fracture (arrows) and also reveals a possible glenoid apophysis (open arrow)

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Check for updates

Humerus

15

Christopher D. Joyce, David Ziegler, Katherine S. Dahab, and Jonathan T. Bravman

Key Points

- Proximal humerus fractures and humeral shaft fractures occur most frequently in younger patients with high-energy trauma or elderly patients with low energy mechanisms.
- Fractures of the humerus are typically diagnosed clinically with deformity, tenderness, and ecchymosis at the site of injury.
- Plain radiographs will be able to detect the majority of humerus fractures, although CT may be useful for intra-articular fractures.
- The majority of humerus fractures will heal without surgical intervention.
- Return to sport is dictated by clinical and radiographic evidence of fracture healing.

Introduction

Injuries to the upper arm in sports can range from minor contusions to limb-threatening fractures in severe instances. Soft tissue injuries about the humerus can include contusions, muscle strains, tendinitis or tendon ruptures, and joint instability. Specifically in the shoulder, rotator cuff injuries and glenohumeral and acromioclavicular instability injuries are common in sports and may result in acute and chronic dysfunction.

Fractures in the humerus are relatively rare in sports as a significant amount of energy is required to cause a humerus fracture in a young and healthy individual. Certain sports that are particularly prone to humerus fractures include skiing, snowboarding, and cycling. Additionally, elderly individuals are at an increased risk of humerus fractures as lower energy falls may result in fracture due to decreased bone strength. This chapter will primarily focus on the epidemiology, presentation, diagnosis, and treatment for fractures of the proximal humerus and humeral shaft. Management of proximal and mid-shaft humerus fracture is summarized in Table 15.1.

Relevant Anatomy

The proximal humerus osteology forms from three separate ossification centers: the articular epiphysis, the greater tuberosity, and the lesser tuberosity. The ossification centers fuse together in early childhood and subsequently fuse to the humeral metaphysis between age 20 and 23 years. They account for important bony structures in the proximal humerus and are important in understanding the subtle differences in the types of proximal humerus fractures.

The greater tuberosity serves as the insertion point for supraspinatus, infraspinatus, and teres minor muscles, while the subscapularis attaches to the lesser tuberosity. The junction between the articular humeral head and the tuberosities is the anatomic neck, while the junction between the tuberosities and the humeral shaft becomes the surgical neck in the adult humerus (Fig. 15.1). The long head of the biceps brachii muscle runs in the intertubercular sulcus between the greater and lesser tuberosities. The pectoralis major, latissimus dorsi, and teres major tendons also insert in the

C. D. Joyce
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: Christopher.Joyce@ucdenver.edu

D. Ziegler
Department of Orthopedics, Denver Health Medical center, Denver, CO, USA
e-mail: David.Ziegler@dhha.org

K. S. Dahab (✉)
Department of Orthopedics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO, USA
e-mail: Katherine.dahab@childrenscolorado.org

J. T. Bravman
Department of Orthopedics, Division of Sports Medicine and Shoulder Surgery, University of Colorado School of Medicine, Denver, CO, USA
e-mail: jonathan.bravman@ucdenver.edu

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Table 15.1 Management guidelines for humerus fractures

	Proximal		Mid-shaft	
Fracture category	adults	Pediatrics	adults	Pediatrics
Initial immobilization	Sling	Sling or hanging arm cast	Coaptation splint	Coaptation splint
Follow up	1 week	1 week	1 week	1 week
Long-term immobilization type	Sling, nothing	Sling, nothing	Sarmiento brace	Sarmiento brace, long arm cast
Length of immobilization	2–4 weeks	2–4 weeks	6 weeks	4 weeks
Indications for referral to orthopedics	Displaced fractures Fracture-dislocations Open fracture Nerve or vascular injury	Displaced fractures Intraarticular fractures Fracture-dislocations Open fracture Nerve or vascular injury	Displaced fractures Open fracture Nerve or vascular injury Ipsilateral forearm fracture	Displaced fractures Open fracture Nerve or vascular injury Psilateral forearm fracture
Healing time	6–12 weeks	6–12 weeks	6–12 weeks	6–12 weeks
Return to sports	>3 months	>2 months	>3 months	>2 months

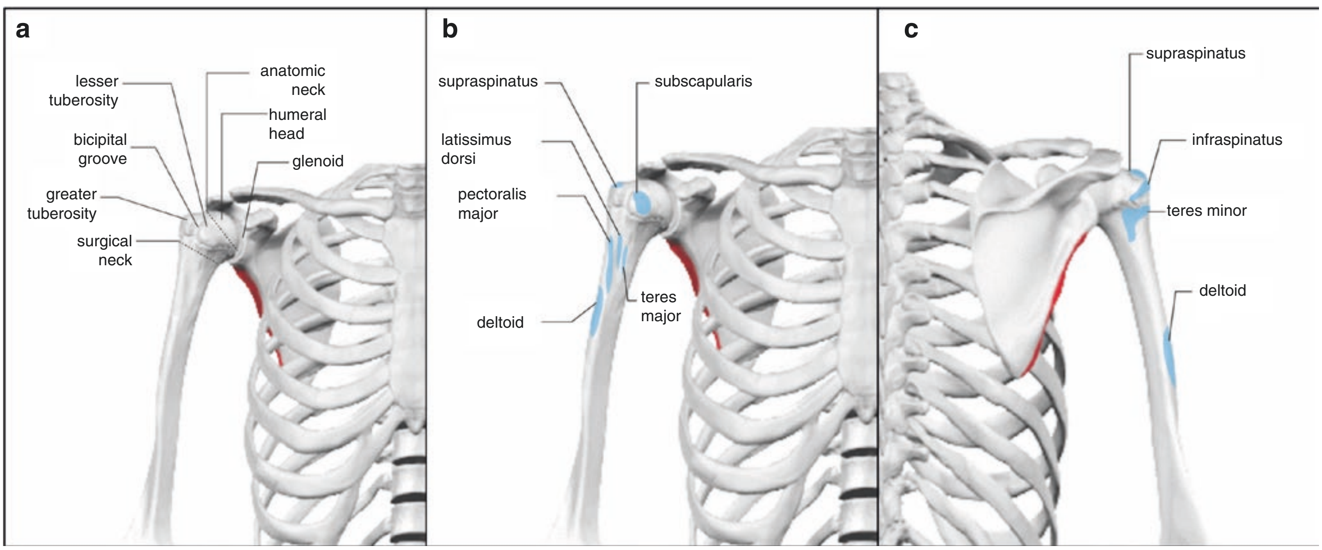

Fig. 15.1 Illustration highlighting the osteology (**a**) and pertinent tendinous insertions of the proximal humerus (**b**, **c**)

intertubercular sulcus in that order, respectively, from medial to lateral (Fig. 15.1).

The average humeral neck-shaft angle is 130°, and the humeral head is typically retroverted in comparison to the humeral shaft by 18°–30° [1]. The blood supply to the humeral head comes primarily from the anterior and posterior circumflex humeral arteries [2].

The mid-shaft of the humerus comprises an area from the surgical neck proximally to the supracondylar ridge near the elbow. The diaphysis of the humerus receives its blood supply from the axillary and brachial arteries. The radial nerve travels along the posterior mid-shaft in the spiral groove, then emerges anteriorly to cross the elbow. It supplies the innervation to the muscles of the posterior compartment of the arm. The deltoid attaches mid-diaphysis at the deltoid tubercle, while the pectoralis major attaches more proximally. The long head of the biceps brachii travels proximally in the bicipital grove.

Proximal Humerus Fractures

Mechanism of Injury in Sports

Proximal humerus fractures in younger patients are typically a result of high-energy trauma such as motor vehicle accidents or falls from height. An isolated proximal humerus fracture in a young and healthy patient is rare, obligating practitioners to perform a thorough examination of the involved extremity as well as the rest of the body. Conversely, proximal humerus fractures in the elderly population normally result after lower energy mechanisms such as a fall from standing. A proximal humerus fracture in an elderly individual is considered a fragility fracture, and an appropriate work up for osteoporosis should be performed [3].

In sports, proximal humerus fractures are rare overall. The most common injury mechanism is still from a fall onto an outstretched hand (FOOSH), but this is typically only seen in sports

with particularly high energy associated with the fall such as in skiing, snowboarding, and cycling. While overall snowboarders sustain more humerus fractures than skiers, skiers proportionally are more at risk for proximal humerus fractures, while snowboarders sustain more distal humerus fractures [4].

Epidemiology

Fractures of the proximal humerus are common injuries, making up roughly 5% of all fractures [5]. Proximal humerus fractures occur in a bimodal type distribution. There is a small peak of proximal humerus fractures in the pediatric and young adult population, typically seen in higher energy trauma. A much larger peak incidence is seen in the elderly population, especially over age 70. Proximal humerus fractures affect females two to three times more frequently than males, and the average age of a patient with a proximal humerus fracture is 71 [6]. A national survey of emergency room visits estimated 370,000 total humerus fractures in 2008, 184,300 of which involved the proximal humerus. This number is expected to grow to 275,000 proximal humerus fractures by 2030 due to a growing elderly population [7]. Proximal humerus fractures are the third most common fracture in elderly patients behind hip and distal radius fractures [8]. The overall incidence of proximal humerus fractures in sports is not well defined in the literature; however, in skiers roughly 14% of all shoulder injuries are proximal humerus fractures [4].

Fracture Calcification

Several different classification systems exist for proximal humerus fractures. The AO/OTA classification for proximal humerus fractures groups them into unifocal extra-articular, bifocal extra-articular, and articular (Fig. 15.2) [9, 10]. A more commonly used classification is the Neer classification of proximal humerus fractures. This classification system groups fractures by the number of displaced parts as defined by angulation greater than 45° or displacement greater than 1 cm or 0.5 cm for the greater tuberosity (Fig. 15.3) [11]. The Neer classification system has moderate inter-observer reliability but has been shown to have superior reliability compared to the AO classification [12]. Extra caution is given to greater tuberosity fractures as they are prone to further displacement and poor functional results if displaced greater than 0.5 cm [13]. Fractures may be through the anatomic neck, the surgical neck, the greater tuberosity, and/or the lesser tuberosity [11]. In fractures involving the tuberosities, pull from the supraspinatus, infraspinatus, and teres minor will tend to cause posterior and superior displacement of the

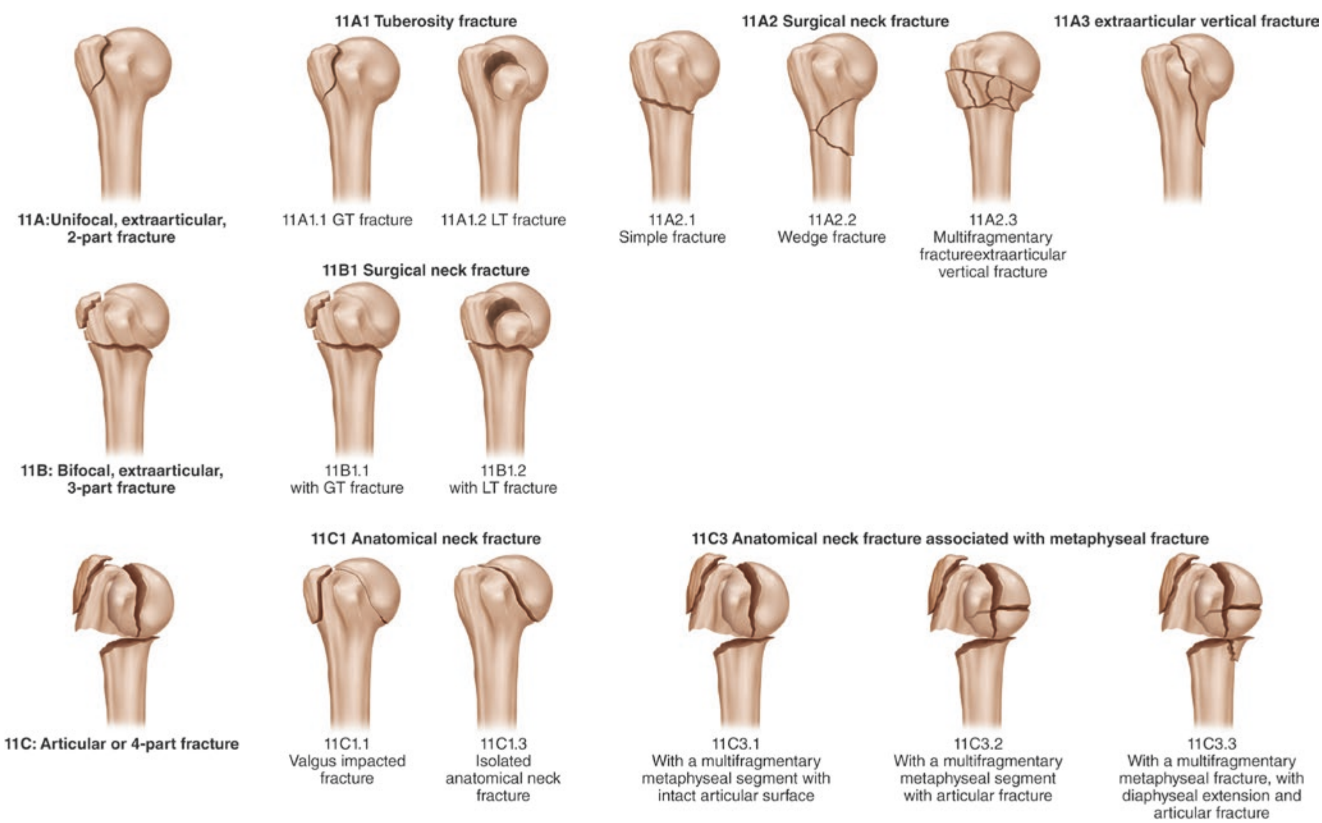

Fig. 15.2 Illustrative depiction of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for proximal humerus fractures [9, 10]

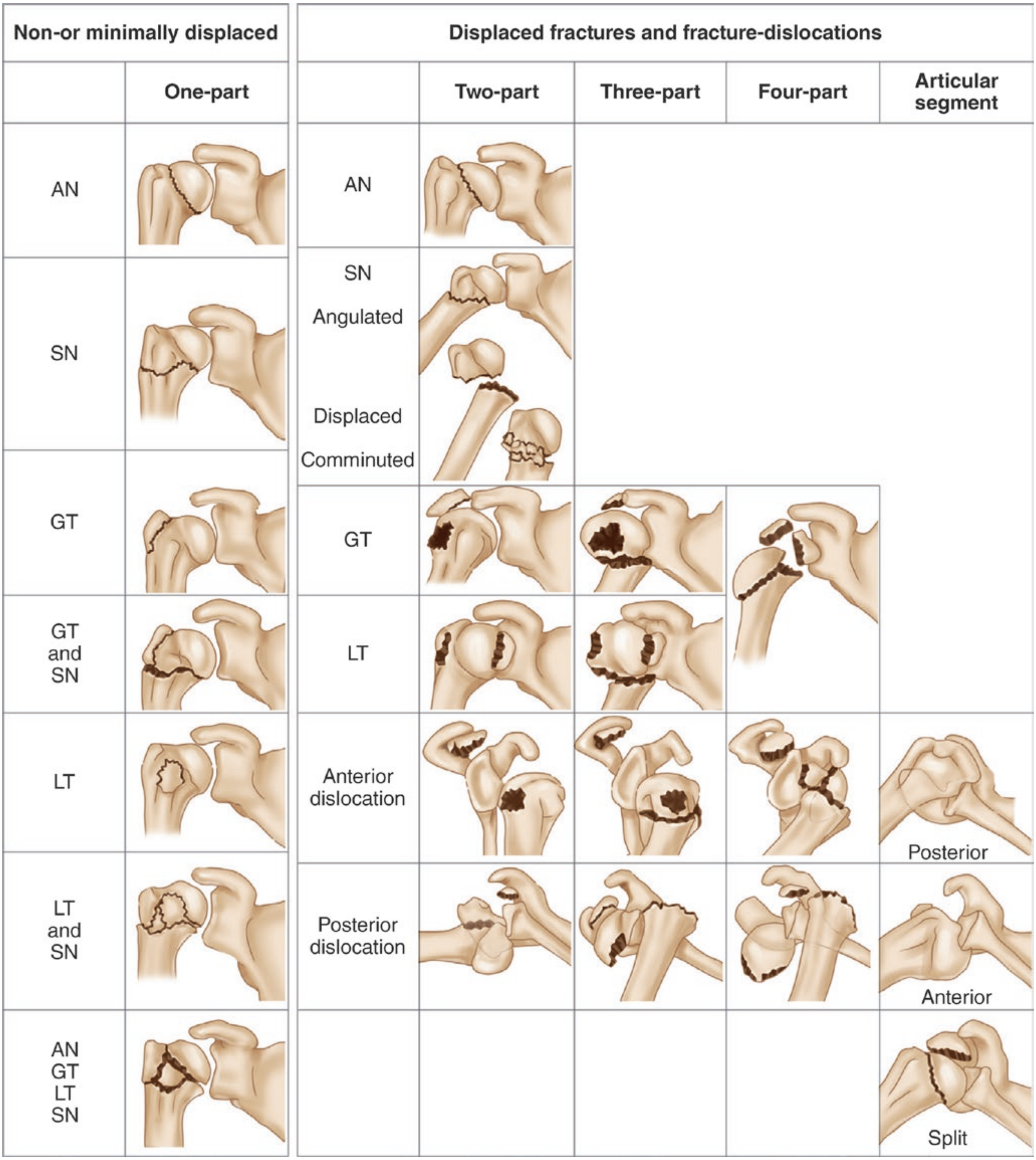

Fig. 15.3 Illustrative depiction of the Neer classification system for proximal humerus fractures [11]. AN, anatomical neck; SN, surgical neck; GT, greater tuberosity; LT, lower tuberosity

greater tuberosity fragment, while pull from the subscapularis will displace the lesser tuberosity fragment medially.

Fracture-dislocations of the proximal humerus must be treated with caution (Chap. 14). An attempted closed reduction of a GHJ dislocation with associated minimally displaced proximal humerus fracture may cause displacement of the fracture and necessitate surgical management. If the GHJ dislocation is unable to be reduced in a closed manner with gentle reduction maneuvers, urgent surgical management is recommended.

Clinical Presentation

On visual inspection of a patient with a proximal humerus fracture, one may note swelling and ecchymosis to the shoulder as well as in the distal extremity and chest due to settling of the hematoma. Some patients may present with an obvious deformity (Fig. 15.4), but most will not. A thorough neurovascular examination is crucial as proximal humerus fractures are associated with neurologic injury up to two third of the time [14]. The most commonly injured nerve is the axillary nerve followed by the suprascapular nerve [14]. The majority of neurologic injuries are transient and resolve in time.

Imaging

A specific series of plain radiographs is required for any patient in which a proximal humerus fracture is suspected. This includes an anteroposterior (AP) view of the GHJ (Grashey view), a scapular Y view, and an axillary view (Fig. 15.5). A true axillary view may be painful for patients, in which case a Velpeau modification is acceptable [15] (Fig. 15.6). Collectively these views allow for adequate visualization of the proximal humerus as well as the GHJ which is critical to assess for dislocation or subluxation of the GHJ. It is important to note that a patient may appear to have inferior subluxation of the humeral head which in fact is just a result of deltoid atony (Fig. 15.7). Additional views may include an AP view with the arm in internal or external rotation. In patients with a diagnosed proximal humerus fracture, full length anteroposterior and lateral radiographs of the humerus are also recommended.

Advanced cross-sectional imaging is not usually necessary in evaluation of proximal humerus fractures. CT is useful in evaluating shoulder fracture dislocations and associated glenoid fractures, and may also be helpful for better evaluation

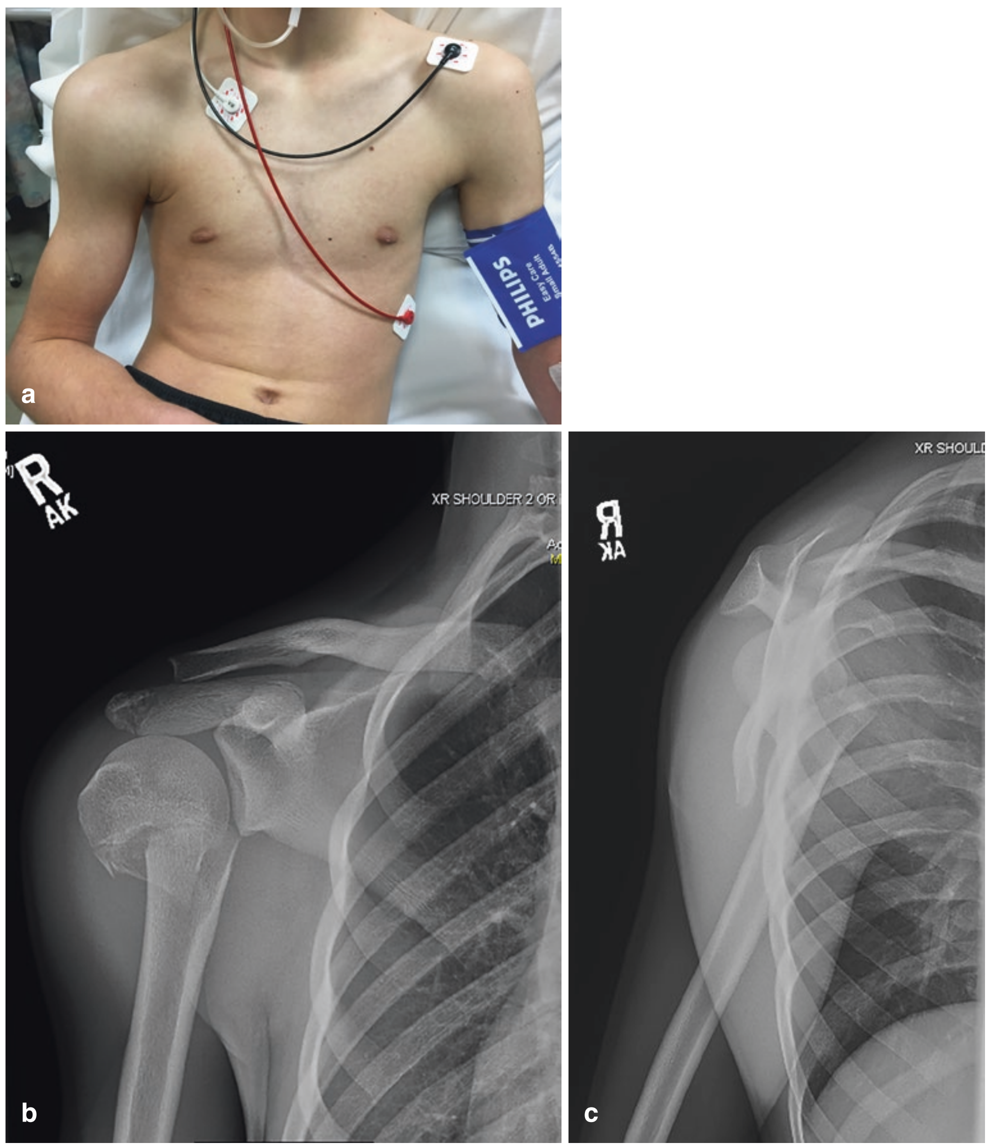

Fig. 15.4 A 15-year-old male who sustained a right Salter-Harris type II proximal humerus fracture after a fall while ski jumping. Clinical photograph demonstrates deformity (effusion due to hemarthrosis) (**a**) and associated AP (**b**) and scapular Y (**c**) view radiographs

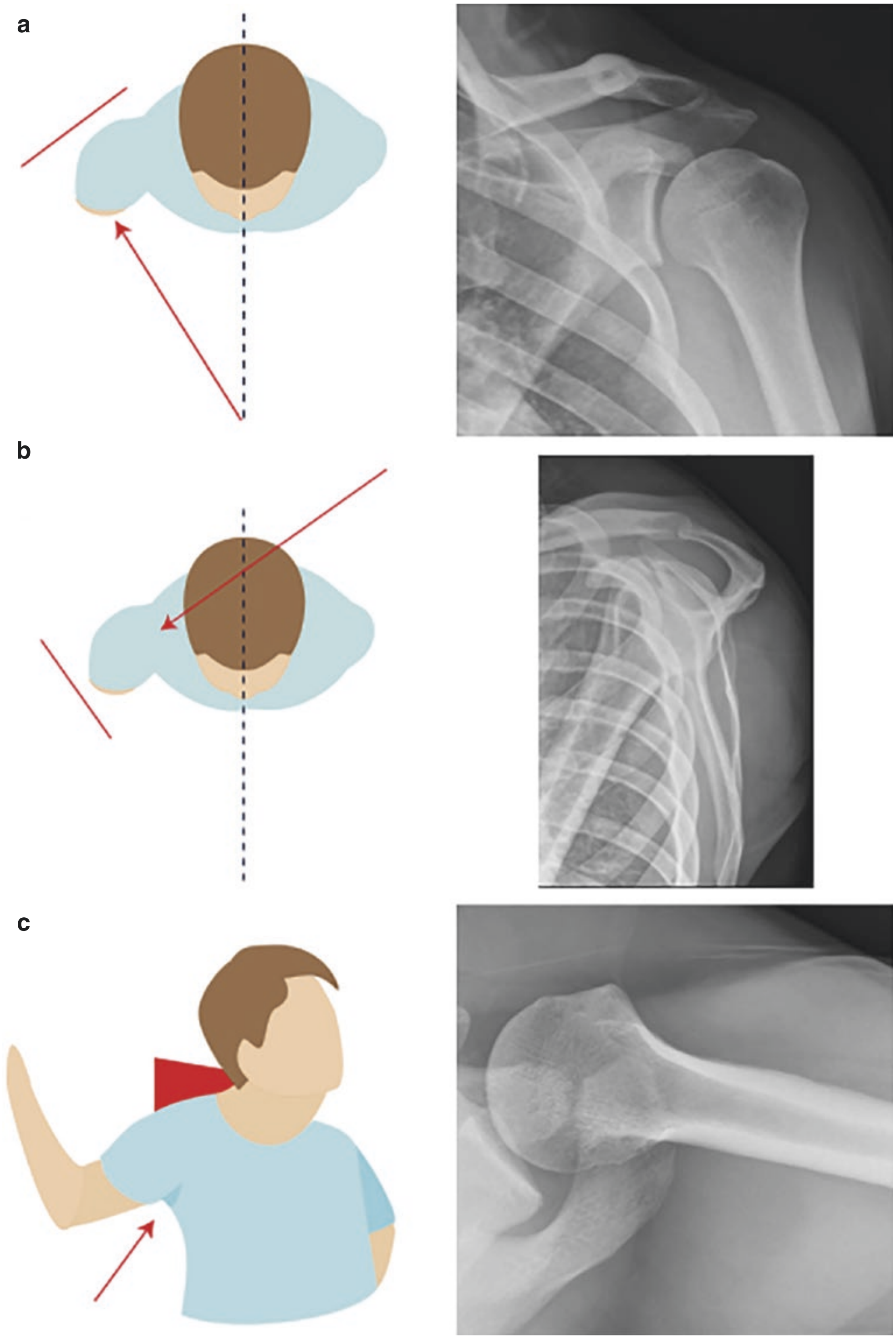

Fig. 15.5 Illustration and corresponding radiographs of the three standard shoulder radiographs for trauma (AP, Scapular Y, and axillary views)

of intra-articular fractures and fractures of the greater or lesser tuberosities (Fig. 15.8). CT scans in proximal humerus fractures have not been shown to improve fracture classification, but they may be helpful in pre-operative planning [16, 17]. MRI is rarely used in the setting of acute proximal humerus fractures, except in the setting of an occult fracture not visualized on plain radiographs or concern for concomitant rotator cuff tear.

Initial Management

When a player on the field is suspected to have a proximal humerus fracture, they must be immediately removed from play. Initial management should include a thorough neurologic and vascular examination. Any clothing must be removed so that the shoulder can be inspected for open wounds, ecchymoses, or deformity. The injured shoulder

Fig. 15.6 Photograph depicting proper positioning and technique for a Velpeau radiograph

Fig. 15.7 AP radiograph of a shoulder demonstrating inferior subluxation of the humeral head in the setting of a proximal humerus fracture due to deltoid atony

should be compared to the non-injured shoulder. Palpation of all bony aspects of the shoulder for tenderness or crepitus is important including the humerus, acromion, scapular spine, and clavicle. A range of motion examination should then be performed if the player is able to tolerate it. If suspicion for a proximal humerus fracture persists, the player must be removed for the remainder of the game and radiographs should be obtained urgently. In a clinical setting, the same principles apply for examination. The primary tool for diagnosing proximal humerus fractures is with plain radiographs; therefore, low threshold should exist for obtaining them urgently if a proximal humerus fracture is suspected.

A standard sling should be applied after evaluation to immobilize the shoulder to both protect the shoulder, and provide pain relief. This may be done prior to obtaining official radiographs. In the initial management phase, a standard sling is acceptable for immobilization. No type of splint or cast exists that can help immobilize a proximal humerus type of fracture.

Indications for Orthopedic Referral

There are several proximal humerus fracture patterns that should be referred to orthopedic surgery. The majority of proximal humerus fractures are treated nonsurgically; however, this decision is made on a case-by-case basis. Any displaced proximal humerus fractures warrant referral. We would also recommend referral for a younger patient with a nondisplaced greater tuberosity fracture as this fracture pattern is at high risk of displacement. Finally, patients with multiple injuries should be referred to orthopedic specialist as surgical fixation may allow for faster mobilization.

Surgical emergencies that must be referred to an orthopedic surgery provider or an emergency department immediately include open fractures, fractures with associated vascular compromise, and proximal humerus fracture-dislocations. These injuries must be addressed either emergently or at a minimum within 24 hours.

Follow-Up Care

As with many orthopedic injuries, a large variety of treatment options exist for proximal humerus fractures. These include nonsurgical treatments, percutaneous surgical treatments, operative fixation, and replacement options. Treatment strategy is determined by a variety of considerations including fracture location and displacement, concomitant injuries, and patient factors. A certain proximal

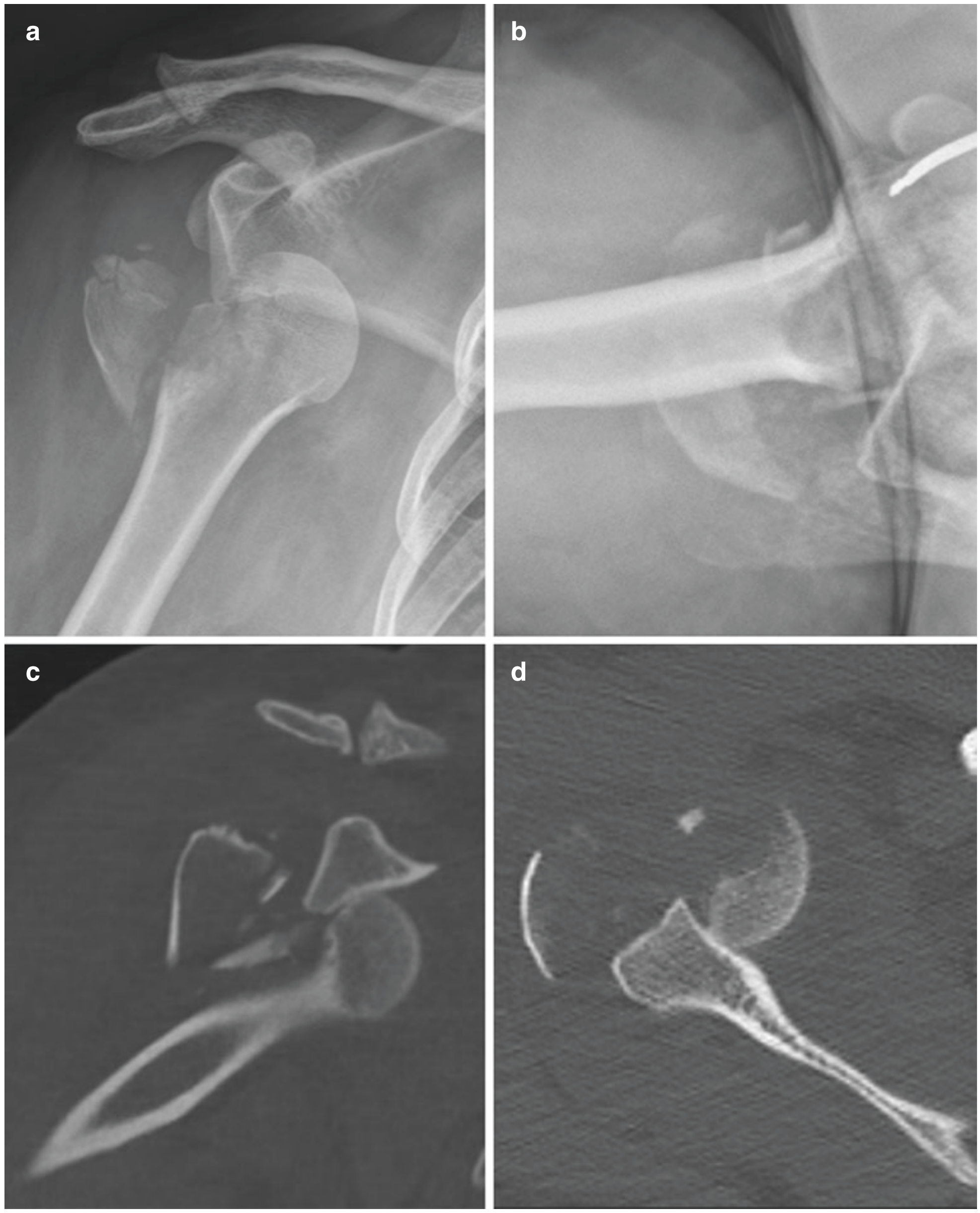

Fig. 15.8 AP and axillary plain radiographs (**a**), (**b**) of a proximal humeral fracture/dislocation with an associated greater tuberosity fracture. Coronal and axillary CT cuts (**c**), (**d**) demonstrate the degree of displacement of the fracture

humerus fracture in one patient may be treated different than a similar fracture in a different patient, and it is up to the treating practitioner to determine the best way to manage the patient as a whole.

Nonoperative Management

The majority of proximal humerus fractures are treated conservatively without surgery, with a recent survey of Medicare database estimated that 84% of all proximal humerus fractures are treated nonsurgically [18]. A significant amount of fracture displacement and angulation can be tolerated in proximal humerus fracture compared to fractures in other parts of the body. This is because the GHJ has more motion than any other joint in the human body, and malunions in this area will still allow for functionally acceptable range of motion.

Traditionally, one-part fractures based on the Neer classification are treated conservatively without surgical intervention. This includes fractures with less than 45° of angulation and 1 cm of displacement, except in greater tuberosity fracture where displacement must be less than 0.5 cm [11]. Other factors to consider are patient age, overall health status, coinciding injuries, and hand dominance. Recent studies have challenged this standard, however, showing that nonoperative management may have a role in displaced proximal humerus fractures as well [19]. The Proximal Fracture of the Humerus Evaluation by Randomization (PROHFER) trial was a multi-center randomized controlled trial that compared non-

operative to operative management in displaced surgical neck proximal humerus fractures [19]. This study found no significant difference in mortality rates, functional outcomes, or quality of life scores 2 and 5 years after the injury [19, 20]. A similar conclusion was drawn from a recent meta-analysis [21]. While this evidence cannot be generalized for all patients, it does suggest that conservative management may be acceptable in displaced surgical neck fractures. When specifically looking at younger patients with sport-related injuries, surgical fixation may be indicated more often as it allows athletes to mobilize the extremity more quickly. Additionally, athletes with high-energy mechanism injuries tend to have a higher incidence of greater tuberosity fractures which may trend towards surgical fixation.

A clear algorithm for nonsurgical treatment of proximal humerus fractures does not currently exist. Most providers immobilize patients for a short period of time, followed by a period of progressive increased motion under the guidance of physical therapy. Immobilization is typically in a standard commercially available arm sling. While this is a cost-effective option, some providers do advocate for a neutral rotation shoulder immobilizer to hold the proximal humerus in a more anatomic position during the healing process. This is particularly important in greater tuberosity fractures, as the greater tuberosity fragment is being displaced in a posterior and superior position, so holding the arm in a relatively internally rotated position may displace this fragment further (Fig. 15.9). Historically, patients have also been immobilized in a hanging arm cast or cuff and collar sling (Fig. 15.10), but these are frequently not well tolerated by patients and lead to worse long term outcomes compared to a regular sling [22]. However, there is no good evidence to support these additional slings.

While proximal humerus fractures were traditionally treated with prolonged immobilization, several studies have demonstrated that early mobilization results in improved functional and pain scores especially within the first 3 months post-injury [21, 23, 24]. These studies found that immediate mobilization within several days after the injury with a physical therapist was superior to immobilization for 3 weeks followed by therapy [21, 23, 24]. Most physical therapy protocols first entail a 2–4 weeks of directed passive range of motion and pendulum exercises. This followed by progressive active range of motion exercises for several weeks, and then strengthening exercises at a minimum of 6 weeks post-injury [23–27].

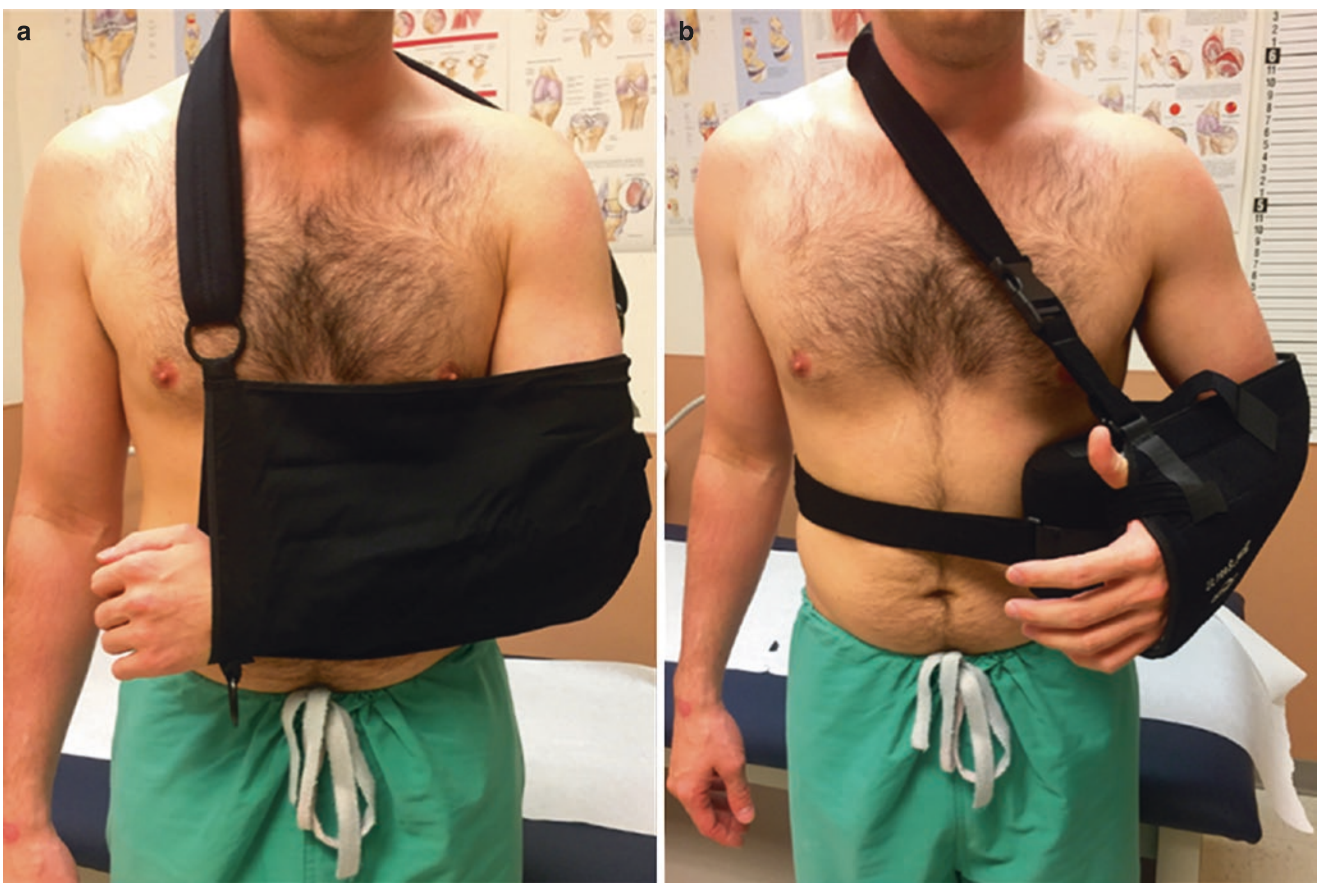

Fig. 15.9 An example of a standard sling (**a**), and a neutral rotation shoulder immobilizer (**b**)

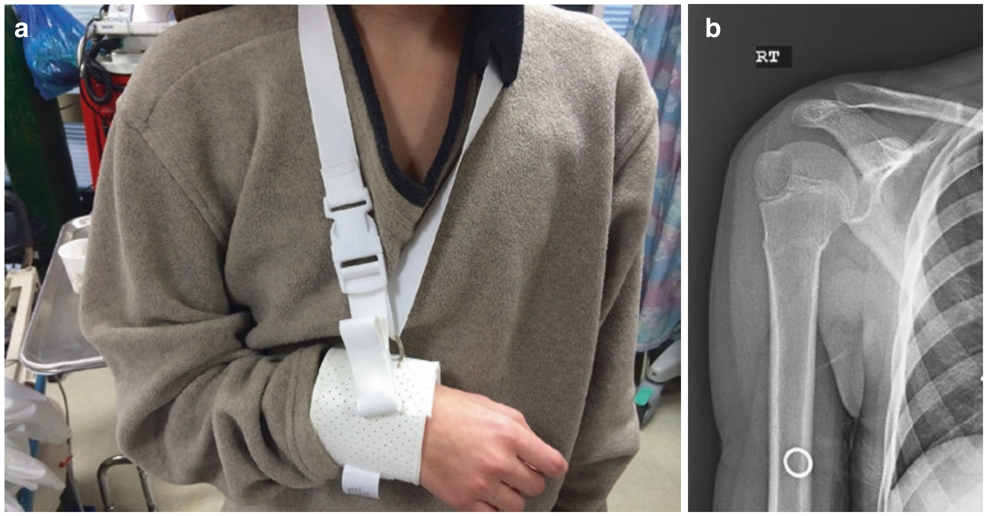

Fig. 15.10 A cuff and collar (**a**) immobilization in a 12-year-old female who sustained an extra-physeal proximal humerus fracture (**b**) during a snowboarding accident

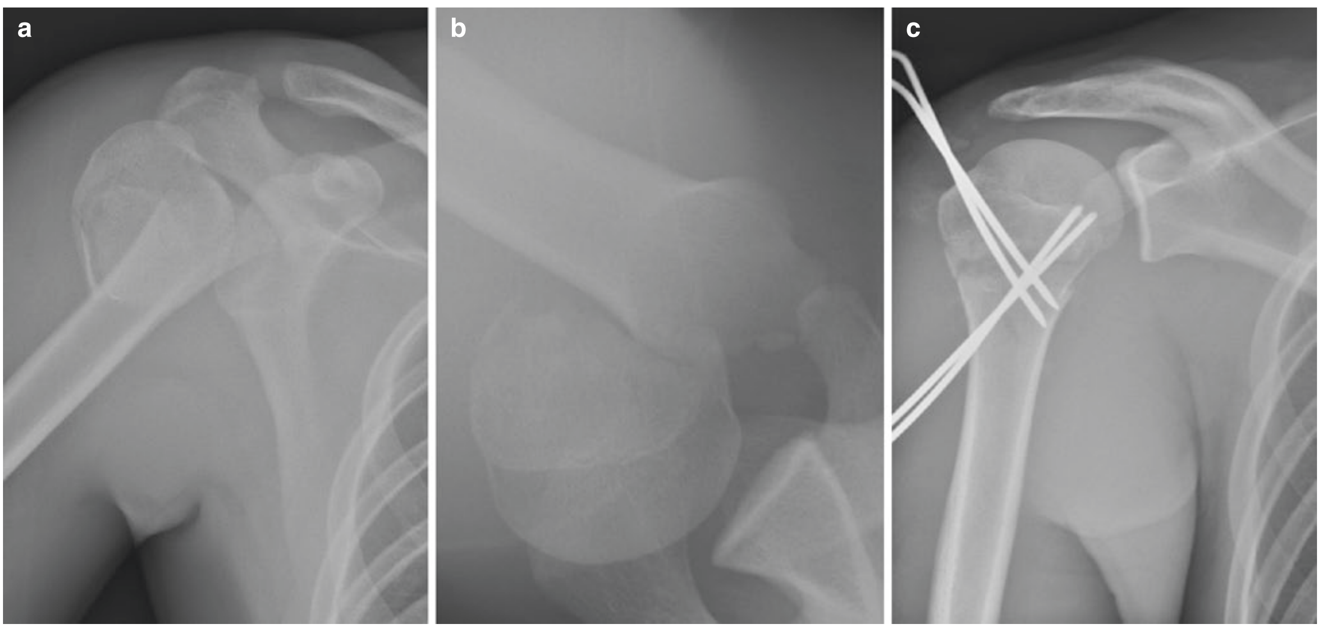

Fig. 15.11 Preoperative (**a**), (**b**) and postoperative (**c**) radiographs after closed reduction and percutaneous pinning of an unstable, completely displaced proximal humerus fracture in a 16-year-old male

When treating proximal humerus fractures nonsurgically, it is recommended to obtain radiographs at the following intervals: 1 week, 2 weeks, 6 weeks, 3 months, 6 months, and 1 year post-injury. If the injury is at particular high risk of displacement, then an additional radiograph 3 weeks after the injury may be indicated as well. We recommend clinical judgment for individualized follow up care and repeating radiography.

Operative Management

The two broad categories of surgical management are reduction with internal fixation and shoulder replacement. The methods of reduction with internal fixation may include closed reduction and percutaneous pinning (Fig. 15.11), intramedullary nailing, or open reduction and internal fixation with a proximal humerus locking

plate (Fig. 15.12). Shoulder replacement type procedures are reserved for lower demand patients, or individuals with severely comminuted fractures (Fig. 15.13). With younger patients and sport-related injuries, the vast majority of cases are going to be with closed reduction and percutaneous pinning or open reduction and internal fixation as shoulder replacement surgeries are reserved for salvage cases or low-demand patients.

Return to Sports

Return to sport after operative or nonoperative management is similar. Patients must be clinically and radiographically healed prior to be released for play without restrictions. This means minimal tenderness at the fracture site, full and painless shoulder range of motion, and radiographic evidence of bony callus healing. This will typically be at minimum 3 months post-injury.

Complications

Short-term complications from proximal humerus fractures are typically related to any nerve or vascular injuries sustained. In closed injuries, most neurologic injuries do improve as they are from a reversible neuropraxic type event. Long-term complications from proximal humerus fractures include fracture nonunion or malunions, muscle weakness, and shoulder stiffness. Weakness and stiffness can typically be addressed with aggressive physical therapy. In patients undergoing surgical intervention, other risks include iatrogenic nerve, vascular, or muscle injury, infection, bleeding, and hardware failure.

Pediatric Proximal Humerus Fractures

Mechanism of Injury in Sports

Proximal humerus fractures in children usually result from a backward fall onto an outstretched arm or a direct fall onto the shoulder [28]. Fractures may also occur from direct trauma to the arm, non-accidental trauma or a pathologic fracture.

Epidemiology

Proximal humerus fractures in the pediatric population are relatively uncommon and account for less than 5% of all pediatric fractures. They are most often seen in adolescents and the most common type is a Salter-Harris type II fracture of the proximal humerus (Fig. 15.4) [29]. In younger children, fractures are more often seen in the metaphysis, which is rapidly growing and is thought to be relatively weak during rapid growth, making it more prone to injury.

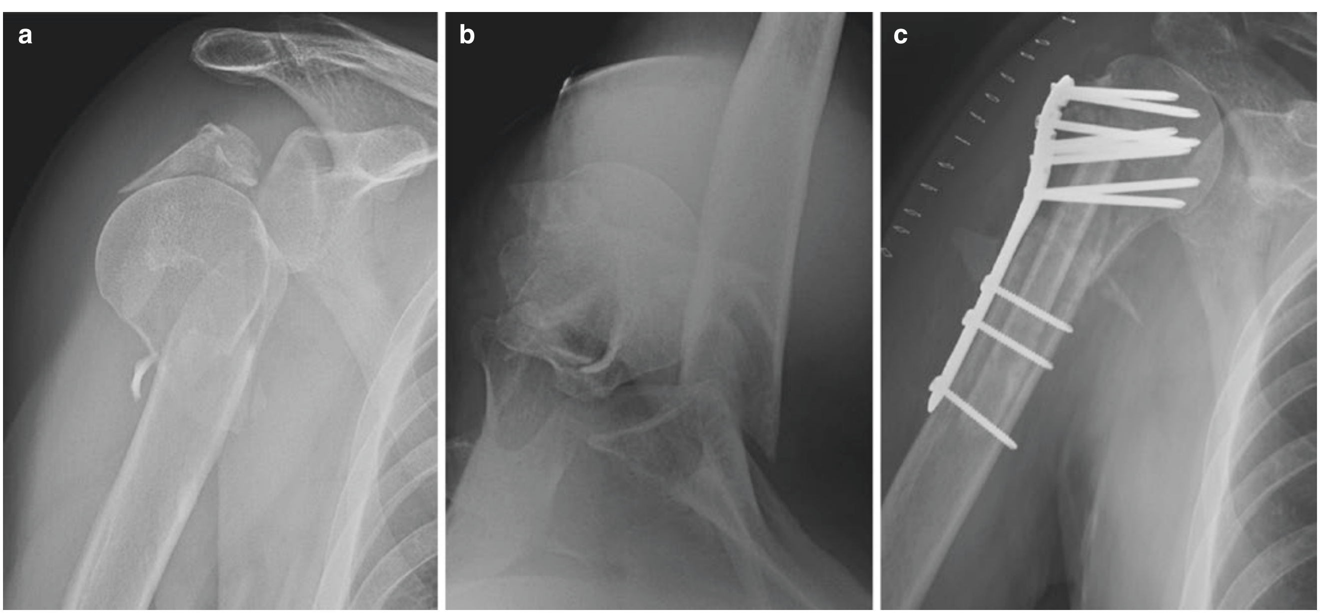

Fig. 15.12 Preoperative (**a**), (**b**) and postoperative (**c**) radiographs of a patient with a proximal humerus fracture undergoing open reduction and internal fixation with a proximal humerus plate

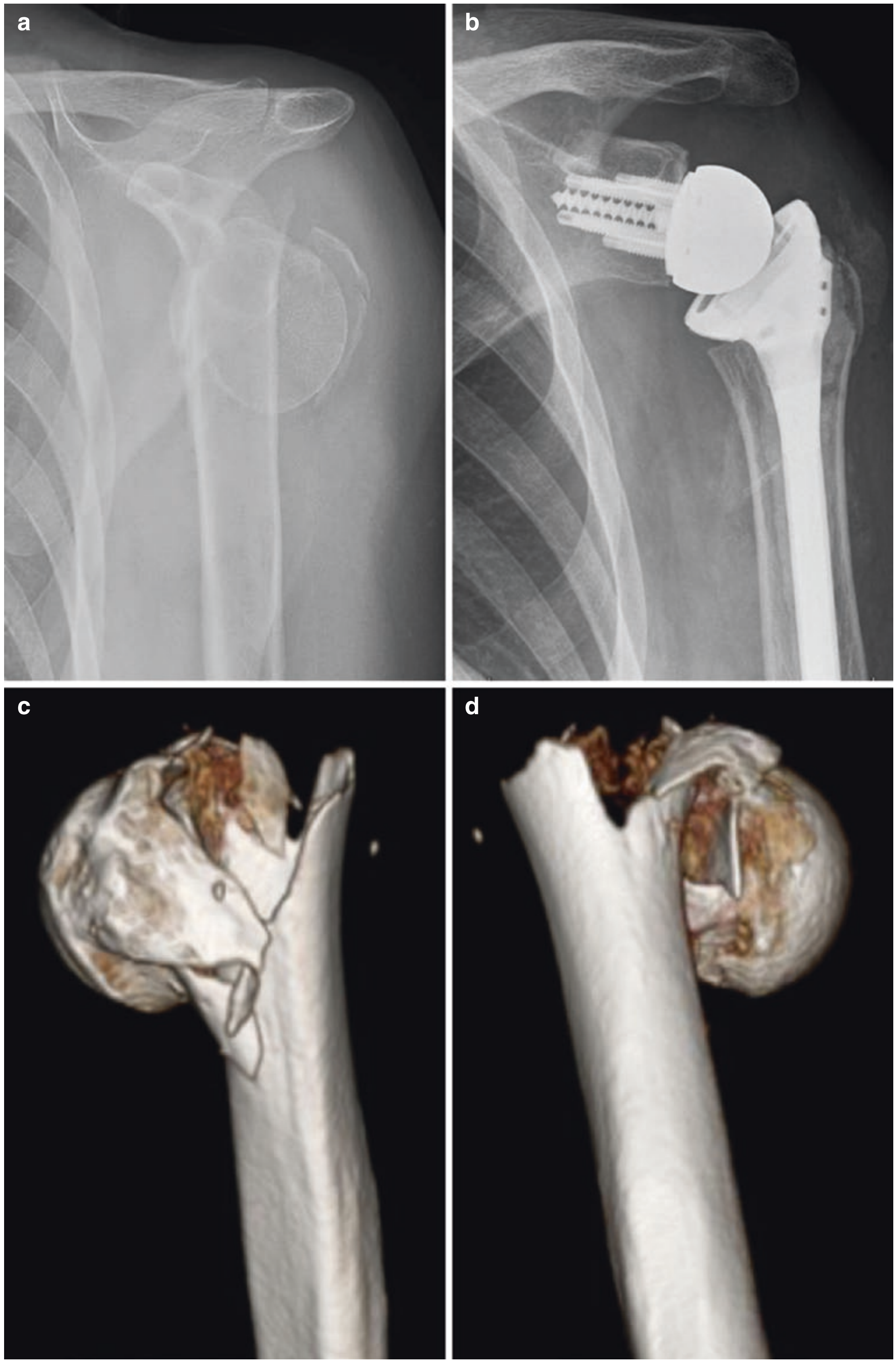

Fig. 15.13 Radiographs of a patient with a proximal humerus fracture (**a**) that was treated with a reverse shoulder arthroplasty (**b**). 3D CT demonstrates the extend of the fracture (**c**), (**d**)

Fracture Classifications

Specifically for pediatric proximal humerus fractures, the Neer-Horowitz Classification system groups fractures into nondisplaced (Type I), displaced less than 1/3 of shaft width (Type II), displaced 1/3 to 2/3 of shaft width (Type III), and displaced greater than 2/3 of shaft width (Type IV). Additionally, the Salter-Harris classification for physeal injuries may be applicable.

Clinical Presentation

The injured child may present with swelling, pain, and decreased motion of the injured arm. Diagnosis is made with plain radiographs.

Management

It is important to assess the neurovascular status of the arm distal to the injury and for any evidence of deformity of the arm. The injured arm should be placed in a sling until further radiographic assessment can be performed.

Most fractures heal well with sling immobilization. In children, healing is more rapid compared to adults and many children will have visible callus on imaging 2–3 weeks after the injury. Children are able to heal and remodel fractures with moderate displacement and angulation without the need for surgery [28].

Return to sports can be considered when the young athlete has no pain and tenderness on exam, full range of motion of the arm at the shoulder and elbow and evidence of healing on X-ray.

Indications for Orthopedic Referral

Emergent referral is necessary with any sign of neurovascular compromise, open fracture, or gross deformity. Proximal humerus fractures that require reduction changes dependent on patient age. If the patient is younger than 10 years, any angulation is typically acceptable as they will remodel significantly. Ages 10–13 years can tolerate up to 60° of angulation, while children over 13 years old can tolerate 45° of angulation [28]. Fractures outside of this range should be referred to an orthopedic surgeon as well as and displaced intra-articular fractures.

Complications

Complications after a humerus fracture in pediatrics are rare. If there is persistent angulation of the fracture after healing, arm function is still usually very good. If the fracture involved the growth plate, there could be premature closure of the injured physis. This could cause relative shortening of the injured arm compared to the contralateral side [28]. However, this may happen with Salter-Harris V.

Humeral Shaft Fractures

Mechanism of Injury in Sports

The mechanism of injury in humeral shaft fractures is similar to proximal humerus fractures as noted above. There is a relative trend toward humeral shaft fractures being associated with higher energy mechanisms that proximal humerus fractures as diaphyseal bone is stronger than metaphyseal bone. Stress fractures, although uncommon in the humerus, may occur with upper extremity dominant sports such as tennis, baseball, or swimming [30].

Epidemiology

Mid-shaft humerus fractures are less common than other types making up approximately 2% of all fractures. In children, they comprise fewer than 3% of all fracture types [31]. There is a bimodal distribution with peak incidences occurring in the third and seventh decades [32]. Trauma in males accounts for the first peak, while simple mechanical falls in females accounts for the second, representing changes in aging bone. Sports-related trauma accounted for 4.6% of all mid-shaft fractures with the predominant type being AO type A.

Fracture Classification

The AO/OTA classification system is often used to characterize humerus shaft fractures (Fig. 15.14) [9, 10]. The classification system is based on location of the fracture (proximal, mid-shaft, and distal), as well as the fracture pattern (simple patterns, wedge patterns, and comminuted patterns).

Fractures that occur at the midpoint of the diaphysis, below the attachment of the deltoid, tend to have an apex lateral angulation. This occurs as the deltoid pulls the proximal fragment laterally and the distal fragment moves medially. Fractures occurring above the deltoid attachment have a proximal segment that is pulled medially by the pectoralis muscle. The distal segment is pulled laterally by the deltoid. Mid-shaft fractures are often shortened as a result of muscle contraction by the deltoid, triceps, and biceps muscles.

Clinical Presentation

Athletes present with pain, swelling, and usually an obvious deformity to the humerus (Fig. 15.15). The fractured segment

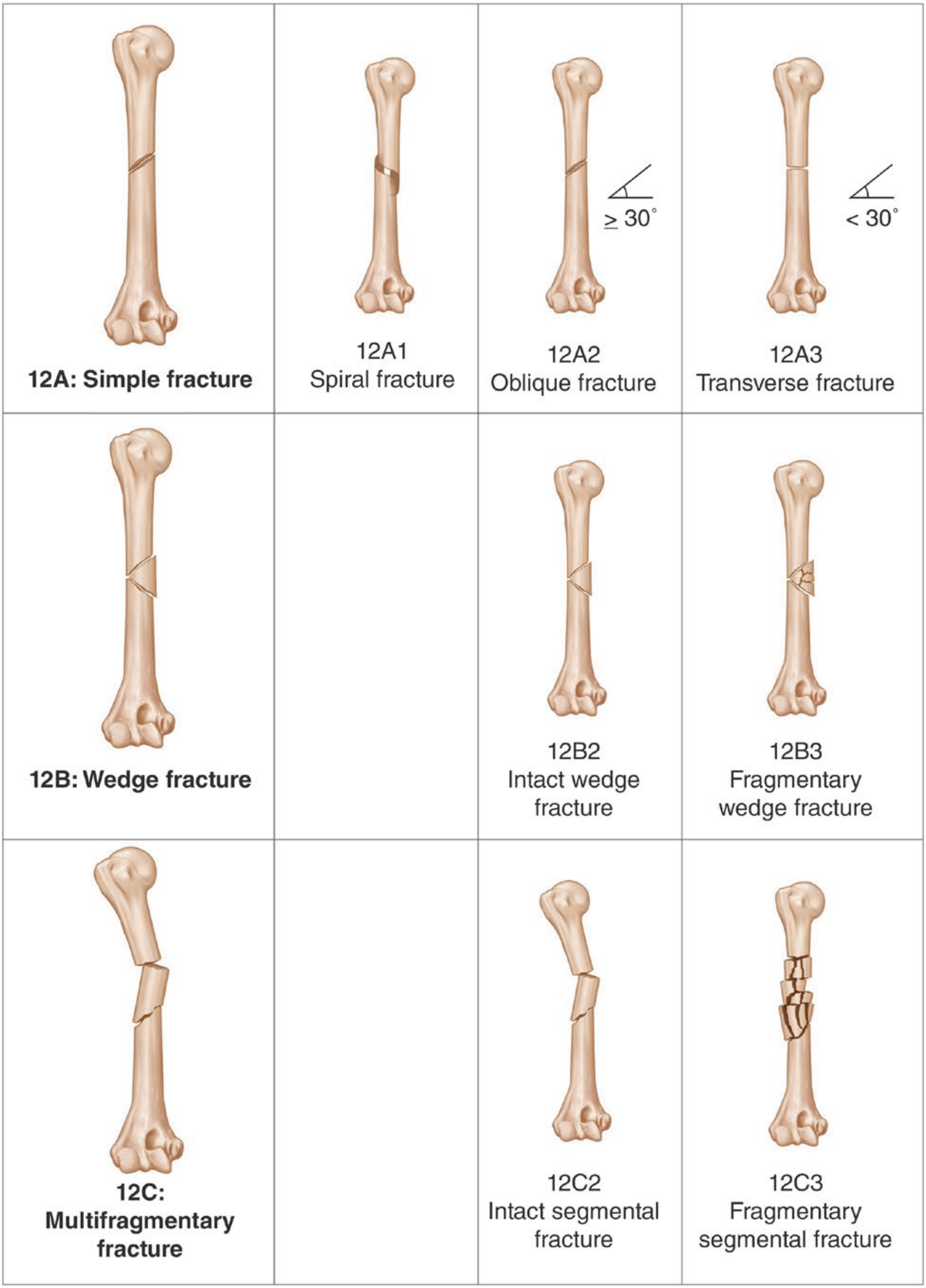

Fig. 15.14 Illustrative depiction of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification for humeral diaphyseal segment fractures [9, 10]

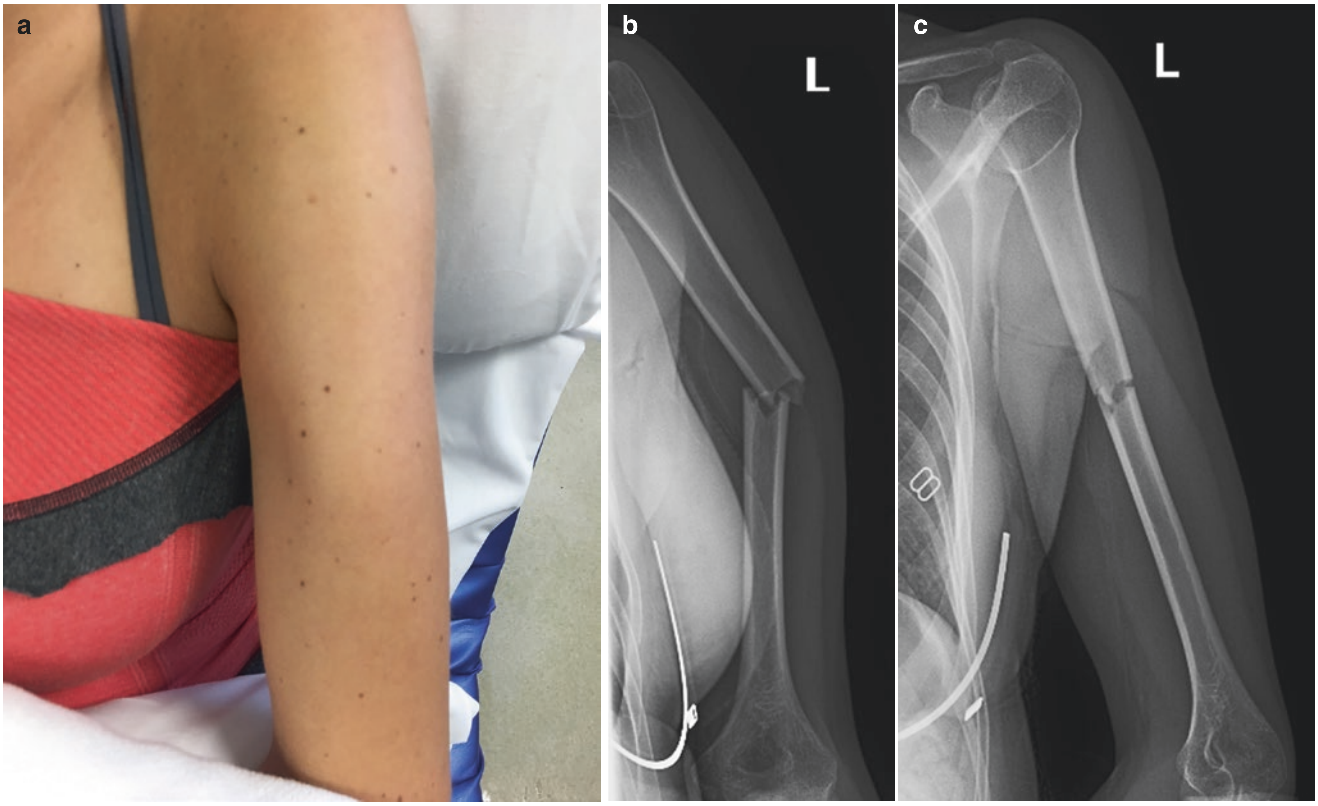

Fig. 15.15 Clinical (**a**) and radiograph (**b**, **c**) images of a 19 year-old female who sustained a humeral shaft fracture with deformity after a fall while skiing

will often be very mobile with crepitus upon palpation. Open fractures occur in less than 10% of cases but necessitate the need for emergent surgical consultation, antibiotics, and a proper dressing [33]. A thorough neurologic examination is essential. Damage to the radial nerve near the fracture site may cause wrist drop as the nerve innervates the common extensor musculature. Sensory examination may reveal decreased sensation in the first web space of the hand. Vascular injury is possible which may decrease the radial pulse at the wrist.

Imaging

Plain radiographs are essential for fracture characterization. Two orthogonal views are necessary and usually sufficient to fully describe the fracture (Fig. 15.16). Spiral and oblique fractures can sometimes be difficult to diagnose. The shoulder and elbow joints should be included in the views to access for extension of any injury [34].

CT scans and MRIs are usually not necessary unless a nerve or arterial injury is suspected.

Initial Management

The initial management on the playing field or in the office is the same as with proximal humerus fractures. Initial management includes relative immobilization with a sling and swathe, cuff and collar, or co-optation splint with sling (Fig. 15.17). Following initial management, the use of a functional brace (Fig. 15.18) has been shown to provide adequate immobilization with low rates of nonunion [35].

Indications for Orthopedic Referral

Emergent referral to an orthopedic surgeon is necessary for open fractures, fractures with associated neurologic or vascular injury, or fractures associated with compartment syndrome. These injuries should be sent directly to an emergency department for further evaluation for surgical intervention.

Nonurgent humeral shaft fractures that require referral to orthopedic surgery include fractures in unacceptable alignment, patients with multiple injuries that could benefit from earlier mobilization, ipsilateral forearm fractures (floating

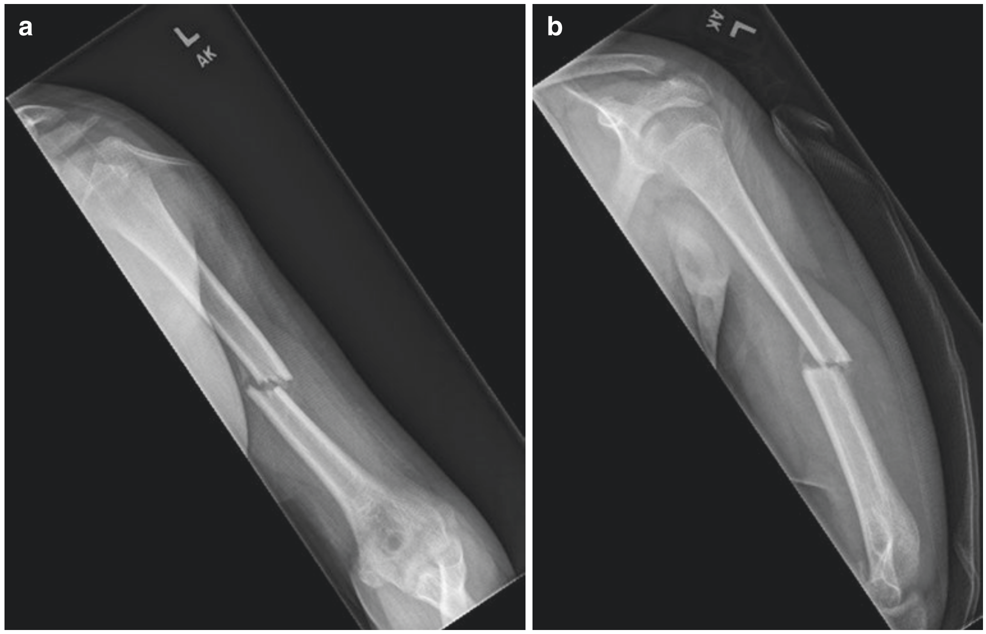

Fig. 15.16 Orthogonal AP (**a**) and lateral (**b**) radiographs of a 13-year-old male who sustained a humeral shaft fracture after a mountain biking accident

Fig. 15.17 Example of a patient being treated with a coaptation splint (**a**), (**b**)

elbow), pathologic fractures, and vascular injury. Radial nerve injuries may not require surgical exploration as most recover over time but an injury should involve consultation with a surgeon [36].

Follow-Up Care

The same as proximal humerus fractures.

Nonoperative

The vast majority of humeral shaft fractures are treated nonoperatively. This is in part due to the extensive range of motion of the shoulder joint, allowing for a large amount of fracture deformity. Acceptable fracture alignment includes 20° of anterior bowing, 30° of varus angulation, 15° of malrotation, and 3 cm of shortening or bayonet apposition [37].

Fig. 15.18 Examples of functional humerus braces (**a**), (**b**)

Follow up is weekly to every other week with repeat radiographs to ensure healing and adequate alignment. The typical healing time is 8–12 weeks [38]. Nonoperative intervention in some studies has shown a higher nonunion rate compared to compression plate fixation, while the rate of transient radial nerve injury was higher in the operative group. Normal range of motion was achieved in both groups [38].

Operative

Operative intervention includes the use of compression plating, rigid intramedullary nails, semi-rigid intramedullary nails.

Return to Sports

There are few evidence-based guidelines for return to play after humerus fractures. Decisions are based on long bone healing physiology, clinical and radiographic healing, and demands of the sport involved. Contact sports may require longer recovery periods than non-contact sports. Gentle range of motion exercises such as pendulums, while in a sling, can be started after 1 week. When there is adequate radiographic and clinical healing, therapy can be started for shoulder and upper extremity strengthening with sport-specific exercises. After the athlete has achieved full shoulder and extremity strength and range of motion, game play can resume. A longer recovery may be needed for sports that apply stress to the humerus including throwing and racket sports and contact sports such as hockey and football.

Complications

Acute complications include neurologic or vascular injuries. Radial nerve palsy can be a significant complication with prevalence approaching 12% of mid-shaft fractures. Transverse and spiral fracture patterns are more commonly associated with palsy versus comminuted and oblique fractures [34].

A long-term complications include nonunion. However, humeral shaft fractures have union rates of 90%–95% with nonoperative and operative treatment [38]. Additionally, with surgical intervention risk of infection, hardware failure, and iatrogenic nerve injury exists as well.

Pediatric Humeral Shaft Fractures

Mechanism of Injury in Sports

The mechanisms of humerus shaft fractures in pediatrics is the same as with adult athletes.

Epidemiology

Less than 3% of fractures in children are mid-shaft humerus fractures. A spiral type fracture is the most common type of mid-shaft humerus fracture in children [39].

Fracture Classification

A specific pediatric humeral shaft fracture classification system is not widely utilized.

Clinical Presentation

In children under the age of 18 months who present with a spiral fracture of the humerus, non-accidental trauma is the most common cause and a thorough investigation is warranted [40]. Children over age of 18 months often sustain fractures from direct trauma to the arm or falling onto a hard object. Non-accidental trauma can still be a cause in children over age 18 months as well, though less likely [41]. In addition, the mid-shaft of the humerus can be common site for pathologic fractures in a child [42]. The injured child may present with swelling, pain. and decreased motion of the injured arm. Diagnosis is made with plain radiographs.

Fig. 15.19 Example of a patient being treated with a hanging arm cast

Management

Assess for neurovascular status of the arm distal to the injury and for any evidence of deformity of the arm. The injured arm should be placed in a sling until further assessment can be performed.

Most fractures heal well with sling immobilization or a hanging cast (Fig. 15.19). The mid-shaft of the humerus has less remodeling potential than the proximal humerus, but most fractures still have an excellent prognosis to heal nonoperatively. Children under age 12 with minimal displacement or angulation can be treated with a sling and swath. Adolescent should be treated as adults for mid-shaft humerus fractures. Healing in children typically will take 4–6 weeks [39].

Indications for Orthopedic Referral

Emergent referral is indicated with any sign of neurovascular compromise, open fracture, or gross deformity or for concern of non-accidental trauma to the child. Urgent referral is also necessary with any concern for pathologic fracture, if the treating physician is not comfortable with the fracture or if the treating provider is unsure if the fracture needs further reduction.

Return to Sports

Return to sports can be considered when the young athlete has no pain on exam, full range of motion of the arm at the shoulder and elbow, and evidence of complete healing on plain radiographs.

Complications

Complications are not very common for mid-shaft humerus fractures in children. Radial nerve injury can occur, but this is less frequent in children compared to adults. Some mild angulation, shortening or overgrowth of the humerus may occur after the injury heals, but this rarely interferes with arm function [39].

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Elbow Joint

16

Jack Spittler and Adam Seidl

Key Points
- Elbow dislocations are most commonly posterolateral and can be associated with radial head and coronoid fractures ("terrible triad").
- Nondisplaced elbow fractures may difficult to appreciate on radiographs, but attention should be paid to the lateral radiograph for the presence of a "fat pad" sign (indicating a joint effusion).
- The most common sports-related isolated articular cartilage disorder of the elbow is osteochondritis dissecans (OCD).

Introduction

Acute sports-related injuries of the elbow may include fractures and dislocations, as well as injuries to tendons, ligaments, and growth plates (child/adolescent athletes). This chapter will mainly focus on fractures and dislocations. These injuries may occur as a result of direct trauma or indirect trauma from a fall on an outstretched hand (FOOSH), but can also occur without trauma in overhead throwing athletes due to the significant forces generated during the throwing motion [1]. Acute elbow injuries are most prevalent in contact sports, such as American football [2]. Treating significant elbow injuries can be challenging because there are high complication rates and therefore orthopedic referral may be warranted in many cases.

General Diagnostic Approach

Diagnosis of elbow injury begins with physical examination, usually followed by plain radiographs. Radiographs can help quickly evaluate for fracture and/or dislocation. Standard imaging should include anteroposterior (AP) and lateral views, but oblique and dedicated radial head views can increase sensitivity of evaluation. Radiographs can also reveal the presence of a joint effusion, which is suggestive of occult fracture [3]. In child or adolescent athletes, it is important to consider their skeletal maturity in relation to injury. Many considerations specific to pediatric patients revolve around injury to the physis. Treatment can be guided by the Salter-Harris classification of injury to the epiphyseal plate [4]. These athletes can also suffer injuries to the accessory growth plates (apophyses), which are especially prevalent in the elbow. Fusion of the capitellum (1–2 years), radial head (2–5 years), trochlea (8–10 years), olecranon process (10–11 years), lateral epicondyle (11–13 years), and medial epicondyle (15–16 years) can occur at mostly predictable times and can help guide evaluation [5]. If apophyseal abnormalities are indeterminate, radiographs of the contralateral elbow should be taken for comparison.

Musculoskeletal ultrasound can be utilized as a cost-effective, timely tool for evaluation of tendon or ligament injuries [6]. Ultrasound is especially advantageous for evaluating ligamentous injury as it allows for dynamic testing with stress views. Limitations of ultrasound include incomplete evaluation of deeper structures within the joint (e.g., osteochondral lesions, fractures) and operator variability [3].

Computed tomography (CT) is used in the acute setting for more detailed evaluation of fractures. It can be helpful in assessing fracture alignment, degree of comminution, and intra-articular extension [3]. These factors can be important

J. Spittler (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: john.spittler@cuanschutz.edu

A. Seidl
Department of Orthopedics, University of Colorado School of Medicine, UCHealth Steadman Hawkins Clinic Denver – Surgery Center, Englewood, CO, USA
e-mail: ADAM.SEIDL@CUANSCHUTZ.EDU

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in determining the need for surgical intervention versus conservative management. Occasionally, CT arthrography can be utilized to evaluate for intra-articular or ligamentous injury in those athletes who have a contraindication to MRI [3].

MRI provides the most comprehensive evaluation of the elbow; however, it may be costly and difficult to obtain in the acute setting. Standard non-contrast MRI provides a thorough evaluation of the major ligaments, tendons, muscles, bones, and neurovascular bundles [3]. Arthrography (either CT or MR) is rarely needed, except to detect subtle ligament or cartilage injuries that could necessitate surgical intervention in high-level athletes [7, 8].

Fractures and Dislocations

This section will review common elbow fractures and dislocations that may occur during sporting activity. We will focus on the mechanism of injury, epidemiology, clinical presentation, imaging, initial management, classification, indications for referral, follow-up care, and complications of these injuries.

Elbow Dislocation

Mechanism of Injury

Posterolateral dislocation accounts for 80% of elbow dislocations and typically occurs after a fall onto outstretched hand (FOOSH) causing a combination of axial loading, supination of the forearm, and a posterolateral valgus force. Complex dislocations have an associated fracture. The "terrible triad" injury of the elbow consists of a radial head fracture, a type III coronoid fracture, and an associated elbow dislocation. Approximately 60% of terrible triad injuries are the result of minor falls on an outstretched arm from standing height [9]. One-third of cases are the result of falls from greater heights, are bicycle related, or occur as a sports injury. There can also be varus posteromedial instability secondary to a fall on an outstretched arm causing a combination of axial loading and posteromedial varus stress. Finally, transolecranon fracture/dislocation can occur as the result of a high-energy mechanism of injury. With the elbow in a flexed position, the trochlea is driven through the greater sigmoid notch. This results in anterior translation of the forearm relative to the elbow.

Epidemiology

The elbow joint is the second most commonly dislocated large joint in adults, behind the shoulder. In the pediatric population, the elbow is the most commonly dislocated joint. Elbow dislocation accounts for a large percentage of elbow injuries (10%–25%) [10]. The highest incidence of elbow dislocation occurs in younger individuals (10–20 years of age) during sports participation. One study reported that 44.5% of elbow dislocations are sustained during sports [9]. Dislocation occurs more frequently in males than females. In high school athletes, elbow dislocations occur most commonly during wrestling (46.1%), followed by football (37.4%) [11]. Recurrent dislocation is uncommon.

Clinical Presentation

Dislocation is usually obvious with deformity of the elbow and dimpling of the skin (Fig. 16.1a). Patients will guard the injured upper extremity. A complete neurovascular examination should be completed prior to radiographs and/or attempted reduction. Once reduced, a repeat neurovascular examination should be performed and documented. Acute neurovascular injuries are uncommon, but if injury is present, the ulnar nerve is most commonly involved, followed by the anterior interosseous nerve [12].

Imaging

Standard AP and lateral radiographs should be obtained and evaluated for associated fractures (Fig. 16.1c, e). Fractures of the radial head and/or coronoid process of the ulna are relatively common with elbow dislocation.

Classification

Elbow dislocation is classified based on the absence or presence of associated fractures and the direction of displacement. The term simple elbow dislocation implies no associated fractures (Figs. 16.2, 16.4, and 16.5), while complex elbow dislocation infers an associated fracture (Figs. 16.1, 16.3, and 16.6). The direction of displacement is characterized based on the position of the ulna relative to the humerus. Posterior or posterolateral dislocations account for the vast majority of elbow dislocations (Figs. 16.1, 16.2, and 16.3), but the elbow can also dislocate anteriorly (Fig. 16.6), laterally (Fig. 16.5), medially, posteromedially (Fig. 16.4), or divergently (radius dislocates laterally and ulna medially).

Initial Management

Reduction of a simple elbow dislocation should be performed as quickly as possible. Unfortunately, reduction is often unsuccessful on the sideline due to the large forces required. If there is concern for fracture or initial reduction of a dislocation is unsuccessful, the elbow should be splinted and the patient transported for further evaluation and management. Adequate relaxation is key to the success of reduction and usually requires sedation. Multiple reductions techniques have been described, but reduction typically involves correction of medial or lateral displacement followed by longitudinal traction and flexion. Upon successful reduction, a palpable clunk is often felt (Fig. 16.1b). If fluo-

roscopy is available, the elbow should be visualized during range of motion to ensure reduction and to document the arc of stability. If not available, post-reduction radiographs should be obtained to confirm successful reduction and evaluate for fractures that may not have been visualized on the initial radiographs (Fig. 16.1d, f). If the elbow is stable through the entire range of motion, it should be splinted at 90° in neutral forearm rotation. If the elbow subluxes or dislocated in extension, stability should be assessed with the forearm pronated. If pronation adds stability, the elbow should be splinted at 90° with the forearm pronated.

Indications for Referral

In cases where the elbow remains unstable or more than 30°–40° of flexion is required for stability, surgical intervention should be strongly considered. Only 2.3% of people who sustain a simple elbow dislocation require surgical intervention for continued instability [13]. Surgical referral is also indicated for complex elbow dislocations (Figs. 16.1g, 16.3, and 16.6). Any irreducible dislocation or neurovascularly compromised cases should be immediately referred to a center with an urgent orthopedic care.

Follow-Up Care

In the case of a simple elbow dislocation that is stable once reduced, the splint should not be used for an extended period of time. With a documented stable arc of motion, the patient can be transferred into a hinged elbow brace from the splint within 1 week with brace limitations based on the documented stabile range of motion. Gradual progression of motion is obtained by increasing the allowed range by adjusting the hinged elbow brace. Goal is to obtain full elbow range of motion by 4–6 weeks post injury. Early range of motion is key to the successful treatment of simple elbow dislocations.

Complications

Prolonged splint immobilization has been associated with significant limitations in final range of motion and unsatisfactory outcomes [14, 15].

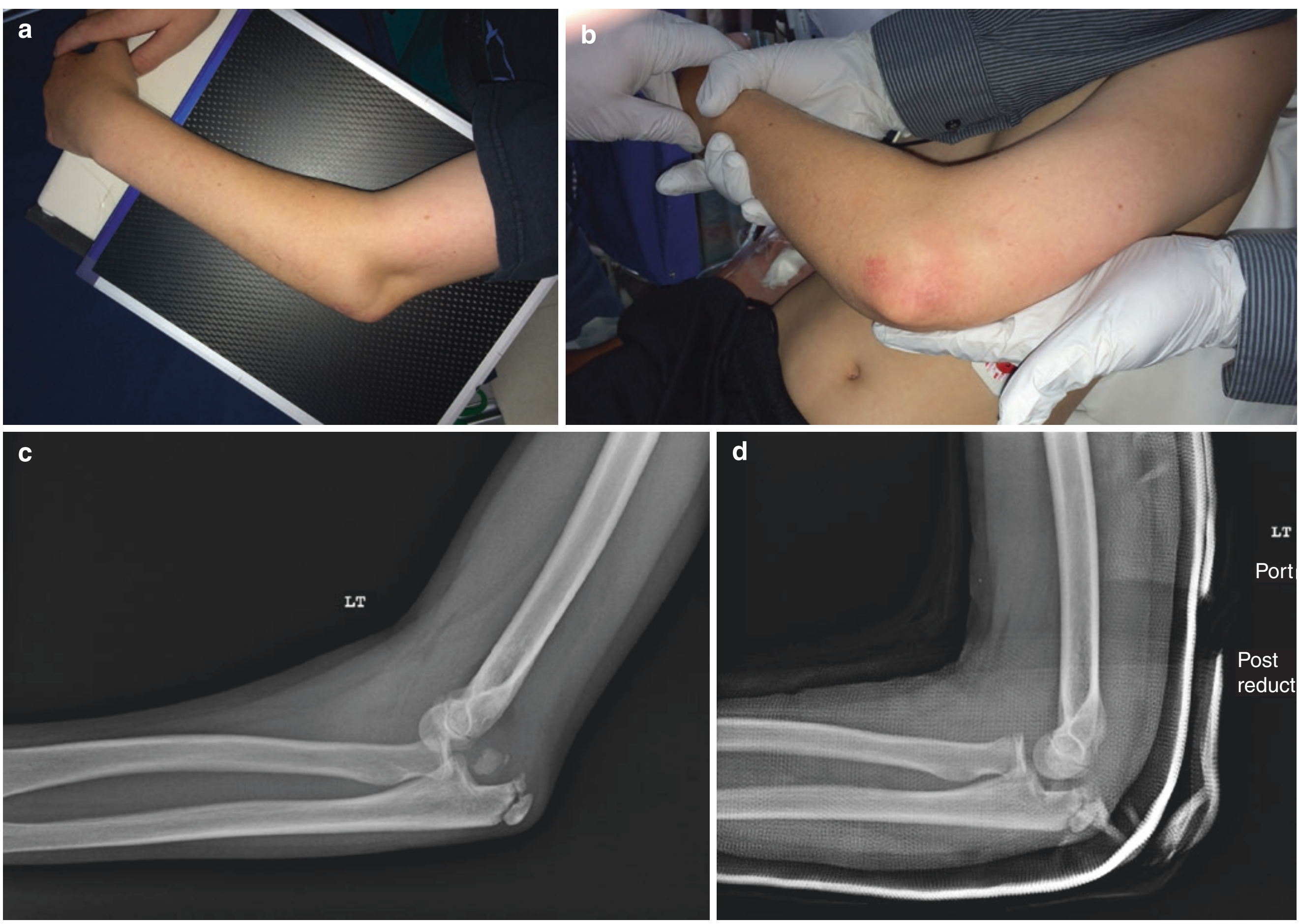


Fig. 16.1 Left elbow injury in a 13-year-old boy as a result of a trampoline injury. Deformity of the elbow (dimpling of the skin) indicative of posterior elbow dislocation (**a**). X-rays revealed postero-lateral elbow dislocation and a lateral condyle fracture (complex) (**c**, **e**). Post-reduction picture and radiographs revealed anatomical alignment (**b**, **d**, **f**). Eventually, his lateral condyle fracture was pinned (**g**)

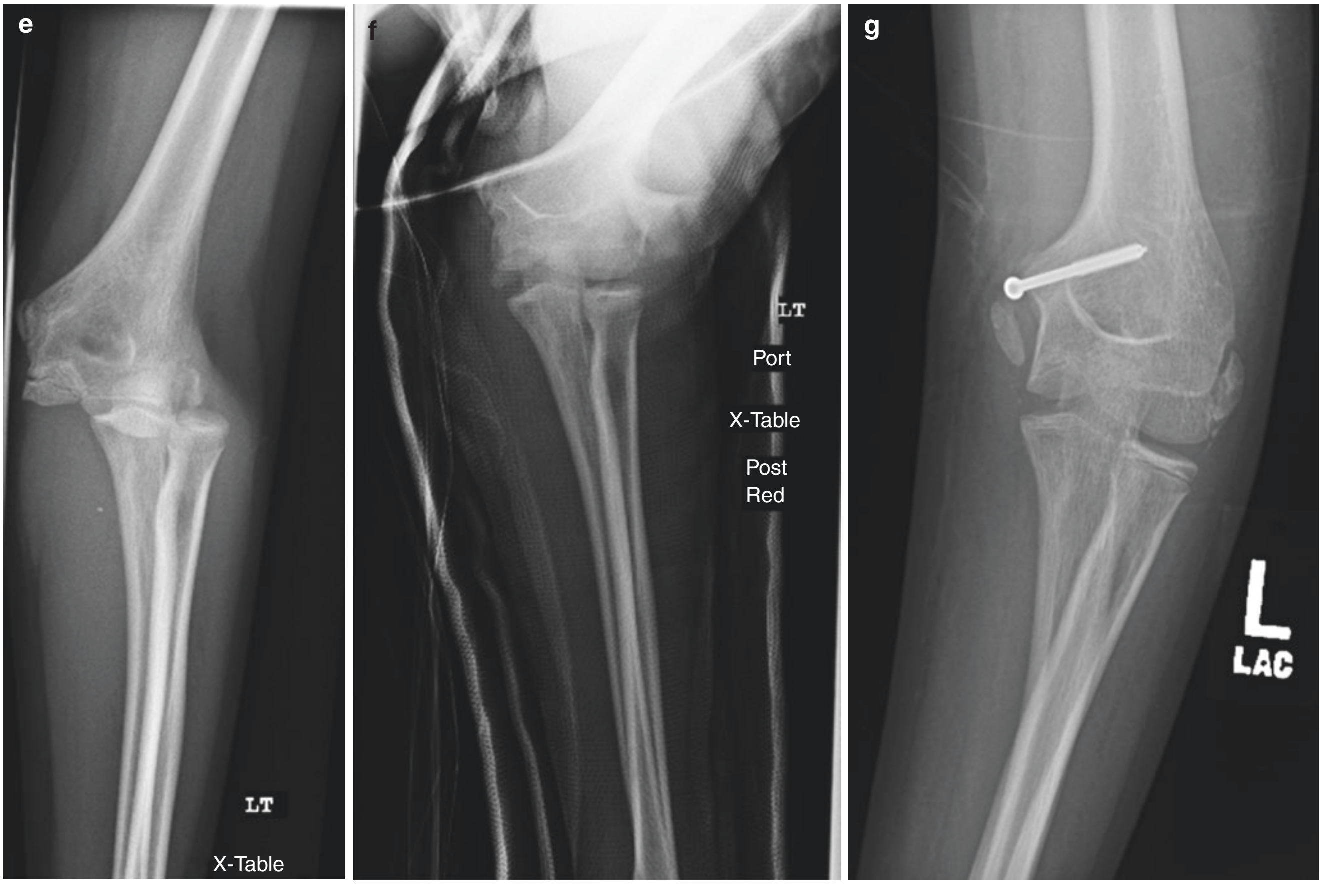


Fig. 16.1 (continued)

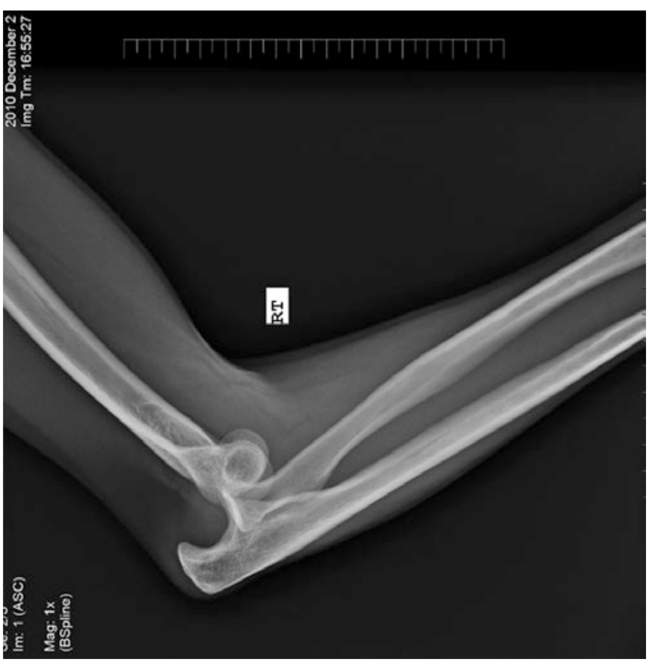


Fig. 16.2 Lateral radiograph of the elbow indicating simple posterior dislocation, with no associated fractures

Fig. 16.3 Radiograph of the elbow indicating complex posterior elbow dislocation, with associated displaced fracture of the coronoid process of the ulna (arrow)

Radial Head/Neck Fractures

Mechanism of Injury

Most often results from a fall onto the outstretched hand. The radial head or neck fractures when it strikes the capitellum with an axial load.

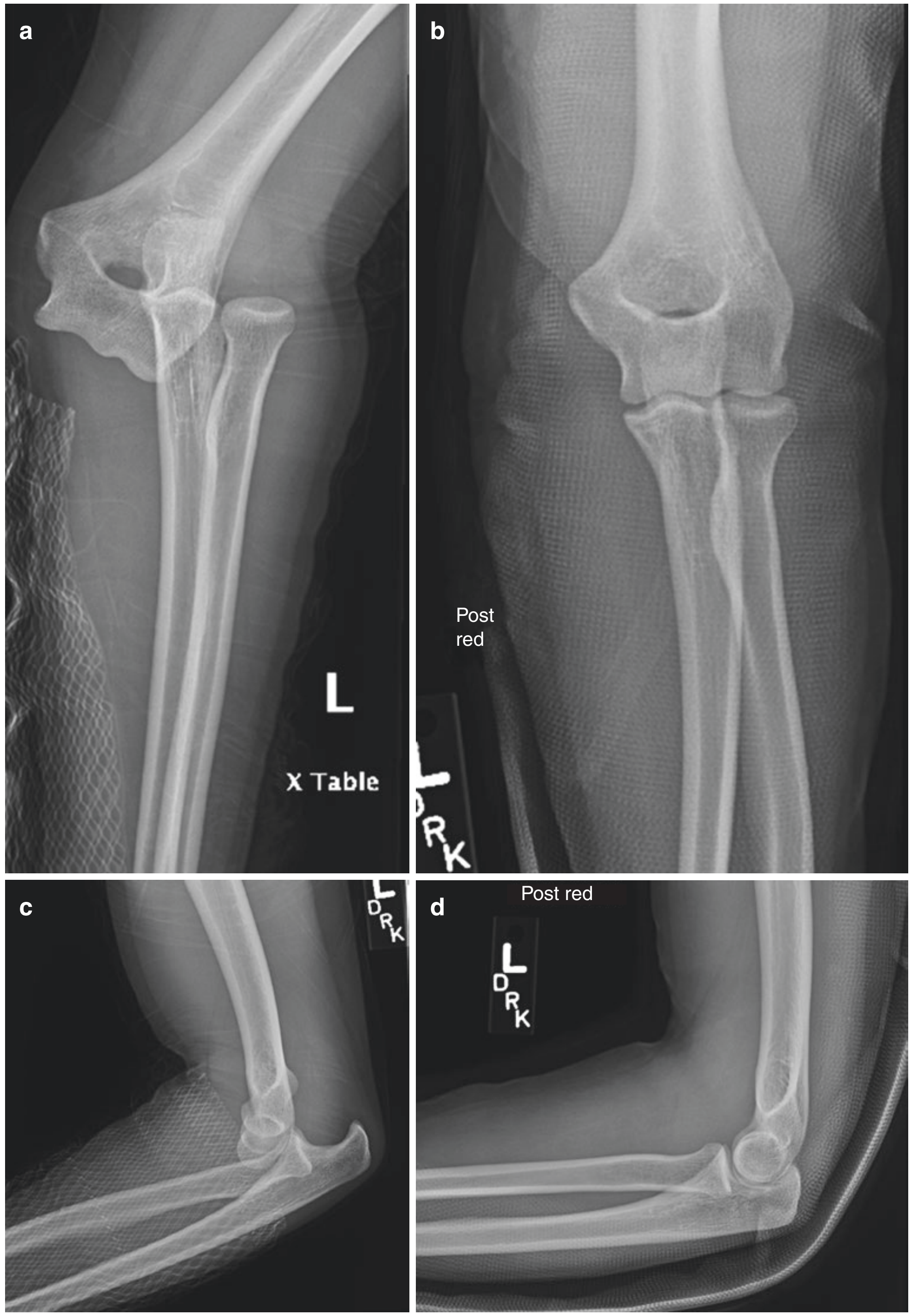


Fig. 16.4 Left elbow injury in a 30-year-old female as a result of a ski injury. X-rays revealed postero-medial elbow dislocation (**a**, **c**). Post-reduction radiographs revealed anatomical alignment (**b**, **d**)

Epidemiology

Fractures of the radial head are very common and account for one third of elbow fractures and between 1.5% and 4% of all fractures in adults. The majority of these fractures (85%) occur between the ages of 20 and 50, with a mean age of 45 years at the time of injury. Females sustain this injury slightly more often than males and are typically older at the time of injury [16]. These injuries are rare in children.

Clinical Presentation

Patients will typically present with pain and limited elbow range of motion. Localized tenderness will be present over the radial head. A moderate to large joint effusion is often present and is best appreciated at the "soft spot" of the elbow. It is crucial to evaluate the ipsilateral distal forearm and wrist, as radial head trauma can be associated with interosseous ligament and distal radioulnar joint disruption (Essex-Lopresti injury).

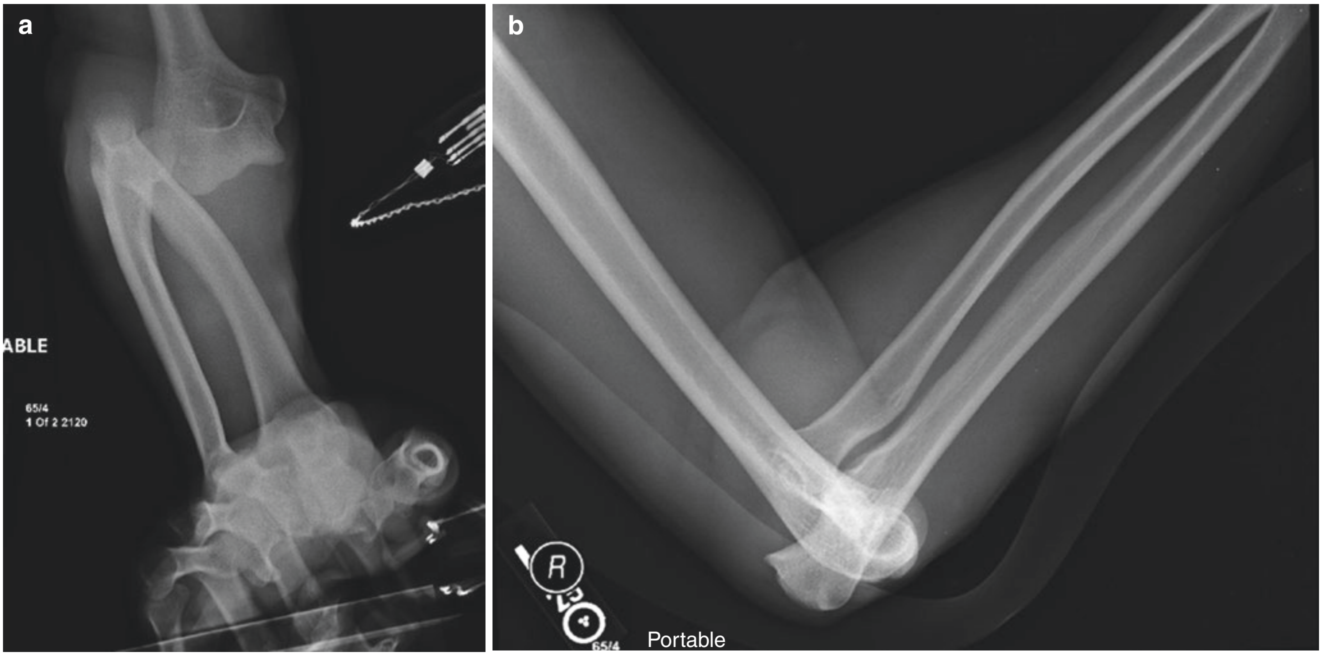


Fig. 16.5 Right lateral elbow dislocation in a 38-year-old male (**a**, **b**)

Fig. 16.6 A complex right volar elbow dislocation with comminuted proximal ulna fracture in a 51-year-old male

Imaging

Anteroposterior, lateral, and oblique radiographs of the elbow should be obtained to diagnose the fracture and determine the degree of comminution and displacement. Nondisplaced fractures may be difficult to appreciate, but attention should be paid to the lateral radiograph for the presence of a "fat pad" sign – indicating a joint effusion (Fig. 16.7). Wrist radiographs (AP and lateral) should be obtained if wrist pain is present to evaluate for associated disruption of the distal radioulnar joint.

Initial Management

The majority of isolated radial head fractures can be treated nonoperatively. For nondisplaced or minimally (<2 mm) displaced fractures, symptomatic management consists of temporary sling wear with early range of motion after 3–5 days [17]. Pain relief can be helped with aspiration of the hemarthrosis and injection of a local anesthetic agent, but this is rarely necessary.

Classification

The most commonly used classification system for radial head and neck fractures was described by Mason which was modified by Broberg and Morrey (Fig. 16.8) [18]:

- Type 1 – Nondisplaced (Fig. 16.7)
- Type 2 – Displaced (>2 mm) fracture of the head or neck (Fig. 16.9)
- Type 3 – Severely comminuted fracture of the head and neck, typically not reconstructible (Fig. 16.10)
- Type 4 – Associated with elbow dislocation

Indications for Referral

Operative management is reserved for displaced fractures and may consist of fixation or replacement, based on the fracture pattern. Displaced fractures can be treated with a posterior splint and referred to orthopedics for open/closed reduction [17].

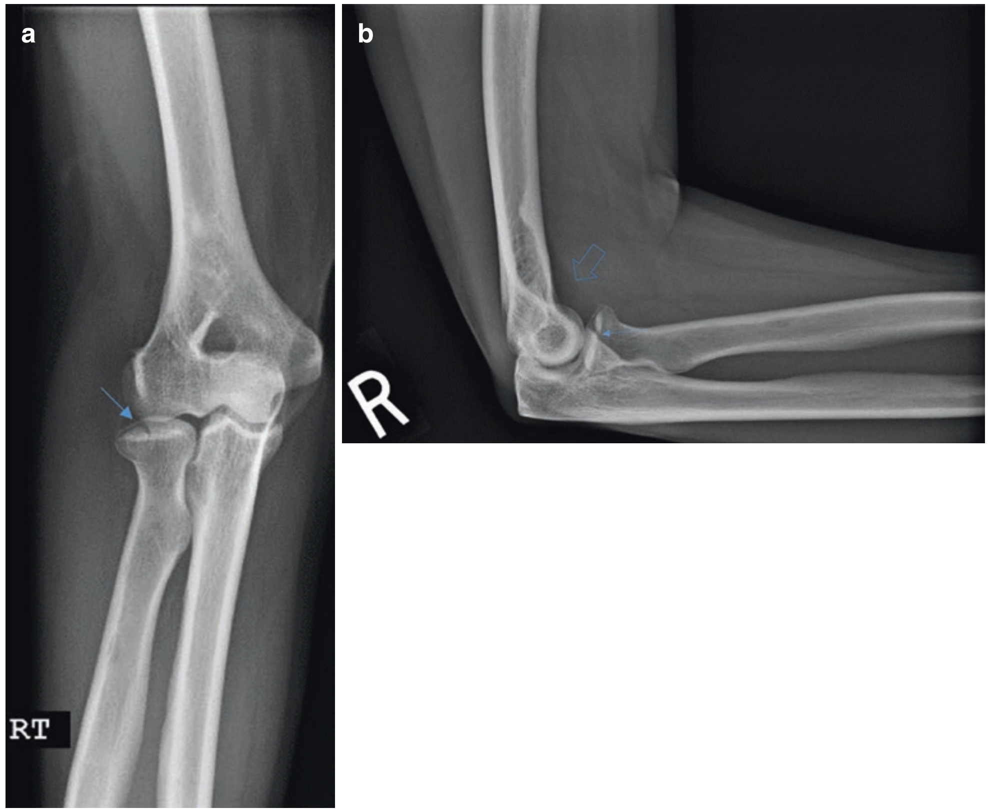


Fig. 16.7 Acute type I (nondisplaced) radial head fracture (solid arrows) with associated joint effusion (open arrow) (**a**, **b**)

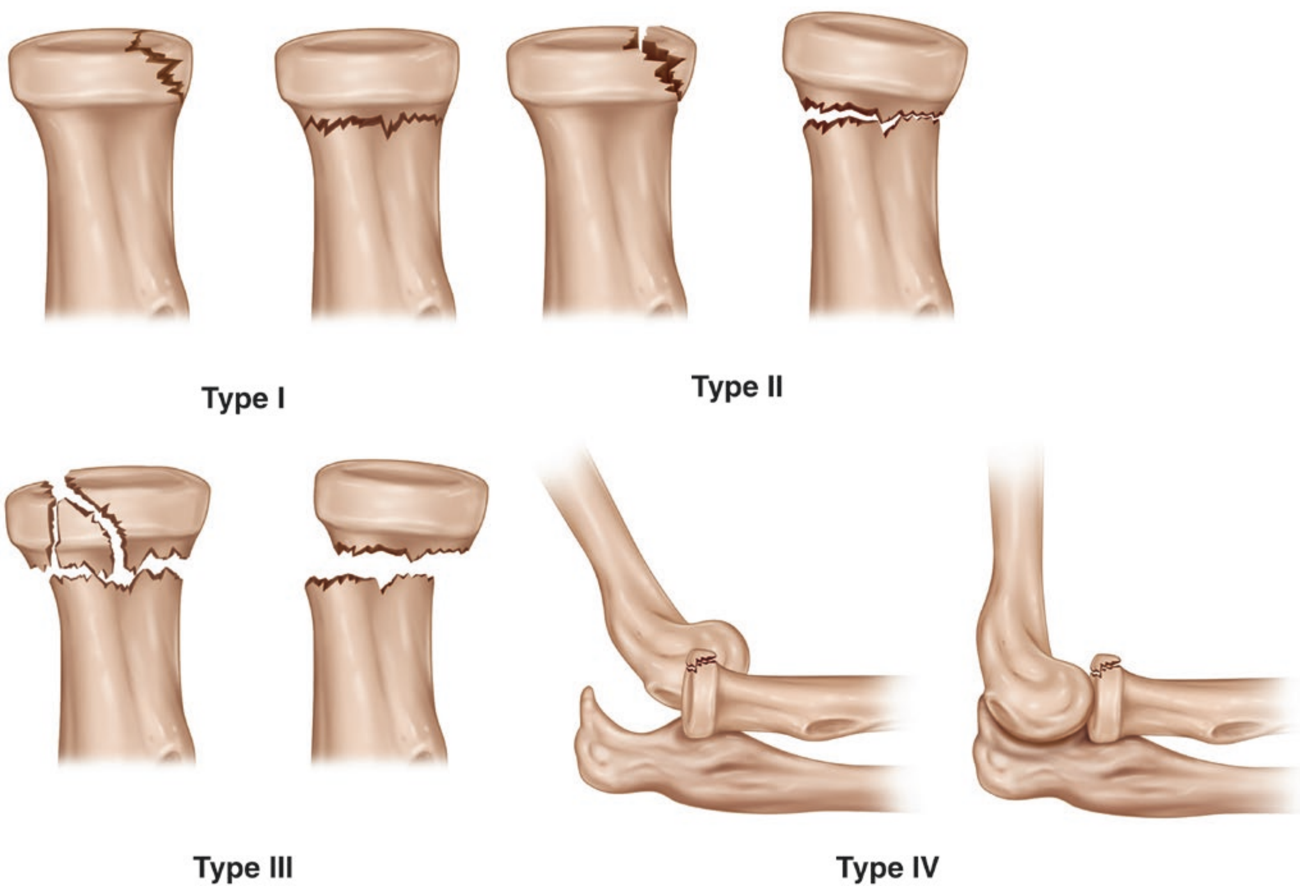


Fig. 16.8 Broberg and Morrey's modification of the Mason classification for radial head and neck fractures [18]

Follow-Up Care
Athlete can be seen back at 7–10 days for repeat examination of nondisplaced fractures and discontinuation of sling use.

Complications
As with the treatment of all elbow trauma, the most common complication is stiffness, which can be avoided with early motion.

Pediatric Considerations
Plain radiographs can evaluate for commonly associated injuries, such as elbow dislocation, olecranon fractures, and medial epicondyle apophysis avulsion fractures [17]. Neck fractures are commonly Salter-Harris type I–III.

Olecranon Fractures

Mechanism of Injury
Two common mechanisms of injury can lead to an olecranon fracture, direct and indirect:

Fig. 16.9 Acute type II (arrow) radial head fracture in a 31-year-old male

- *Direct* – A fall directly onto the olecranon typically results in a comminuted fracture pattern.
- *Indirect* – A fall onto the outstretched arm leading to a strong contraction of the triceps. This results in a simpler transverse or oblique fracture pattern.

Epidemiology
Olecranon fractures occur in a bimodal age distribution. Younger individuals sustain this fracture as the result of high-energy trauma, such as motor vehicle accident, fall from height, or contact sport. Older individuals typically sustain this fracture from a simple ground level fall.

Clinical Presentation
Patients present with pain and typically support the injured extremity with the contralateral hand. A bony defect may be palpated over the subcutaneous ulna at the fracture site. Detailed inspection of the overlying skin should be performed, as open fractures are not uncommon given the subcutaneous nature of the proximal ulna and olecranon. If the fracture is displaced, patients will typically be unable to extend the elbow actively against gravity. Loss of active elbow extension indicates discontinuity of the triceps mechanism. A careful neurosensory evaluation should also be performed, as associated ulnar nerve injury can occur.

Imaging
AP and lateral radiographs of the elbow should be obtained. The lateral radiograph is most crucial to diagnose the fracture and determine the degree of comminution and displacement (Figs. 16.11, 16.12, and 16.13).

Initial Management
Nonoperative treatment is reserved for truly nondisplaced fractures or displaced fractures in older individuals with limited baseline function. Nonoperative treatment consists of a

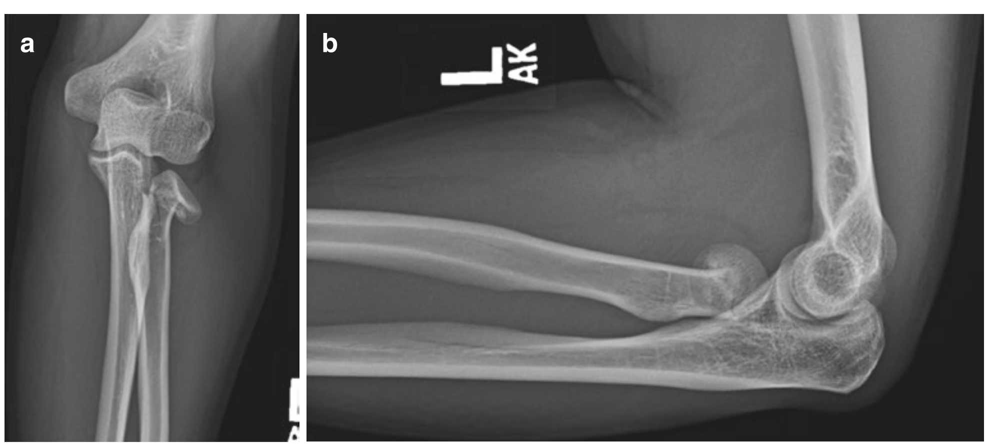


Fig. 16.10 Acute type III (displaced) radial head fracture in a 27-year-old male as a result of a mountain bike injury (**a**, **b**)

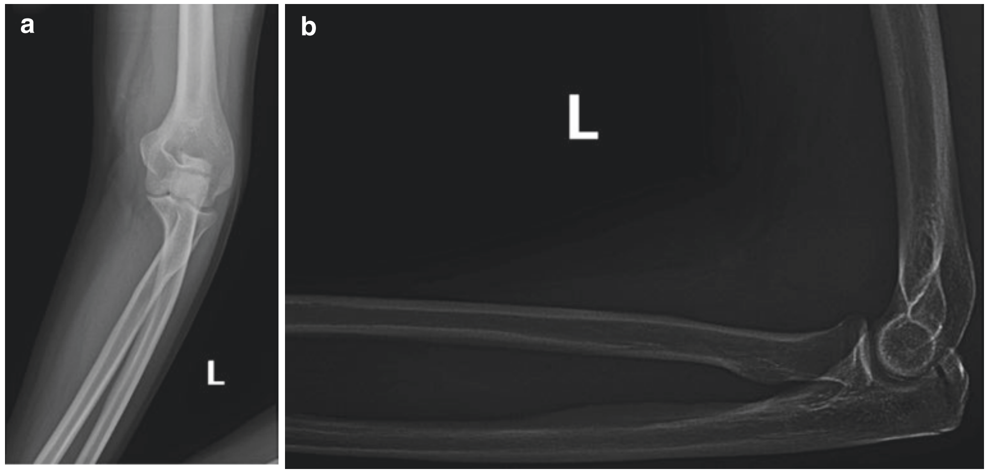


Fig. 16.11 Mildly displaced olecranon fracture in a 25-year-old male (**a**, **b**)

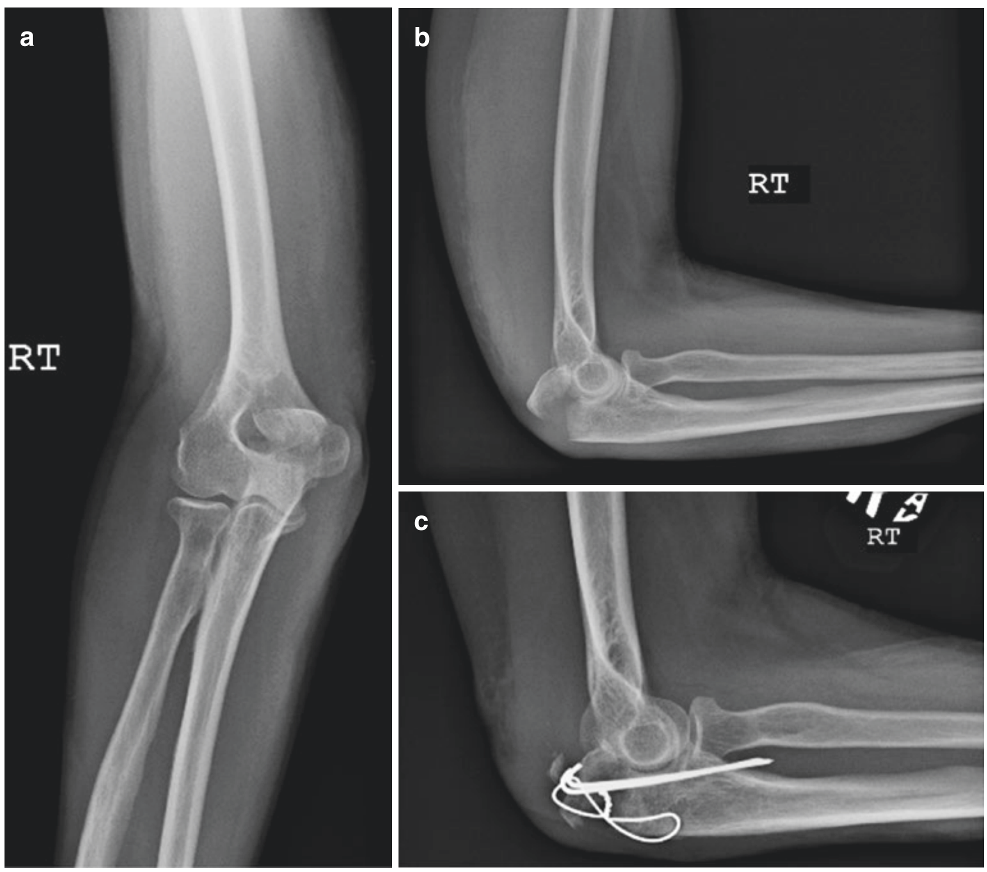


Fig. 16.12 Displaced olecranon fracture in a 59-year-old female as a result of a fall while mountain biking (**a**, **b**). The alignment was improved with surgery (**c**)

short period in a long arm splint or cast at approximately 45° of flexion.

Classification

Although several classification systems for olecranon fractures have been described, it is easiest to describe as either displaced or nondisplaced and comminuted or noncomminuted.

Indications for Referral

Operative treatment is recommended for any displaced olecranon fracture (Figs. 16.12 and 16.13). Fracture displacement indicates disruption of the elbow extensor mechanism.

Follow-Up Care

Range of motion is initiated within 7 days with repeat radiographs shortly after motion is initiated to rule out fracture

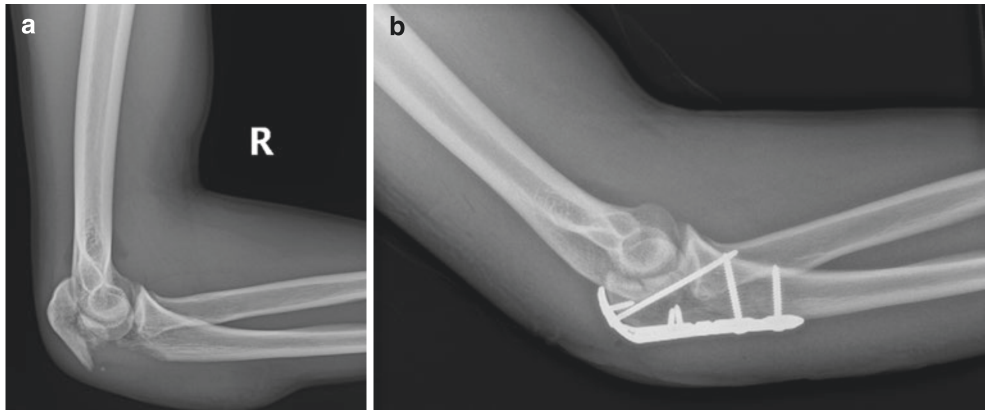


Fig. 16.13 Severely displaced and comminuted olecranon fracture in a 16-year-old male (**a**). Near anatomic alignment was achieved with surgery (**b**)

displacement. Fracture healing typically takes between 6 and 8 weeks.

Complications

Significant functional loss can be expected if displaced fractures are treated nonoperatively.

Coronoid Process Fractures

Mechanism of Injury

As coronoid fractures often occur with an elbow dislocation, they follow a similar pattern of injury. They usually are caused by a fall on the elbow or FOOSH injury from standing height or higher [19]. Specific mechanisms vary and can involve twisting and flexion or hyperextension [20, 21].

Epidemiology

Coronoid fractures are often associated with an elbow dislocation, but can be an isolated injury. Overall they are relatively uncommon injuries, occurring in only 2%–15% of elbow dislocations [22]. They may be part of the "terrible triad" of the elbow consisting of posterior elbow dislocation, radial head fracture, and coronoid process fracture [20].

Clinical Presentation

Most patients with coronoid fractures present following elbow dislocation. If the dislocation has not been reduced, there will be a deformity visible. There is also usually pain, swelling, and decreased range-of-motion present [21].

Imaging

AP and lateral radiographs of the elbow should be obtained. The lateral radiograph is most crucial to diagnose the fracture and determine the degree of comminution and displacement (Figs. 16.14 and 16.15). Small coronoid fractures are easy to miss and may be difficult to distinguish from radial head fractures [23]. If there is uncertainty in diagnosis, CT is often preferred for advanced imaging due to better bony definition [21].

Fig. 16.14 Nondisplaced coronoid process fracture (arrow) in a 22-year-old female as a result of a ski injury

Initial Management

Again, this usually follows the treatment for elbow dislocation as they almost always occur with that injury. Therefore, initial management consists of reduction of elbow dislocation (if present) followed by immobilization in a posterior elbow splint at 90° of flexion. See "elbow dislocations" section for further details.

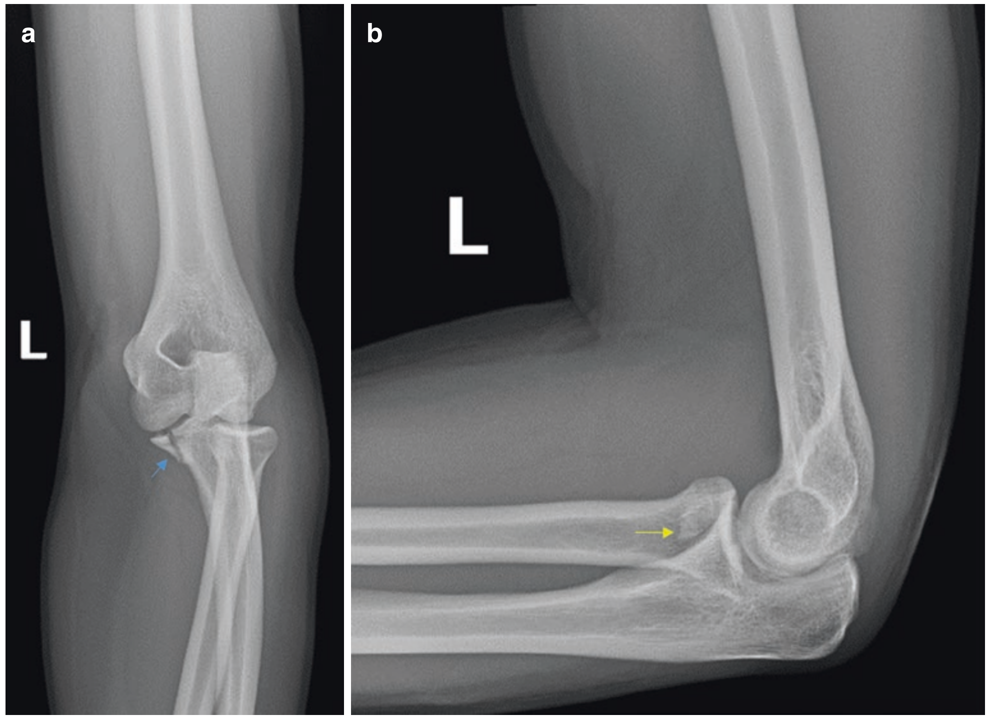


Fig. 16.15 Mildly displaced lateral coronoid process fracture (arrows) in a 26-year-old female as a result of a ski injury (**a**, **b**)

Classification

The most common classification for coronoid fractures was described by Regan and Morrey as three types: tip avulsion (type I), fractures involving less than 50% of the coronoid (type II), or fractures involving more than 50% of the coronoid (type III) [24].

Indications for Referral

Typically Morrey type I fractures with instability, type II fractures, and type III fractures are repaired surgically and therefore warrant orthopedic referral [25].

Follow-Up Care

Early mobilization within the first few weeks is preferred. During the early rehabilitation stages, a hinged elbow brace with the terminal 30° of extension blocked can help with ROM while protecting against valgus and varus stress [24, 26]. Serial radiographs can be utilized to confirm fracture healing and maintenance of a successful reduction. As with the initial radiographs, a true lateral radiograph is the most important view to visualize the coronoid during follow-up care [21].

Complications

As with most elbow injuries, prolonged immobilization (more than 3–4 weeks) often leads to persistent stiffness, pain, and loss of function [26].

Distal Humerus or Supracondylar Fracture

Mechanism of Injury

Most commonly occurs after low-energy falls in elderly patients with decreased bone density. In the younger population, distal humerus fractures occur with high energy impact. Elbow position at the time of injury affects the fracture type. In children, this fracture usually occurs with a FOOSH injury that results in sudden elbow extension, but can also result from a direct blow to the elbow with the elbow flexed [27].

Epidemiology

Supracondylar fractures are the most common elbow fracture in children (up to 70%) [28]. Motor vehicle and sporting accidents are common causes of injury in young individuals. Fractures of the distal humerus are relatively uncommon in adults, accounting for 2% of all fractures and 30% of all humerus fractures [29–31]. The majority of distal humerus fractures in adults occur in older female patients, but these fractures can also occur in young individuals as the result of high energy trauma.

Clinical Presentation

In children, small, nondisplaced or minimally displaced fractures may be difficult to detect on examination. Displaced fractures are often associated with obvious deformity, ecchymosis, and swelling. Skin inspection is performed to ensure

that the injury is closed. If range of motion is attempted, crepitation is usually present. A thorough neurologic examination is imperative as there is a large risk of neurologic injury (10%–15%), often anterior interosseous nerve (AIN) palsy [32]. Vascular injury is also possible and distal pulses (especially radial) should be evaluated.

Imaging

AP, lateral, and oblique radiographs of the elbow should be obtained to diagnose the fracture and determine the degree of comminution and displacement (Fig. 16.16). Traction radiographs can be extremely helpful in understanding fracture pattern but are not well tolerated by the patient. If articular involvement and/or comminution is suspected (Fig. 16.17), CT may be utilized for preoperative planning. These fractures can often be subtle on radiographs in children. A positive anterior fat pad sign, posterior joint effusion, or if the capitellum projects posterior to the anterior humeral line may indicate fracture even when a distinct fracture line is not visualized (Fig. 16.18). It is also important to get radiographs of the forearm to rule out associated distal radius fracture.

Initial Management

The vast majority of distal humerus fractures require surgical fixation in adults. Nonoperative treatment is typically reserved for completely nondisplaced fractures and/or patients medically unfit for surgery. Initial stabilization of all distal humerus fractures with a well-padded long arm splint should be performed prior to referral to an orthopedic surgeon.

Classification

Multiple established classification systems exist for distal humerus fractures. Internationally, the AO classification system is most commonly used. This system categorizes fractures in adults as extra-articular, partial articular, and articular. Fracture comminution and specific patters are further defined by a number system (Fig. 16.19) [33, 34].

Indications for Referral

Given that almost all of these fractures require surgical fixation in adults, orthopedic referral is recommended. In children, many of these fractures are nondisplaced and can be treated nonoperatively (see "Pediatric Considerations").

Follow-Up Care

Close observation with frequent radiographs is necessary when nonoperative treatment is chosen for nondisplaced fractures, as late displacement can occur.

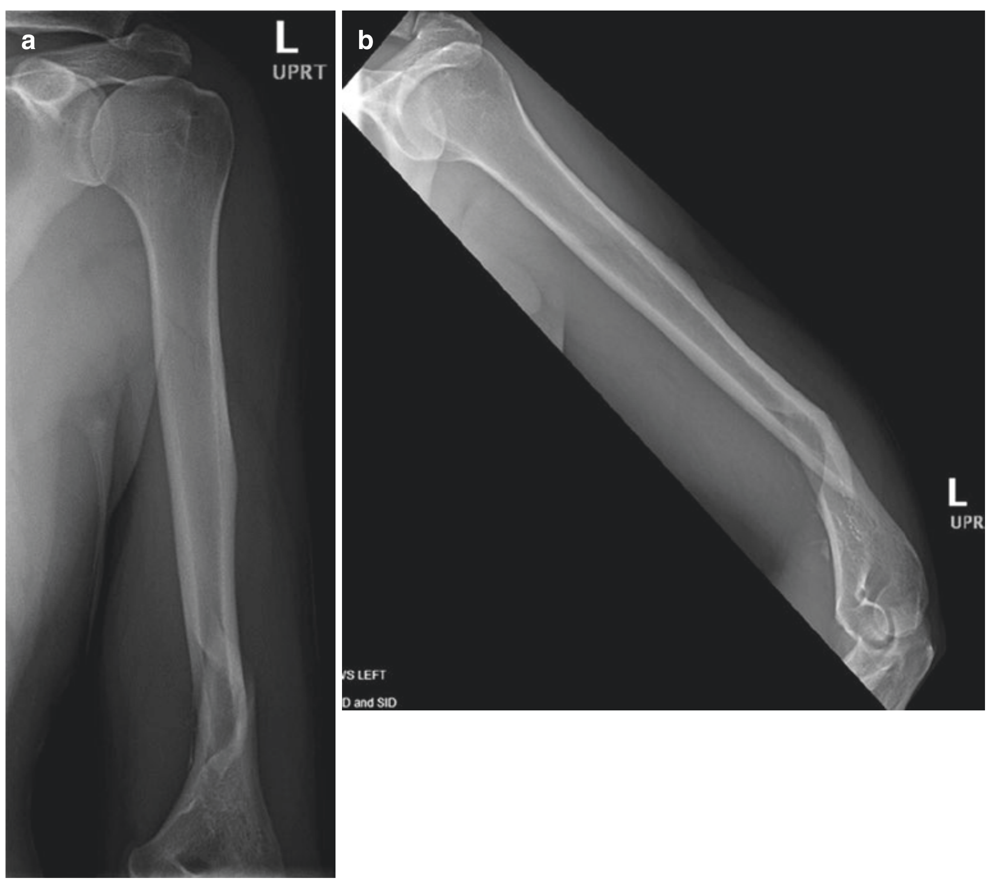


Fig. 16.16 Mildly angulated and displaced, comminuted fracture of the distal humerus (**a**, **b**)

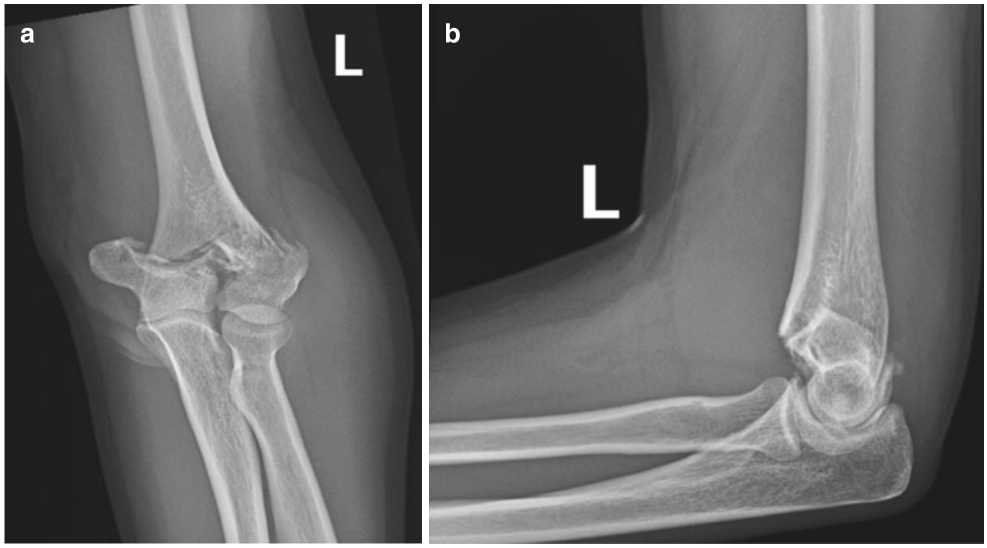


Fig. 16.17 Comminuted and intra-articular supracondylar humerus fracture in a 30-year-old female as a result of a ski injury (**a**, **b**)

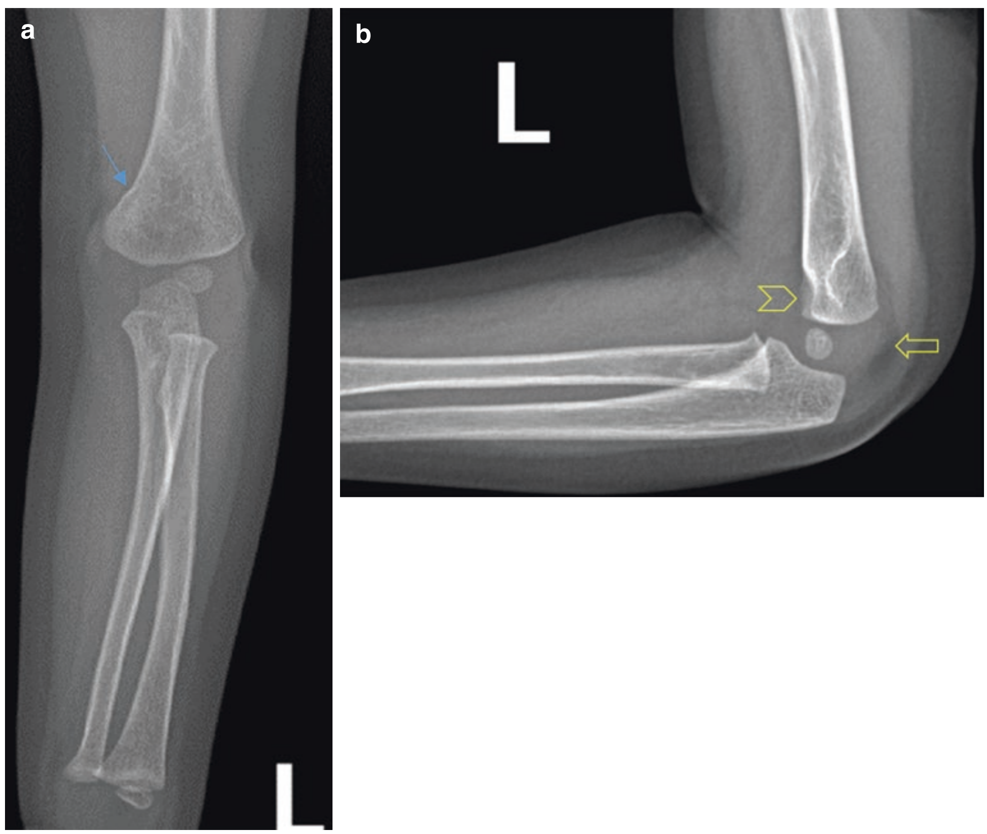


Fig. 16.18 Nondisplaced, incomplete supracondylar humerus fracture that can be difficult to detect on plain radiographs. Mild buckle deformity seen on the AP image (**a**) along the medial aspect of the supracondylar humerus (arrow). On the lateral view (**b**), the capitellum also projects posterior to the anterior humeral line (arrowhead), and there is a visible joint effusion present (open arrow)

Complications

If not frequently evaluated, late displacement of the fracture can occur with nonoperative management. This may result in inadequate healing and disturbance of elbow function.

Pediatric Considerations

Supracondylar fractures can be classified into three different types in children. Type I fractures are non-displaced and can often be difficult to detect on plain radiographs. Type II fractures still have intact posterior periosteum and appear to be "hinged" posteriorly. Type III fractures have complete displacement of the distal fragment relative to the humeral shaft [27]. Type I fractures are treated nonoperatively with a long arm posterior splint with gutters – neutral rotation and flexion to 90°. The patient can be transitioned from the long arm splint to a long arm cast within a few days after injury, with about 3–4 weeks of total immobilization. Type II and III fractures almost always require reduction and percutaneous pinning.

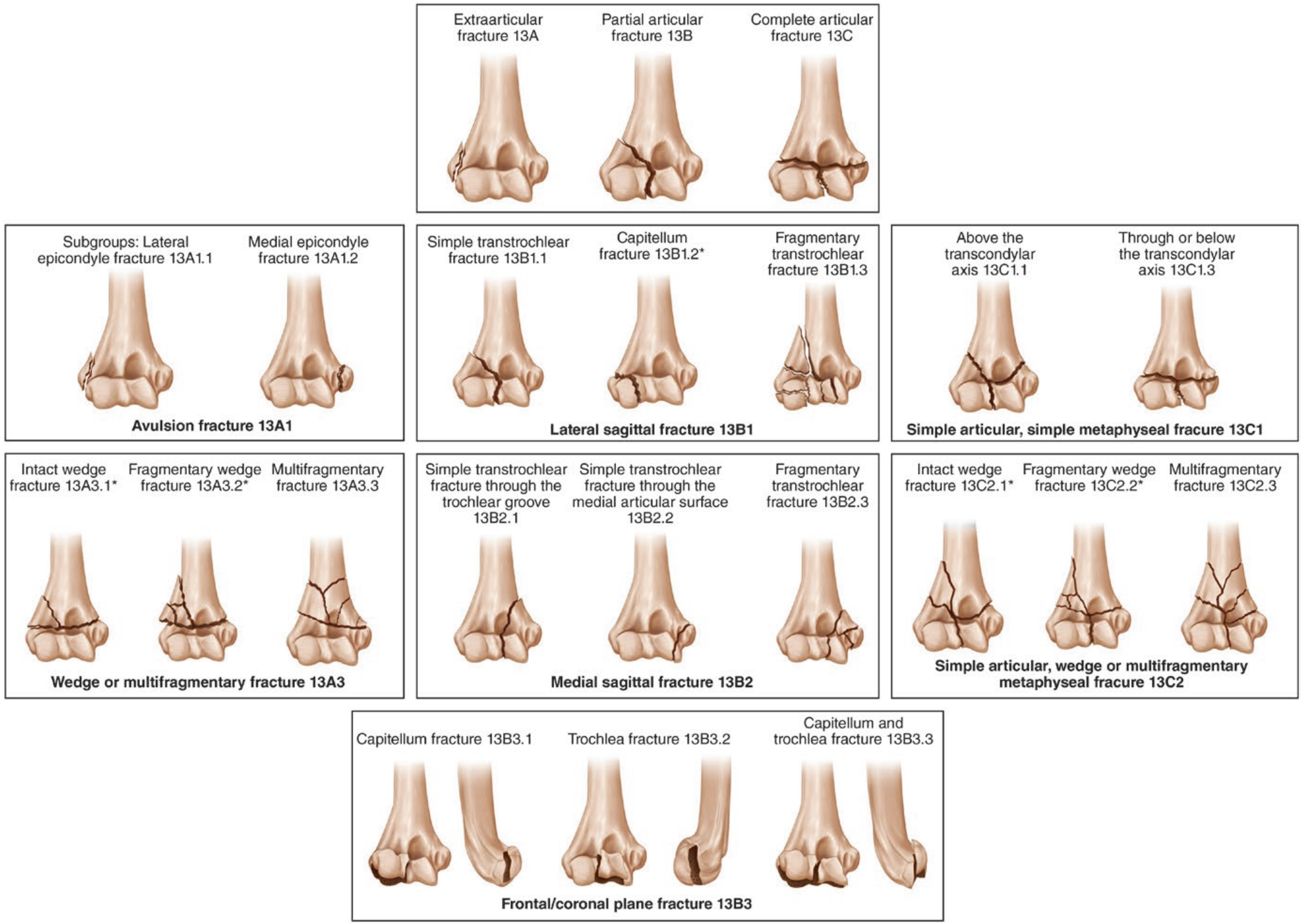


Fig. 16.19 Illustrative depiction of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification for humeral distal end segment fractures [34]

After 4–6 weeks of immobilization with a long arm splint, the pins can be removed and range-of-motion exercises started [35].

Monteggia's Fracture

Monteggia's fracture is a fracture of the proximal third of the ulna with dislocation of the radial head.

Mechanism of Injury

May result from low- or high-energy injuries. Low-energy injuries occur from a fall from standing height onto the outstretched hand with forearm in a forced pronated position. Higher-energy injuries can occur during sporting events, falls from height, and motor vehicle accidents.

Epidemiology

The predominant injury in children is an anterolateral diaphyseal forearm fracture-dislocation [36].

Clinical Presentation

Usually the elbow is tender and range-of-motion is limited. Swelling may not be present initially, but can increase during the days following the injury. Sometimes the fractured ulna or the dislocated radial head may be palpable. Since injury to the posterior interosseous nerve is common, decreased sensation in the dorsum of the thumb and second/third fingers and weakness in thumb abduction can heighten suspicion of Monteggia's fracture [17].

Imaging

Standard views of the elbow, forearm, and wrist should be obtained, but the injury is most commonly visualized in lateral radiographs.

Initial Management

The arm should be placed in a sugar tong or reverse sugar tong splint and referred to an orthopedic surgeon.

Classification

The Bado classification of Monteggia's fracture (Fig. 16.20) is as follows [37]:

- Type I – Proximal ulna fracture with *anterior* dislocation of the radial head (Figs. 16.21 and 16.22)
- Type II – Proximal ulna fracture with *posterior* dislocation of the radial head (Figs. 16.23 and 16.24)
- Type III – Proximal ulna fracture with *lateral* dislocation of the radial head
- Type IV – Fractures of both the radius and ulna at the same level with anterior dislocation of the radial head

Indications for Referral

All of these fractures should be referred to an orthopedic surgeon. Initial attempt at closed reduction is made (usually under general anesthesia). If closed reduction is successful, immobilization for 4–6 weeks in a long arm cast results in healing [17]. If closed reduction is not successful, ORIF should be completed.

Follow-Up Care

Athlete can follow-up at 4–6 weeks for cast removal and examination. A protective forearm splint can be utilized for the first 1–2 months after injury for return to sports.

Lateral Condyle Fracture

Mechanism of Injury

This usually occurs from a fall on outstretched hand, causing axial load to the elbow.

Epidemiology

This is predominantly an injury of children. These are the second most common type of elbow fractures that require surgical repair in children [38].

Clinical Presentation

There is frequently an ecchymosis over the lateral elbow due to bleeding of the intramuscular septum [27]. Similar to supracondylar fractures, a thorough neurovascular examination is important; however, risk is less due to location in relation to neurovascular structures.

Imaging

Nondisplaced fractures may be missed as they can be difficult to visualize on radiographs (Figs. 16.25, 16.26, and 16.27). In addition, they are often misdiagnosed as supracondylar fractures. Therefore, advanced imaging with CT or MRI may be warranted if diagnosis is unclear.

Initial Management

Only fractures that are truly nondisplaced should be treated nonoperatively [39]. The patient can be placed in a long arm posterior splint with lateral gutter.

Indications for Referral

Lateral condyle fractures with any displacement should be treated operatively with open reduction and percutaneous pinning. After 4–6 weeks of immobilization with a long arm splint, the pins can be removed and range-of-motion exercises started [27].

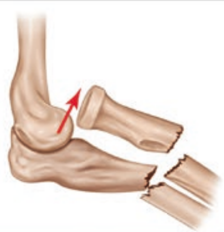
	Bado type	Frequency	Description
	Type I	55–75% (less common in adults)	Anterior dislocation of the radial head with fracture of the ulna shaft (diaphysis)
	Type II	10–15% (rare in children)	Posterior dislocation of the radial head with fracture of the ulna shaft (diaphysis) or metaphysis
	Type III	10–20% (Primarily in Children)	Lateral dislocation of the radial head with fracture of the ulna metaphysis
	Type IV	Rare	Anterior dislocation of the radial head with fracture of the shaft (diaphysis) of the ulna and radius

Fig. 16.20 Bado classification of Monteggia fracture-dislocations [37]

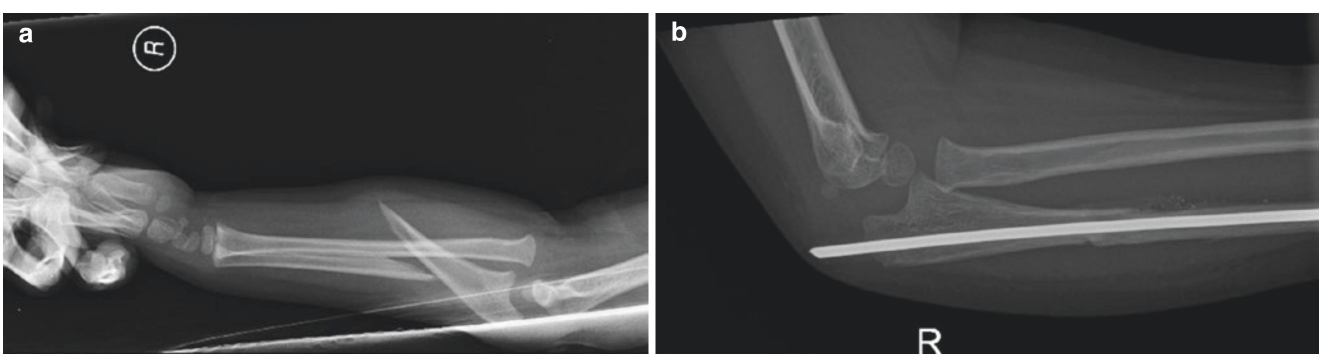


Fig. 16.21 Right proximal ulna fracture and anterior radial head dislocation (Monteggia I) in a 4 year-old girl (**a**). This required an intramedullary rod (**b**)

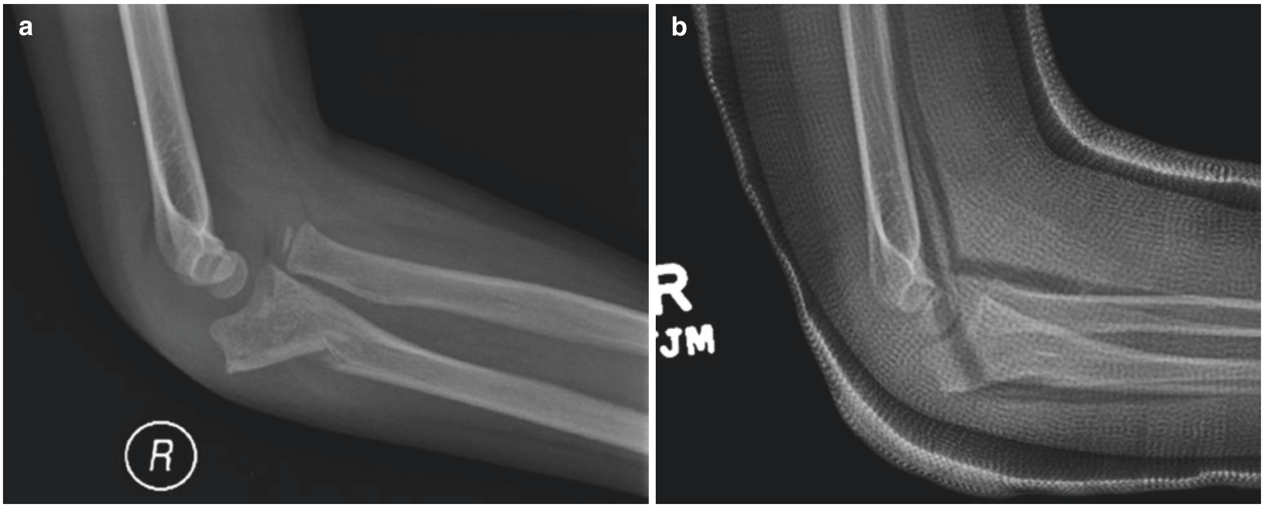


Fig. 16.22 Right proximal ulna fracture and anterior radial head dislocation (Monteggia I) in a 7 year-old boy (**a**). Post-reduction radiograph showed near anatomical alignment (**b**)

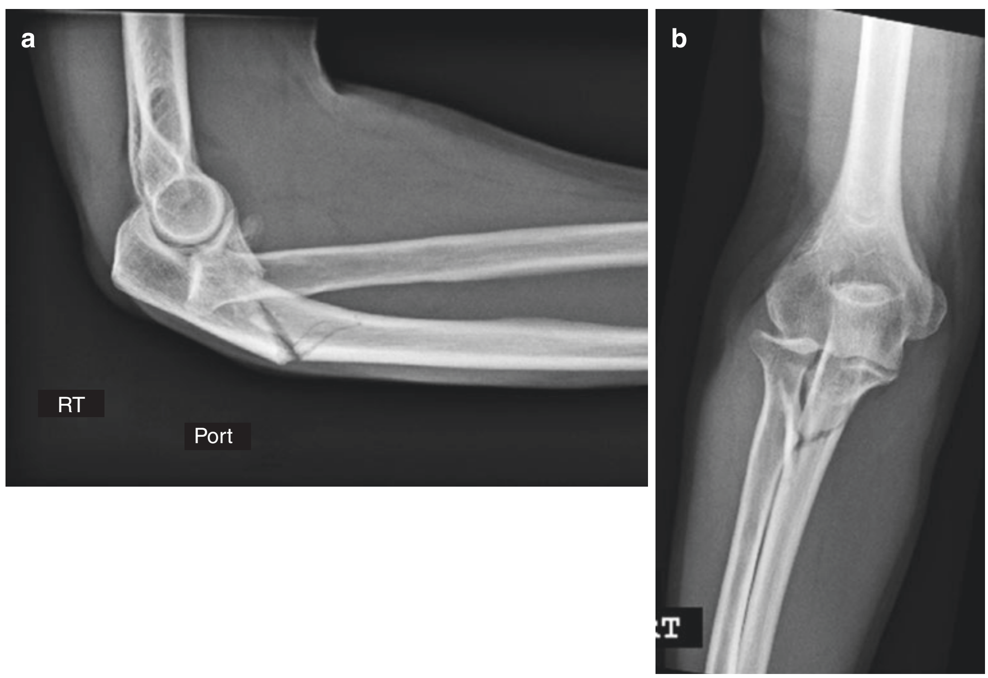


Fig. 16.23 Right proximal ulna fracture and posterior radial head dislocation (Monteggia II) in a 65 year-old female as a result of a mountain bike injury (**a**, **b**)

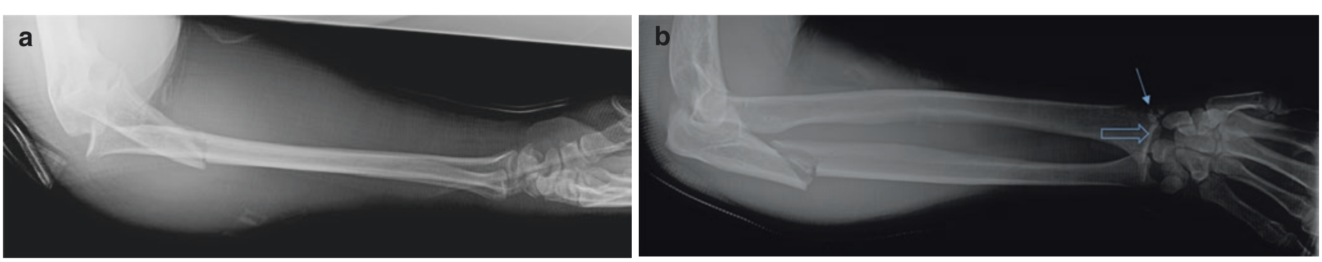


Fig. 16.24 Right proximal ulna fracture and posterior radial head dislocation (Monteggia II) in a 39 year-old male as a result of a fall (**a**). Post-reduction radiograph showed better anatomical alignment (**b**). There is also a distal radius fracture (arrow) with widening of the scapholunate interval (open arrow)

Fig. 16.25 Avulsion fracture of the left lateral condyle (arrow) in a 68-year-old female as a result of a posterior elbow dislocation

Follow-Up Care

The long arm splint can be transitioned to a long arm cast after a few days with about 4 total weeks of immobilization. Then range-of-motion exercises can be started.

Medial Epicondyle Fracture

Mechanism of Injury

Fractures of the medial epicondyle are often caused by a valgus force that causes an avulsion injury of the proximal flexor-pronator mass [40]. This may be due a fall or from a forceful throwing motion.

Epidemiology

This is another injury that occurs predominantly in children. Medial epicondyle fractures account for about 10% of pediatric elbow fractures, and about half of the time are associated with elbow dislocations [41].

Clinical Presentation

There is often pain and swelling over the medial elbow. Paresthesias in the ulnar distribution may be present due to the proximity of the ulnar nerve just posterior to the epicondyle.

Imaging

Nondisplaced or minimally displaced fractures may be difficult to recognize on plain radiographs, especially in young children, due to the presence of the accessory ossification center (Figs. 16.27, 16.28, 16.29, and 16.30). If the presence of fracture or displacement is unclear, comparison views of

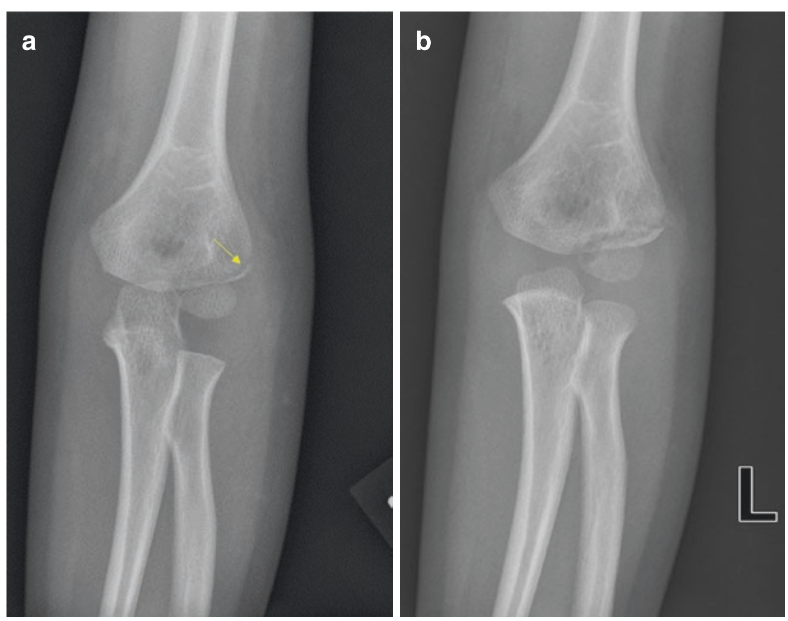


Fig. 16.26 Nondisplaced lateral condyle fracture of humerus in a 5-year-old boy (**a**). Repeat X-rays a month later demonstrated improvement in the fracture line (**b**)

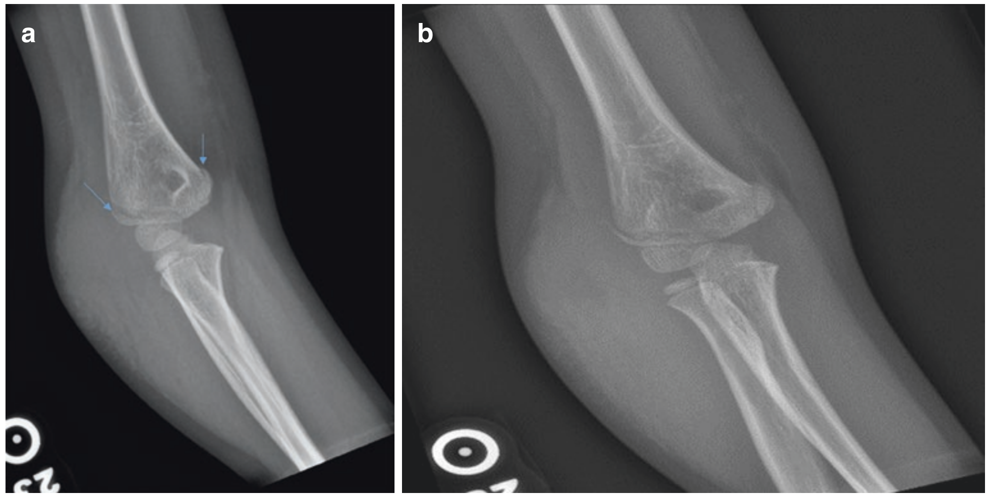


Fig. 16.27 Nondisplaced lateral and medial condyle fractures of humerus (**a**, **b**) in a 5-year-old girl (arrows)

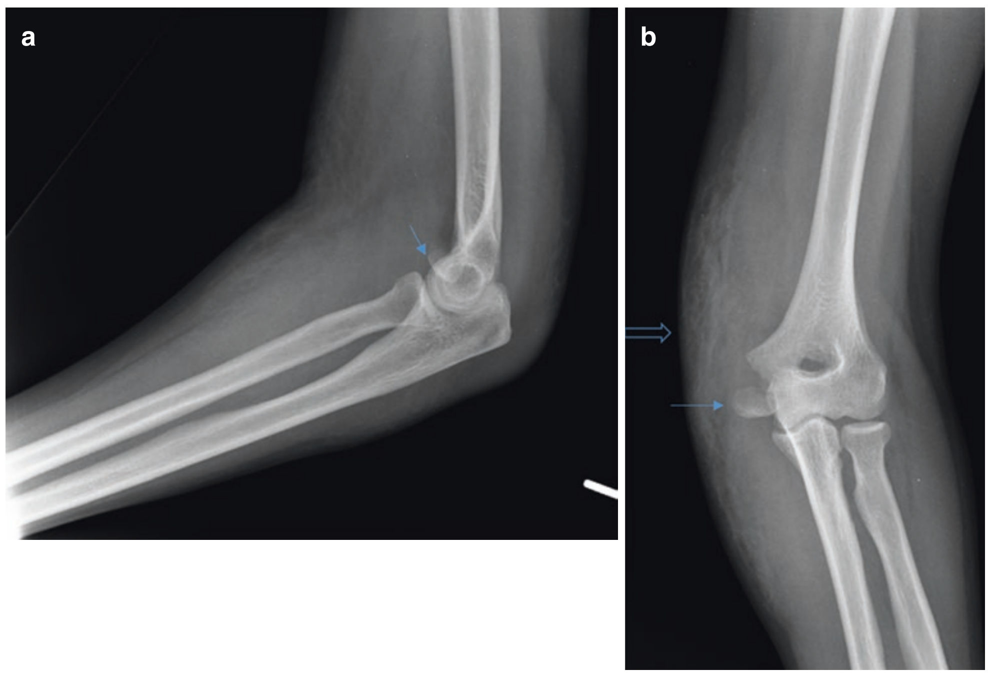


Fig. 16.28 Displaced fracture of the medial condyle of humerus (arrows) in a 30-year-old female (**a**, **b**). There is a significant soft tissue edema (open arrow)

the unaffected elbow can be obtained. If still unclear, advanced imaging with CT or MRI may be indicated.

Initial Management

Minimally (<5 mm) or nondisplaced fractures are usually treated nonoperatively in a posterior elbow splint.

Indications for Referral

Treatment of displaced fragments within the joint (incarcerated) requires reduction. Initial attempt at closed reduction can be made, but if unsuccessful, it will require open reduction and pinning. Treatment of displaced fragments not within the joint (non-incarcerated) is more controversial. Both operative and nonoperative groups have done well in certain studies [27, 42, 43].

Follow-Up Care

The splint can be transitioned to a long arm cast at 90° of elbow flexion for 2–4 weeks [44]. Then the athlete can be transitioned back to a removable posterior splint with range-of-motion exercises out of the splint several times per day for a total of 6–8 weeks of immobilization [44]. Depending on the sport, a hinged elbow brace can be used for return-to-play.

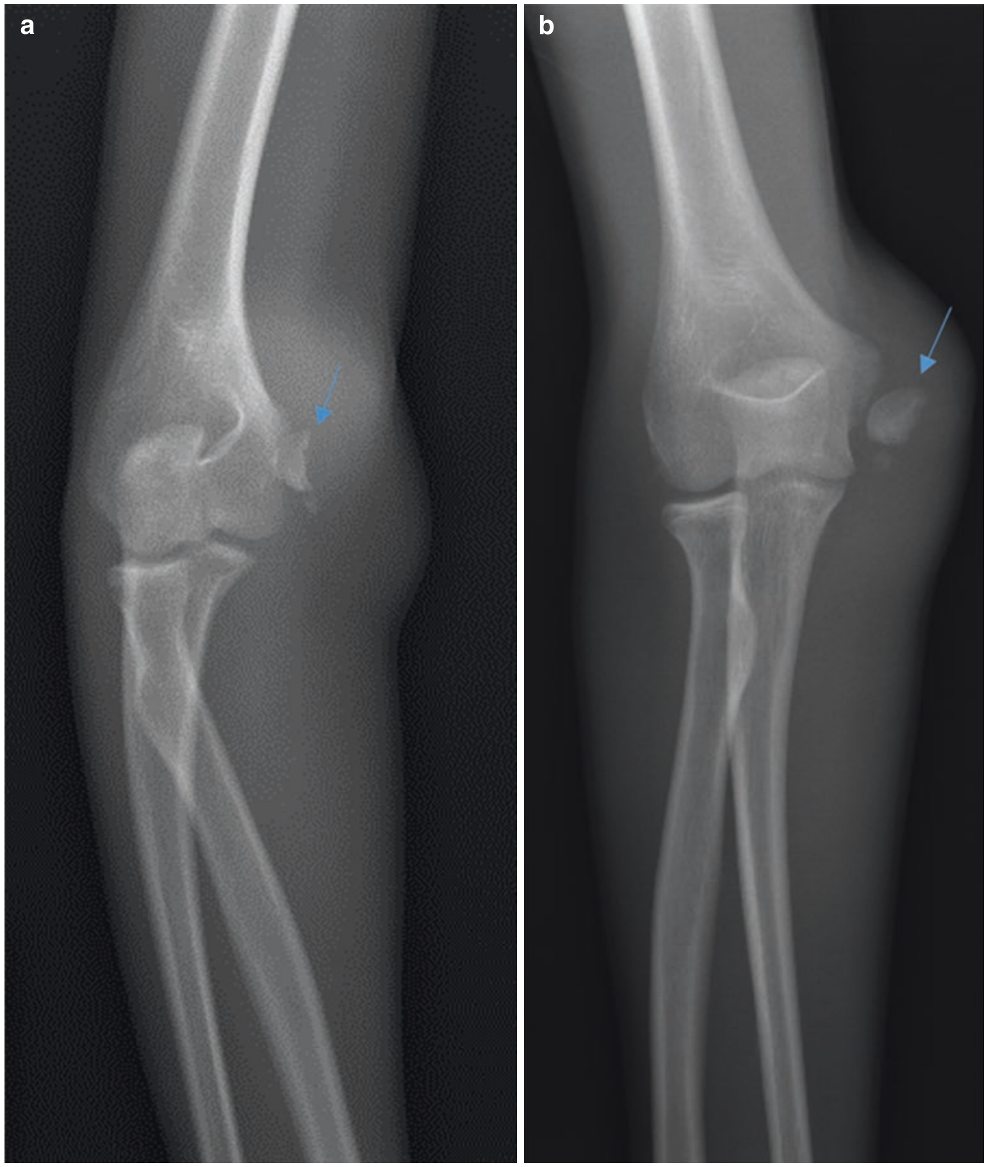


Fig. 16.29 Displaced fracture of the medial epicondyle of humerus (arrows) in a 15-year-old male (**a**, **b**)

Capitellar Fracture

Mechanism of Injury

This injury usually results from a fall on outstretched hand, causing an axial load on the elbow.

Epidemiology

Fractures of the capitellum are relatively uncommon, but can occur in children. These represent less than 1% of pediatric elbow fractures [45].

Clinical Presentation

Delay in diagnosis can lead to worse outcomes. Examination will reveal pain and often demonstrates limited range of motion with flexion/extension and may include swelling.

Imaging

Standard plain radiographs (AP, lateral, and oblique) should be obtained for initial evaluation (Fig. 16.31).

Classification

Three main types of fractures are present: anterior shear injuries (type I), posterior shear injuries (type II), and acute chondral shear injuries (type III) [46]. Type II injuries are often associated with elbow dislocation. These can be further divided into "type a" (nondisplaced) and "type b" (displaced) fractures (Fig. 16.32). CT or MR imaging may be helpful to further evaluate and classify Type I and II injuries. Type III fractures are best diagnosed with MRI since they represent chondral damage [46].

Fig. 16.30 Nondisplaced fracture of the medial epicondyle of humerus with subtle lucency (arrow)

Fig. 16.31 Displaced fracture of the capitellum (arrow) in a 5-year-old boy

Initial Management

Type Ia and IIa injuries can be treated nonoperatively with long arm splint or cast immobilization [45].

Indications for Referral

Type Ib and IIb injuries are best treated with open reduction and internal fixation (ORIF). Type III injuries are rare but surgical treatment with loose-body excision (arthroscopic or open) +/−microfracture has shown good results in small numbers of patients [46].

Follow-Up Care

Nonoperative fractures can often be immobilized for 3–4 weeks, with progression to range-of-motion exercises [45].

Radial Head Subluxation (Nursemaid's Elbow)

Mechanism of Injury

This injury most often happens when a child is lifted or swung by the arm which causes sudden traction. The annular ligament, which holds the radial head to the ulna, slips in between the radius and capitellum, allowing the radial head to sublux.

Epidemiology

Nursemaid's elbow is one of the most common elbow injuries in young children. It usually occurs between ages 1 and 4, with peak onset at age 2–3 [47].

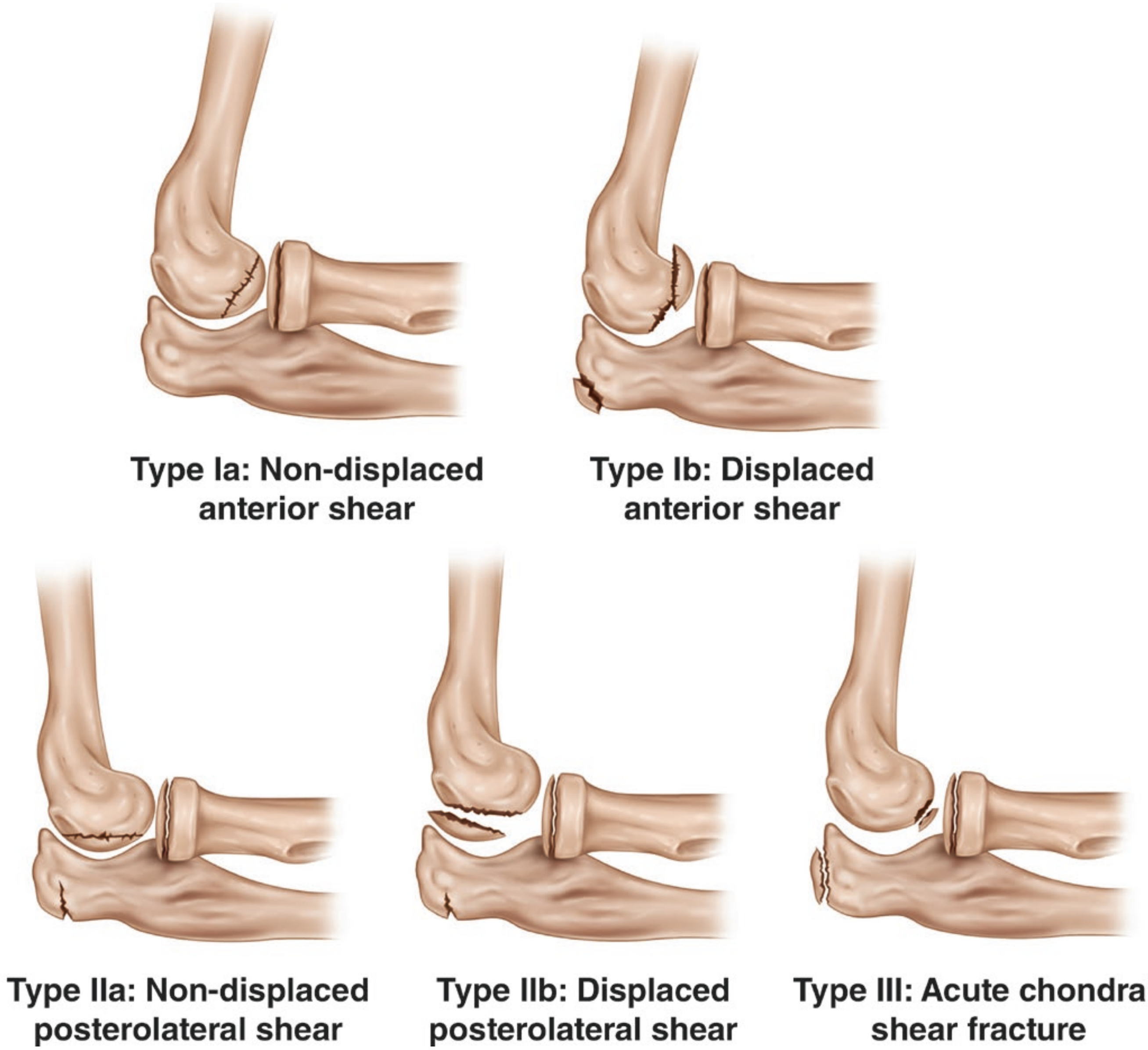


Fig. 16.32 Classification of pediatric capitellar fractures

Clinical Presentation
The child is often in significant pain and will not move the affected arm. Pain is usually most exacerbated by supination of the forearm.

Imaging
If the mechanism is classic for this injury, radiographs may not be needed. Radiographs are indicated if there is no traction mechanism, reduction is unsuccessful, or the examination is concerning (deformity, swelling, neurovascular compromise, etc.).

Initial Management
Fortunately radial head subluxation can be easily and quickly treated. Reduction is most commonly achieved by supination and flexion of the forearm (Fig. 16.33). A "pop" and release of resistance usually signifies a successful reduction. The child will then return to normal use of the arm almost immediately. If this is not the case, re-evaluation with possible radiographs may be warranted [17].

Articular Cartilage Injury

Mechanism of Injury

Isolated articular cartilage injuries of the elbow are rare. Chondral injuries can occur with significant elbow trauma, such as dislocation or fractures extending to the articular surface. A common sports-related isolated articular cartilage

Fig. 16.33 Reduction technique for Nursemaid's Elbow. (**a**) Elbow is extended with forearm pronated. (**b**) Forearm is then supinated, while pressure is applied over the radial head. (**c**) Elbow is maximally flexed with continued pressure over the radial head. A palpable click is often present with successful reduction

disorder of the elbow is osteochondritis dissecans (OCD). OCD is an acquired idiopathic condition involving the articular cartilage and subchondral bone and affects primarily young, active adolescents.

Epidemiology

OCD has been well described in joints other than the elbow including: the knee, ankle, hip, and shoulder [48]. OCD of the elbow typically affects the capitellum of the humerus and most often presents in adolescent athletes engaged in repetitive overhead (baseball, tennis, volleyball) or upper extremity weight-bearing activities (gymnastics, weight-lifting). Boys are more commonly affected than girls, and the dominant elbow is most frequently involved; however, bilateral involvement can occur in up to 20% of patients [49].

Elbow OCD must be distinguished from Panner's disease (osteochondrosis of the capitellum). Panner's disease occurs in a younger child (typically age 4–12) and is a self-limiting, benign condition that resolves with rest. In contrast, OCD can be a progressive lesion and may require surgical intervention.

Clinical Presentation

Delay in diagnosis is very common in elbow OCD; therefore, treating physicians should have a high level of suspicion when treating adolescent athletes with elbow pain. The typical patient is a young male overhead athlete presenting with pain, swelling, and tenderness over the lateral elbow [50]. In the late stages of the process, patients can present with loss of terminal extension and mechanical symptoms (catching, locking) with elbow motion. In the early stages of OCD, physical examination findings may not be distinct.

Imaging

Plain radiographs (AP, lateral, and oblique) should be obtained as an initial screening tool; however, radiographs will often be normal in the early stages of OCD. Approximately one half of the radiographs obtained in patients with an elbow OCD are normal [51]. In later stages of OCD, plain radiographs may show the flattening of the capitellum, a focal defect of the articular surface, and/or loose bodies. Because of the low sensitivity of plain radiographs in detecting elbow OCD, advanced imaging is often necessary. MRI or CT should be utilized if there is any concern for elbow OCD. MRI is helpful in demonstrating early OCD and helps determine the stability of the fragment [52, 53]. CT has also been shown to have a high sensitivity for detecting elbow OCD and may be better than MRI at depicting loose bodies [49].

Initial Management

The choice of conservative versus operative treatment is based on severity of symptoms and characteristics of the OCD lesion (size, location, stability). Stable lesions may be reversible and typically warrant an attempt at nonoperative management. Stable lesions are most often characterized by an open capitellar physis, flattening or radiolucency of subchondral bone on radiographs, and normal range of motion [54]. In contrast, unstable lesions are characterized by a closed capitellar physis, fragmentation on radiographs, and restriction in range of motion [54]. A period of rest with cessation of repetitive stress on the elbow is the mainstay of conservative treatment. Nonsteroidal anti-inflammatory medications can be used. Success of conservative treatment is primarily related to stability of the lesion. Healing potential of OCD is high if rest is used in patients with open capitellar growth plates [55]. Conversely, healing potential with rest is extremely low in advanced OCD lesions in patients with closed growth plates [55–57].

Classification

There are several classification systems described for elbow OCD.

Minami et al. described a classification system based on plain radiographs as follows [58]:

- Grade 1 – stable lesion with translucent cystic shadow in the capitellum
- Grade 2 – clear zone present between the OCD and adjacent subchondral bone
- Grade 3 – presence of loose bodies

Itsubo et al. described a classification system based on T2-weighted MRI findings that focuses on stability of OCD [59]. Stages 1 and 2 are considered stable. Stages 3–5 are considered unstable.

- Stage 1 – normally shaped capitellum with spotted areas of high intensity signal. Signal intensity of spotted areas is less than that of the articular cartilage.
- Stage 2 – Spotted areas as with stage 1 but signal intensity is higher than that of the articular cartilage.
- Stage 3 – presence of discontinuity and noncircularity of the chondral surface of the capitellum. No high signal interface present between the lesion and the floor.
- Stage 4 – lesion separated by a high intensity line.
- Stage 5 – displaced capitellar lesion or defect noted.

Lastly, the International Cartilage Repair Society described a classification system for capitellar OCD that is based on arthroscopic findings [60]:

- Grade 1 – stable lesion with continuous but softened area covered by intact cartilage
- Grade 2 – lesion with partial discontinuity but stable when probed
- Grade 3 – lesion with complete discontinuity but not displaced
- Grade 4 – lesion with loose fragment within the bed or a defect from a displaced fragment

Indications for Referral

Surgical treatment is recommended for OCD lesions that do not respond to nonoperative treatment and for unstable lesions [61]. Numerous arthroscopic and open surgical techniques to address capitellar OCD have been described and are beyond the scope to this text. In general, studies have shown satisfactory results with operative treatment resulting in decreased pain, high level of return to sports, and improved elbow function [61–66].

Follow-Up Care

Patients can be followed at various intervals during their rest period to ensure clinical healing of a nonoperative OCD lesion. If symptoms do not improve or worsen, repeat advanced imaging may be needed.

Complications

If athletes do not undergo a period of rest to allow for healing, an initially stable lesion could progress to an unstable lesion. This may require surgery or could cause permanent problems with elbow function.

Pediatric Considerations

It is important to emphasize again that elbow OCD must be distinguished from Panner's disease (osteochondrosis of the capitellum).

Return to Play

An elbow fracture or dislocation will often be a season-ending injury due to the extended course of treatment and rehabilitation that is required. After initial immobilization, 1–3 months of further rehabilitation are often needed to regain normal function of the elbow. Simple dislocations or fractures should ultimately do well with early range-of-motion and progressive rehabilitation; however, more complex fractures or dislocations may not allow return to previous level of sport.

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Radius and Ulna

17

Emily A. Sweeney, Andrew John Maxwell Gregory, Jessica J. Wingfield, and Frank Scott

Key Points

- Fractures of the shaft of the radius and ulna are less common than distal radius and ulna fractures and are more likely to be unstable.
- Radius and ulna fractures are very common in pediatric patients. They typically heal well, but displacement and angulation may require closed reduction or surgery.
- Radiocarpal dislocations are extremely rare in sports and typically only occur with polytrauma.

Introduction

The radius and the ulna are commonly fractured in sports, often due to falls onto an outstretched hand (FOOSH). The radius and ulna are connected proximally by the annular ligament and elbow capsule. The capsule of the wrist, triangular fibrocartilage complex (TFCC), dorsal radioulnar ligament, and volar radioulnar ligament connect the radius and ulna distally. In between the radius and ulna is the interosseous membrane which provides significant stability between the two bones. The unique nature of the forearm allows the radius to roll over the ulna, allowing for pronation of the wrist. In addition, the radius has an anatomic bow which allows for this movement as well as to allow grip strength.

Midshaft Forearm Fractures

Fracture Classification

Forearm fractures are classified based on a number of descriptors of the fracture: closed vs open, radius vs ulna fracture or both bone fractures, location of the fracture, fracture pattern (e.g., greenstick, oblique, transverse, comminuted), presence or absence of instability at the wrist or elbow, and presence or absence of angulation and displacement [1]. Galeazzi and Monteggia fractures are classified separately as fracture-dislocations (see Chap. 16 for details on Monteggia fractures). Galeazzi fractures, sometimes referred to as reverse Monteggia fractures, are fractures of the radial shaft with disruption of the distal radioulnar joint (DRUJ) [2]. An Essex-Lopresti fracture is a radial head fracture with interosseous membrane rupture and DRUJ dislocation due to proximal migration of the radius [2].

Mechanism of Injury in Sports

Forearm fractures may occur due to an axial compression such as from a FOOSH, bending of the forearm, rotation of the forearm, or from direct trauma [3]. Most forearm fractures are due to a high-energy mechanism, such as a fall from height or motor vehicle accident. In sports, athletes in football and wrestling are most likely to have forearm fractures with an incidence of 0.48 and 0.21 per 10,000 high-school athletic exposures, respectively [4]. Galeazzi fracture-dislocations typically occur due to a fall on an extended wrist with a hyperpronated forearm [5].

E. A. Sweeney (✉)
Department of Orthopedics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO, USA
e-mail: Emily.sweeney@childrenscolorado.org

A. J. M. Gregory
Department of Orthopedics, Vanderbilt University Medical Center, Nashville, TN, USA

J. J. Wingfield
Department of Orthopedics, Hand Surgery, University of Colorado School of Medicine, Denver, CO, USA

F. Scott
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA

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Epidemiology

It is estimated that 10–14% of all fractures occur in the forearm [3]. Diaphyseal forearm fractures are much less common than distal forearm fractures. They are most common in 10–20-year-old males and in females over age 60 years [6]. Galeazzi fracture-dislocations account for 7% of adult forearm fractures [2].

Clinical Presentation

Patients typically present with pain and swelling. Patients with a displaced or angulated fracture may have deformity noted on clinical examination. Clinicians should also evaluate for soft tissue injuries such as lacerations, contusions, neurovascular injury, and open fractures. A patient with an associated DRUJ disruption may have a swollen, tender wrist with limited pronation and supination [2]. If a patient has numbness or tingling along the radial or palmar aspect of the hand, a median nerve injury should be suspected. In comparison, a radial nerve injury will affect the posterior forearm and dorsal aspect of the radial side of the hand. The posterior and anterior interosseous nerves may be injured if the interosseous membrane is disrupted. The posterior interosseous nerve supplies motor innervation for the wrist and finger extensors including the extensor digitorum, extensor indicis, and extensor carpi ulnaris. The anterior interosseous nerve supplies motor function to the flexor pollicis longus, lateral half of the flexor digitorum profundus, and pronator quadratus. With an Essex-Lopresti injury, patients have ulnar-sided wrist pain with a radius fracture. They may also have decreased forearm rotation and DRUJ instability and positive ulnar variance on radiographs.

Imaging

Initial imaging for a suspected forearm fracture includes anteroposterior and lateral radiographic views of the forearm (Fig. 17.1). If the patient has tenderness at the wrist or elbow, radiographs of these joints should also be obtained. Clinicians should closely evaluate the DRUJ in patients with a radial shaft fracture for possible Galeazzi fracture-dislocation. If the patient's radiograph shows an ulnar styloid fracture, widening of the DRUJ on the PA view, dorsal displacement of the ulna on the lateral view, or ulnar positive variance of >5 mm compared to the unaffected side, there should be a high suspicion for injury to the DRUJ [1]. Radial head dislocation indicates a Monteggia fracture-dislocation. MRI is occasionally needed if there is suspicion of damage to the TFCC, interosseous membrane, or to the articular cartilage [1–3]. CT may be needed for surgical planning or to evaluate for nonunion.

Initial Management

On the sideline, the patient's arm should be splinted, taking care to immobilize the elbow at 90°, the forearm in neutral, and the wrist in a neutral or slightly extended position. Neurovascular status should be evaluated. In the clinic or emergency room, forearm fractures without displacement should be initially treated with a sugar-tong or long arm splint. Once swelling has improved after 1–2 weeks, the patient should be placed in a long arm cast with the forearm in neutral or slightly supinated to reduce the forces of the supinator and biceps brachii muscles [7]. Isolated ulnar shaft fractures (Fig. 17.2) can be treated with a long arm posterior splint if there is >50% overlap of the fracture fragments, <10° of angulation, and no radial head dislocation and the fracture is in the distal two-thirds of the ulna [1]. The splint should hold the elbow at 90° with the forearm in neutral. Fractures of the proximal third ulna typically require surgery. A 2012 Cochrane review found that there is insufficient evidence for the best method of treatment for isolated ulnar shaft fractures in adults [7]. Galeazzi fractures require ORIF in adults; however, closed reduction may be attempted in pediatric patients [8]. Patients with isolated radial shaft fractures should be placed in a double sugar-tong splint [1]. Occasionally, both bone forearm fractures can be treated without surgery. Initial treatment should include a well-molded, long arm cast that is bivalved.

Indications for Urgent and Nonurgent Orthopedic Referral

Closed reduction of forearm fractures in adults typically results in poor outcomes as patients may develop improper rotational alignment, DRUJ instability, or unstable fractures even when casted [1, 2]; therefore, many forearm fractures including isolated ulnar shaft and radial shaft fractures as well as both bone forearm fractures require referral to orthopedics [3, 9]. Pediatric patients, however, can frequently be managed with nonoperative care (see Pediatric Considerations). Patients with an open fracture or signs of vascular insufficiency require emergent orthopedic referral. Neurologic symptoms also require urgent referral to orthopedics. Adults who have Galeazzi or Essex-Lopresti fracture-dislocations displaced fractures, or fractures with >10° of angulation will likely need surgical fixation with an urgent referral to orthopedics. Plates, screws, nails, bone

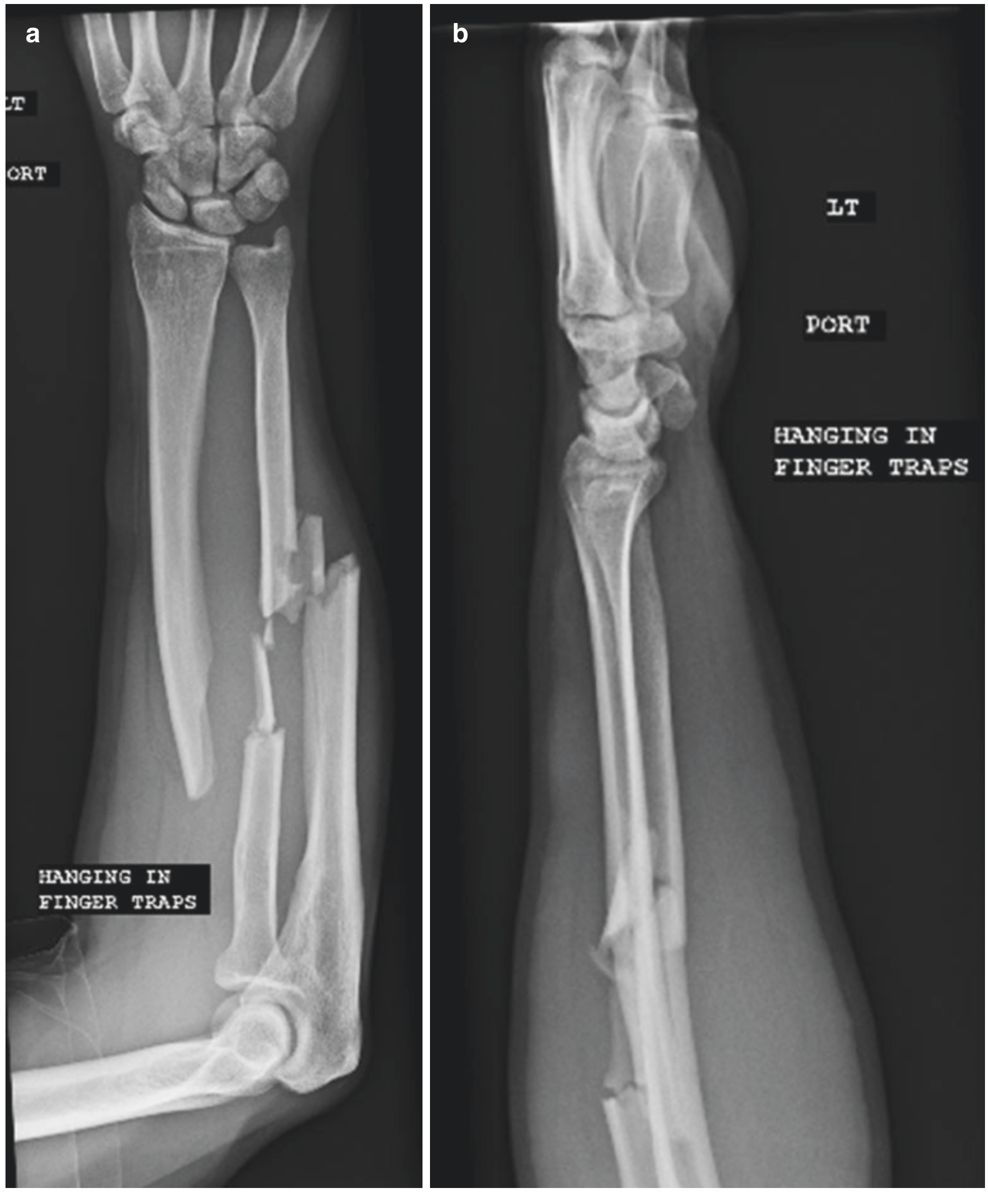

Fig. 17.1 Both bone forearm fractures (**a**, **b**) in a 49-year-old male after hitting a tree while skiing

grafting, and external fixators may be used, but the specific methods for surgical fixation are beyond the scope of this text [3, 8].

Follow-Up Care

Adult patients with forearm fractures who are able to be managed without surgery should be followed with weekly X-rays, especially in the first 2–4 weeks, to ensure stable position of the fracture [3]. Patients should be splinted for 7–10 days. They should then be transitioned to a long arm cast that extends from the deltoid insertion on the humerus to the MCP joints for 2–4 weeks. Sometimes, a bivalve cast is needed for the first 2 weeks if the patient has significant swelling [3]. The patient should then be transitioned to a functional long arm or forearm brace for 4–6 weeks (for a total of 8 weeks) or until radiographic healing is achieved (Table 17.1) which may take 12–16 weeks [1, 10]. The brace should allow full flexion and extension of the elbow and wrist as well as full pronation and supination. Adult patients typically will require PT to regain full motion and strength, as well as to progress back to sports. PT should start once they are in the brace. Patients who develop increased angulation or displacement during their care should be referred to orthopedics.

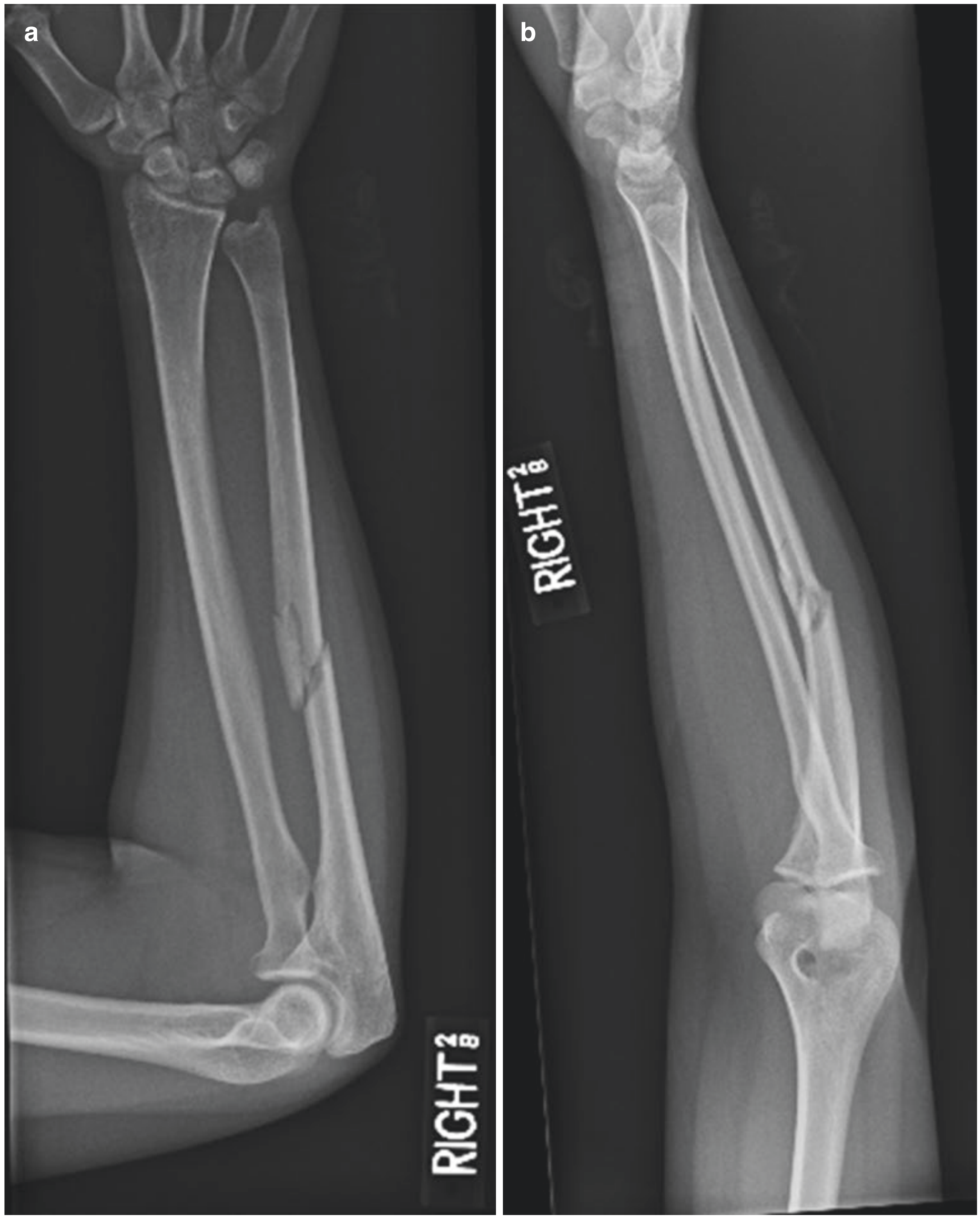

Fig. 17.2 Ulnar shaft fracture (**a**, **b**) with angulation in a 24-year-old female who fell while skiing

Table 17.1 Treatment of radius and ulna fractures

Fracture type	Initial treatment	Further treatment	Total length of immobilization	Return to sport
Isolated radial or ulnar shaft fracture	Sugar-tong or long arm splint	Long arm cast/splint for 2–4 weeks, then functional brace	8–12 weeks	8–16 weeks
Both bone forearm fractures	Sugar-tong or long arm splint	Long arm cast/splint for 2–4 weeks, then functional brace	8–12 weeks	8–16 weeks
Distal radius and ulna fractures	Sugar-tong splint	Short arm cast for 4–6 weeks followed by removable brace	6–8 weeks	8–12 weeks

Return to Sports

After casting is complete, adult patients with forearm fractures will require physical therapy (PT) to regain range of motion and strength of the arm. Return to sport typically requires 12–16 weeks; however, athletes who do not require excessive upper extremity strength (e.g., cross-country) can often return in 8–12 weeks. The specific time to return to sports often depends on the type of injury, specific sport, and evidence of radiographic healing [3]. Pediatric patients typically heal faster and return to sports faster than adults with forearm fractures.

Complications

Disruption of the distal radioulnar, proximal radioulnar, or radiocapitellar joints may occur in forearm fractures. If not addressed, patients typically have poor outcomes [2]. It is important to ensure the radius maintains its anatomic bow as more than a 5% change can result in a loss of forearm rotation and grip strength [8]. DRUJ instability is a common complication after forearm injuries. Nerve laceration or entrapment sometimes occurs days to weeks after the injury and, if suspected, should be evaluated by MRI or US [1, 11]. Other complications include myositis ossificans, malunion and nonunion (2–10%), and radioulnar synostosis (1–6%) [1, 3]. Joint stiffness is a common complication, especially with prolonged immobilization of the elbow.

Pediatric Considerations

Midshaft forearm fractures are common in children and are very often associated with significant deformity. Just like distal forearm fractures, these fractures can often be reduced in a closed fashion. However, angulation is not as well tolerated in the midshaft as the remodeling potential is less as the fracture is farther away from the physis. In addition, these fractures are often very unstable and difficult to keep aligned even in a long arm cast. They should be monitored weekly with X-rays until callous becomes visible. If alignment cannot be maintained in a long arm cast, they may require internal fixation (Fig. 17.3). Often these fractures take longer to heal than those of the distal radius and can take up to 3–4 months. Galeazzi fractures are rare in children. Closed reduction of the fracture usually leads to reduction of the corresponding joint dislocation. If closed reduction is not successful, then open reduction with internal fixation is indicated. PT is usually not required after treatment as pediatric patients regain motion and strength pretty quickly. Occasionally pediatric patients will get elbow stiffness and require PT and dynamic bracing.

Distal Radius and Ulna Fractures

Classification

Multiple methods have been used to categorize distal radius fractures. Classifications such as the Melone, Fernandez, and Frykman systems have all been proposed. The Melone system (Table 17.2) describes injury patterns of intra-articular distal radius fractures by their mechanism and severity of injury [12]. The Fernandez classification scheme (Table 17.3) recognizes five different patterns of injury based on their mechanism of injury and radiographic fracture pattern produced [13–15]. Lastly, the commonly cited Frykman classification (Table 17.4) describes wrist fractures based on the radiographic appearance of the radius and the presence or absence of an associated distal ulna fracture [14, 15]. This comprehensive classification system includes extra-articular and intra-articular fractures (Fig. 17.4) [15].

In addition to formal classification systems, there are numerous eponyms that have historically been used to describe various fracture patterns. A Smith's fracture describes an extra-articular distal radius fracture with volar displacement of the distal fragment and apex dorsal angulation [13, 14]. Barton fractures (Fig. 17.5) are a related injury where there is extension of the fracture line into the articular surface [13, 14]. In this injury pattern, there is also volar displacement of the distal fracture fragment. The carpus tends to displace volarly along with the fracture fragment and is characteristically an unstable fracture pattern. Chauffer fractures are intra-articular distal radius fractures where the primary fracture fragment is composed of the radial styloid [13, 14]. This term was initially used to describe a unique fracture that occurred in individuals injured while handling a car starter that backfired [14]. Chauffer fractures are also known for their association with concomitant injuries of the scapholunate ligament [2]. Perhaps the most well-known eponym for wrist injuries is the Colles' fracture (Fig. 17.6). The term is classically used to describe an extra-articular distal radius fracture with dorsal tilt or apex volar angulation. Despite the number of eponyms and classification systems that have been described here, many others remain important and are used to define the nature of distal radius and ulna fractures in clinical practice.

Mechanism of Injury

The predominant mechanism of injury that produces a distal radius or ulna fracture is a FOOSH from a standing position. With the wrist often held in an extended position, the distal radius is then forcefully driven into a hyperextended position producing a dorsally displaced fracture with apex volar angulation most frequently [16]. In the older adult, this is sometimes the result of postural instability while ambulating or standing [16]. This low-energy mechanism produces a fracture secondary to underlying osteopenia or osteoporosis. In the adolescent or young adult, distal radius fractures are frequently the result of high-energy injuries such as a motor vehicle accident or in the setting of athletics [13, 16, 17].

Epidemiology

In the United States, annually greater than 640,000 distal radius fractures are diagnosed [14]. A bimodal distribution is

Fig. 17.3 An 11-year-old male who had a FOOSH injury when he fell from the top of a slide (**a**, **b**). After closed reduction, he had good alignment of his fractures (**c**, **d**). However, 1 week later, the reduction was lost (**e**, **f**). He eventually had surgical reduction with fixation

Table 17.2 Melone fracture classification of intra-articular distal radius fractures [12]

	Description of radius fracture
Type I	Stable fracture, no displacement
Type II	"Die punch" fracture, unstable IIA: reducible IIB: irreducible
Type III	"Spike" fracture with radial shaft projecting or spiked into the flexor compartment, unstable
Type IV	"Split" fracture with severe disruption of radial articular surface, unstable, severely comminuted
Type V	Explosion injury due to compression and crush injury leading to severe comminution

Table 17.3 Fernandez fracture classification [13–15]

	Description	Treatment options
Type I	Bending fracture of metaphysis	Cast if fracture is stable, percutaneous pinning or external fixation if unstable
Type II	Shearing fracture that extends to radiocarpal joint	Open reduction or screw fixation
Type III	Compression fracture that extends to radiocarpal joint	Typically require surgery with percutaneous pinning
Type IV	Avulsion fracture that extends to radiocarpal joint with dislocation	Closed or open reduction, may require pin or screw fixation
Type V	Combination of above types	Surgery based on fracture appearance

Table 17.4 Frykman fracture classification [15]

	Radius	Ulna
Frykman I	Extra-articular radius fracture	No ulna fracture
Frykman II	Extra-articular radius fracture	Distal ulna fracture
Frykman III	Intra-articular radius fracture of radiocarpal joint	No ulna fracture
Frykman IV	Intra-articular radius fracture of radiocarpal joint	Distal ulna fracture
Frykman V	Fracture extends to radioulnar joint	No distal ulna fracture
Frykman VI	Fracture extends to radioulnar joint	Distal ulna fracture
Frykman VII	Fracture extends to radiocarpal and radioulnar joint	No distal ulna fracture
Frykman VIII	Fracture extends to radiocarpal and radioulnar joint	Distal ulna fracture

found, with the initial peak in the young male demographic, often secondary to injuries sustained during sports. The average age for those injured during an athletic activity has been reported to be less than 40 years of age, with 72% of distal radius fractures occurring in males [17]. A second peak is seen in the elderly female population and are often considered fragility fractures. Wood et al. reported on a large series of 408 adolescent patients who sustained various upper and lower extremity fractures [18]. In their review, 23.9% of adolescent fractures occurred during a sporting event. Of these, 83.6% of injuries were isolated to the upper extremity, with 23% of the total injuries accounted for by distal radius and or ulna fractures [18]. In a similar review of 5953 adult fractures, a 12.8% prevalence of sports-related injuries was found [19]. In this same review, distal radius fractures occurred during a variety of sporting activities including soccer, rugby, skiing, and basketball most frequently [19].

Fig. 17.4 Frykman II distal radius fracture

Fig. 17.5 Barton's fracture with volar displacement of the distal fracture fragment

Fig. 17.6 Colles' fracture with dorsal tilt on lateral X-ray

Clinical Presentation

After a distal radius fracture, the patient typically presents with the acute onset of pain following an identifiable traumatic event. Injuries can be produced from a low-energy mechanism such as a fall from a standing position or low height. Alternatively, a high injury mechanism such as a collision during a sporting event or a fall onto an outstretched arm while running at high speed can produce wrist injuries (Fig. 17.7). In patients with marked displacement of a fracture fragment, an obvious extension or flexion deformity may be visible (Fig. 17.8). A comprehensive physical examination including an assessment of the soft tissues for abrasions or lacerations should be undertaken. The soft tissues should be carefully inspected circumferentially to rule out of the possibility of an open fracture, particularly in high-energy injuries. A thorough skin examination is critical as some open injuries including distal ulna fractures may present with only a poke hole with subtle venous bleeding [14]. A vascular examination should be performed to assess the perfusion of the hand including an evaluation of the radial and ulnar pulses as well as capillary refill in the fingers. A neurologic examination should be performed to assess for a sensory or motor deficit in the hand. Compartment syndrome is rare in the setting of a

distal radius fracture and can be diagnosed with a detailed physical examination. Acute carpal tunnel syndrome is an uncommon occurrence, but is an important consideration in the clinical evaluation of a patient with a distal radius fracture. The acute onset of paresthesias in the thumb, index, long, and ring finger can alert the clinician to the potential of median nerve compression secondary to fracture displacement or a hematoma [14]. If symptoms are not immediately improved following a bedside reduction, an urgent orthopedic evaluation is warranted. Suspicion for compartment syndrome or median nerve compression is particularly relevant in the evaluation of wrist fractures secondary to a high-energy mechanism.

Imaging

Plain radiography is an essential component in the evaluation of distal radius and ulna fractures. In patients with point tenderness or clinical deformity evident on examination, three views of the affected wrist should be obtained [20]. A quality anterior posterior (AP), oblique, and lateral view all are necessary for the accurate assessment of distal radius fractures. Dedicated imaging of the hand should be considered if point tenderness is present within the hand or fingers. Similarly, proximal imaging is warranted to evaluate the forearm or elbow if clinical concern exists for an associated injury. X-rays are usually the only imaging modality

Fig. 17.7 Mild swelling in the distal dorsum of the radius (**a**, **b**) in a 36-year-old male as a result of an injury (FOOSH) while mountain biking. X-rays reveal intra-articular and non-displaced distal radius fracture (**c–e**)

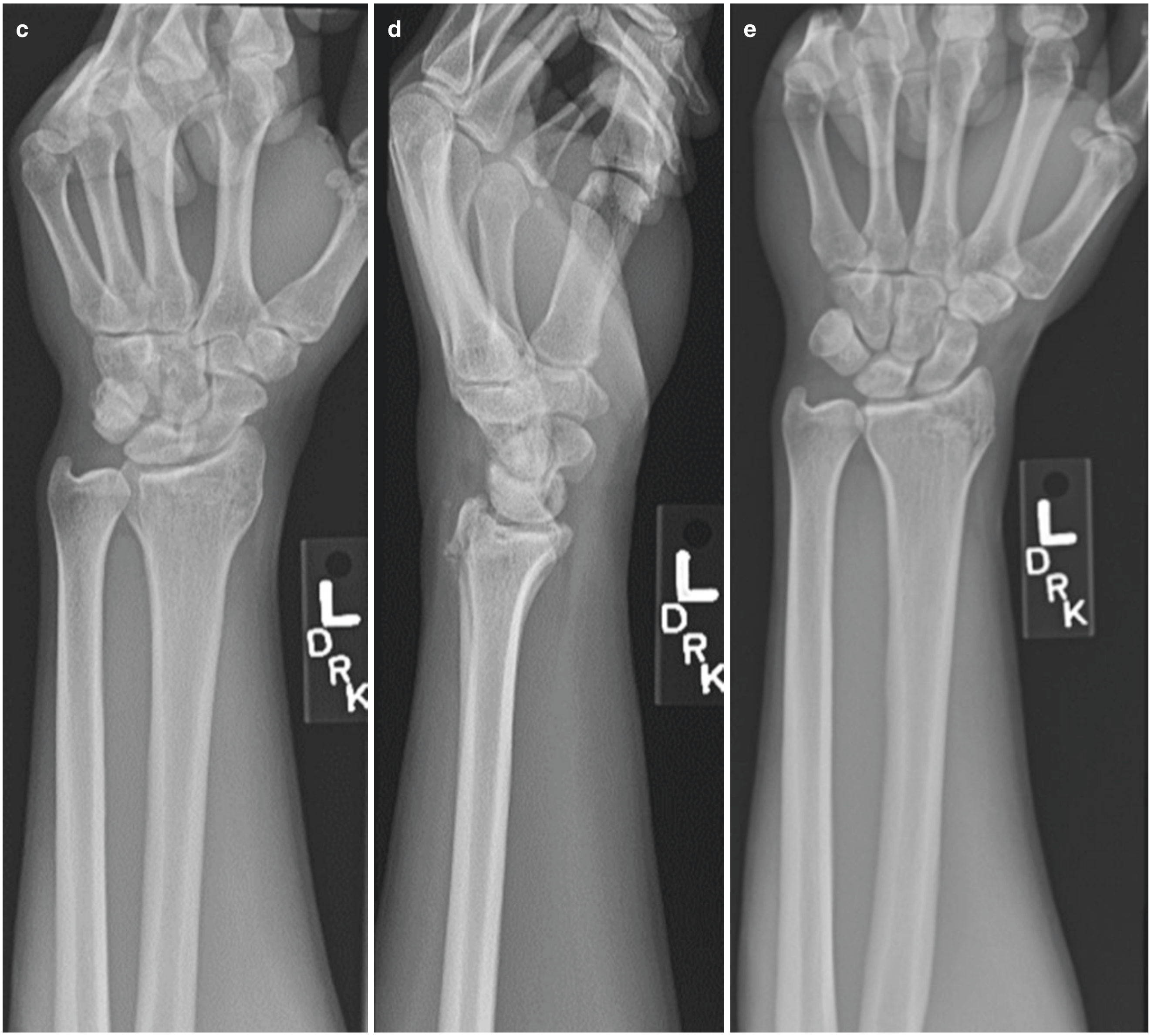

Fig. 17.7 (continued)

necessary for the evaluation of distal radius or ulna fractures. Quality radiographs allow for the evaluation of articular involvement and radiocarpal joint congruity, as well as displacement in both the coronal and sagittal planes (Fig. 17.9).

While distal ulna fractures rarely occur in isolation in the adult patient, distal ulna fractures including ulnar styloid avulsions are commonly associated with distal radius fractures [21]. Ulnar styloid fractures (Fig. 17.10) may represent underlying instability of the distal radioulnar joint or associated triangular fibrocartilage complex injuries [21].

Occasionally, CT is utilized to better define articular involvement; however, this is rarely needed prior to orthopedic referral [13]. MRI can be a useful adjunct in select scenarios, such as the evaluation of suspected scapholunate ligament injuries in radial styloid fractures if clinical concern exists.

The role of ultrasound is evolving in the diagnosis and treatment of distal radius and ulna fractures. The use of portable ultrasound has been reported with a 100% sensitivity and 90–95% specificity for the diagnosis of distal radius fractures [22]. A proposed benefit of ultrasound is the portability of devices outside of the hospital setting, potentially expanding its role to sideline use in the screening of athletes for fractures [22]. An additional utility of ultrasound is its role in assisting with the confirmation of fracture reduction. Lau et al. reported 76–93% sensitivity and 93–94% specificity in the identification of successful closed reduction of distal radius fractures when compared to conventional

Fig. 17.8 A dinner-fork deformity (**a**) in a 29-year-old male as a result of an injury (FOOSH) while skiing. X-rays reveal distal radius fracture with posterior displacement (**b**, **c**). His alignment seems to be acceptable after a closed reduction (**d**, **e**)

Fig. 17.9 AP wrist X-ray with distal radius and ulna fractures

Fig. 17.10 AP X-ray demonstrating a distal radius fracture and associated ulnar styloid fracture

radiography [22]. Comparison of fluoroscopic-assisted reduction and ultrasound-assisted closed reduction of distal radius fractures has demonstrated no difference in radiographic outcomes including restoration of volar tilt, radial height, and radial shortening [23].

Initial Management

In the setting of an athletic event, suspected distal radius and ulna fractures can be initially treated with splint application with the forearm and wrist in a neutral position. Obvious deformities can be reduced with gentle traction to restore overall limb alignment. A sugar-tong splint with the elbow in 90° of flexion can be applied. Alternatively, a dorsal and ulnar volar slab splint can be applied (Fig. 17.11).

Prior to splint application, a comprehensive clinical evaluation including an assessment for open fractures and neurovascular injury should be performed. Athletes with suspected wrist fractures should be removed from play until radiographs and a formal clinical examination are completed. After initial splinting or sling application, patients can be evaluated in the emergency room or clinic setting depending on the initial severity of their injury.

In the clinic or emergency room setting, orthogonal radiographs out of splint material should be obtained. If an open fracture is suspected, intravenous antibiotics and indicated tetanus prophylaxis should be promptly administered. Pediatric or adolescent patients with closed fractures should undergo attempted closed reduction maneuvers. In the hospital or urgent care setting, this can be performed after the administration of a hematoma block or light sedation if available. Traction is often needed prior to an attempt for reduction (Fig. 17.12). Immobilization with a fiberglass splint or in some cases with a cast should be applied. If a fiberglass cast is applied in the acute setting, the cast should be valved to accommodate soft tissue swelling. In the adult population,

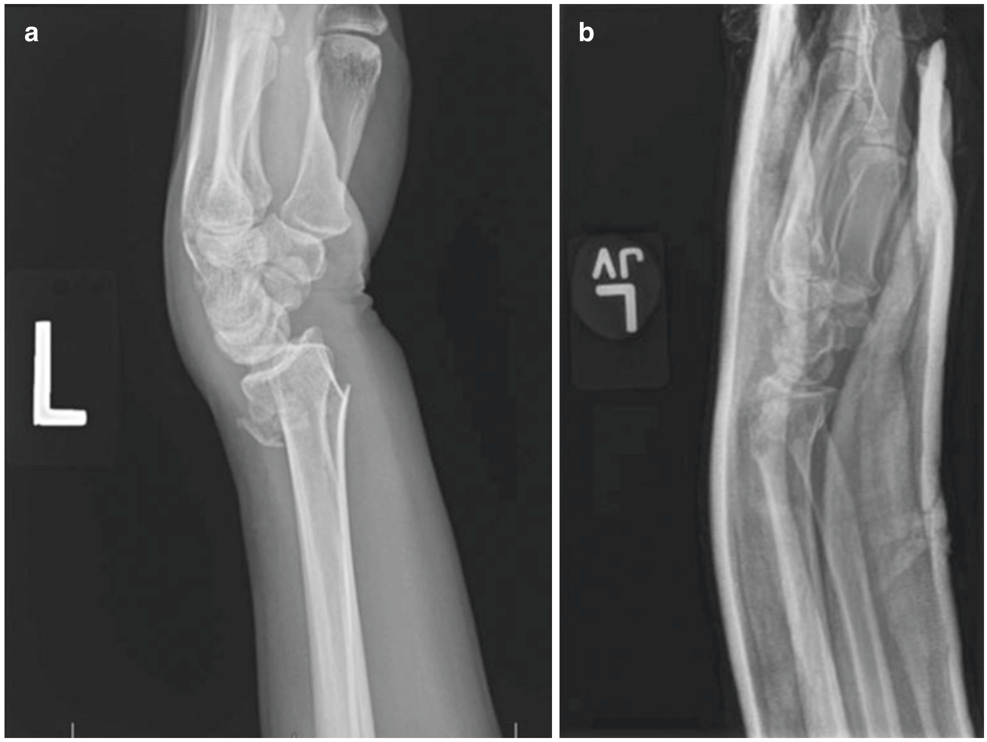

Fig. 17.11 Dorsally displaced distal radius fracture before (**a**) and after (**b**) closed reduction and splint application

initial immobilization is typically accomplished using splints after a closed reduction is performed.

Indications for Urgent and Nonurgent Orthopedic Referral

Non-displaced fractures without articular extension can often be treated nonsurgically with cast immobilization [20]. Although protocols vary, many clinicians treat distal radius fractures amenable to nonoperative treatment with a total of 6 weeks of cast or splint immobilization [20]. Fractures that demonstrate displacement at the time of injury and undergo closed reduction maneuvers are at risk for subsequent displacement with closed treatment. Early orthopedic evaluation within 1 week of injury may be essential to identify patients that would benefit from operative treatment. Certain non-displaced fracture patterns including radial styloid fractures are at particularly high risk of subsequent loss of reduction. Additionally, because of their association with scapholunate interosseous ligament injuries, consideration should be given to early referral of radial styloid fractures. Numerous risk factors have been reported to aid in identifying patients at higher risk for loss of radiographic reduction. These include the initial degree of fracture displacement, the presence of dorsal comminution, and increasing age [13, 14, 20, 24]. Consideration should be given to the presence of these factors when determining if an early orthopedic referral is warranted. Multiple prospective studies have demonstrated improved outcomes with surgical treatment of fractures in non-elderly patients who do not have acceptable radiographic outcomes after a closed reduction attempt [25]. Surgical treatment may be warranted if there is greater than 3 mm of loss of radial height, greater than 2 mm of articular incongruity (Fig. 17.13), or greater than 10° of dorsal tilt on lateral imaging following closed reduction [25]. These patients should be promptly referred for orthopedic evaluation. The evidence for this recommendation is “moderate” based on AAOS guidelines, indicating the benefits exceed the potential risks, but the strength of the supporting evidence is weak [25].

Occasionally an urgent orthopedic consultation is warranted for immediate evaluation. The suspected presence of an open fracture, compartment syndrome, or acute carpal tunnel syndrome is an indication for immediate evaluation by an orthopedic surgeon in the emergency department setting. Additionally, significant fracture displacement with skin tenting and impending soft tissue compromise may warrant urgent orthopedic evaluation if a satisfactory closed reduction cannot be performed.

Follow-Up Care

Patients with distal radius fractures that are amenable to nonsurgical treatment should be followed with weekly X-rays for the first 3 weeks following an injury; however, this evidence is based solely on expert opinion [13, 16, 25]. If a closed

Fig. 17.12 Traction with finger traps and 5–10 kg of weight is often required before close reduction of a displaced distal radius fracture

reduction was initially required, follow-up imaging can be obtained without removal of a splint or cast in order to help prevent loss of reduction. If quality images cannot be obtained through the splint or cast material, it should be removed and X-rays should be repeated. Imaging should be critically assessed to ensure there is no loss of reduction while immobilized. In patients where a gradual or complete loss of reduction is noted, prompt orthopedic referral should be considered to evaluate whether surgical reduction and fixation are warranted. In those initially treated with sugar-tong splint placement, a transition into a short arm cast at 2–3 weeks can be made after swelling has improved. A short arm cast with the wrist in neutral or slight extension should be applied up to the metacarpophalangeal joints. The fingers and thumb should remain free and early finger motion should be encouraged to

Fig. 17.13 AP X-ray of an intra-articular distal radius fracture with articular incongruity

prevent stiffness [14]. A total of 6 weeks of immobilization is commonly recommended (Table 17.1) [16]. Following the completion of immobilization, an examination should be performed to clinically assess for fracture healing by ensuring that tenderness over the fracture has resolved. When the fracture is clinically healed, immobilization is discontinued and the patient is transitioned into a removable brace. Brace wear is discontinued as tolerated by the patient and activity is slowly increased. Those that do not quickly recover wrist and finger motion or strength may be referred for occupational hand therapy to aid in their progress.

Return to Sports

Following nonsurgical treatment of a distal radius fracture, patients are typically advised to avoid contact sports for a minimum of 8–12 weeks after their injury [16]. Patients should demonstrate restoration of near-normal wrist motion and strength, as well as clinical and radiographic evidence of fracture healing prior to return to sports. The specific time to return to sports is dependent on the initial nature of the injury and the sport in which the athlete participates. In contact sports such as football, consideration should be given to delayed return to sport or the transient use of protective braces. A period of 8–9 weeks after initial injury has been reported as an average time period for return to play of athletes following distal radius fractures [26].

Complications

Distal radius fractures treated nonsurgically typically heal, and nonunion of closed distal radius fractures is uncommon [14]. In fractures where loss of reduction is unrecognized or remains untreated, malunion can occur [14, 20]. Fracture malunion most commonly results in a loss of normal volar tilt in the sagittal plane, which clinically can result in the loss of normal wrist flexion. Additionally, radius fractures that heal in a shortened position can develop ulnar positive variance, where the ulna becomes long relative to the radius at the level of the wrist. This may lead to ulnocarpal impingement over time and warrants evaluation in individuals with ulnar-sided wrist pain with a history of a distal radius fracture [16]. If symptomatic malunion occurs, an orthopedic referral may be warranted for consideration of a corrective osteotomy. Articular incongruity may predispose the patient to developing post-traumatic radiocarpal arthrosis. Additionally, long-term adaptive changes can occur in the midcarpal joint as a result of a distal radius malunion.

Transient stiffness is a common complication of both surgical and nonoperative treatment of distal radius fractures. This can be addressed during the treatment period by encouraging early finger motion. After discontinuation of immobilization, individuals may benefit from formal therapy sessions if wrist or finger stiffness is a concern. Tendon rupture is a rare complication, but has been described after nonsurgical treatment of distal radius fractures. Immediate or delayed rupture of the extensor pollicis longus tendon can be seen after a non-displaced distal radius fracture and warrants orthopedic referral for evaluation and treatment [13]. This is addressed most often with an extensor indicis proprius transfer for reconstruction. Ruptures of additional flexor and extensor tendons have been described, but typically occur as the iatrogenic sequela of surgical treatment with plate fixation [14].

Pediatric Considerations

Children are more likely to fracture bones than adults because of their softer bone and the presence of growth plates. Forearm fractures are one of the most common fractures in pediatrics. Fracture patterns in children are different than those in adults. Buckle (torus), greenstick, and physeal fractures are unique to children and are often subtle. Providers must be aware of these patterns and look for them on x-ray.

A buckle fracture (Fig. 17.14) is a compression fracture of one cortex (usually dorsal) at the junction of the metaphyseal and diaphyseal bone. These are very common and heal quickly. A greenstick fracture is a combination of a buckle fracture on one cortex and a break through the opposite cortex (usually volar). This creates a hinge of periosteum that is still intact and stabilizes the fracture (Fig. 17.15). A physeal fracture is a fracture through the physis at the end of the long bone. There are several patterns of physeal fracture that can involve the physis alone or with part of the metaphysis, epiphysis, or both (please refer to the Salter-Harris classification system). Physeal fractures are of particular concern as they can lead to problems of growth with early physeal closure or bridging.

As in adults, a FOOSH is the most common mechanism for sustaining a distal radius or ulna fracture. A child will complain of pain at the distal forearm and will limit use of the arm. Swelling and bruising may not be present. They may have limited supination or flexion and will be tender to palpation on the distal radius and/or ulna. Navicular or scaphoid fractures can happen in children but are less common than adults (see Chap. 18 – Carpus). They should still be considered in the physical examination and appropriate imaging ordered.

As with adults, any child with obvious wrist deformity should be reduced closed and splinted or casted (Fig. 17.16). If closed reduction is not successful or the reduction is lost, open reduction with internal fixation is indicated. In general, it is less common to require open reduction in children because some angulation of the distal radius is acceptable because of the process of remodeling. As the bone lengthens with growth, it straightens itself out. This is more effective with younger age, close proximity to the physis, and angulation in the flexion/extension plane. Angulation of the distal radius up to 25° in children less than ten years old is considered acceptable, whereas angulation over 10° in children over 10 years old is unlikely to correct. Displacement is tolerated much better than angulation. Bayonet apposition

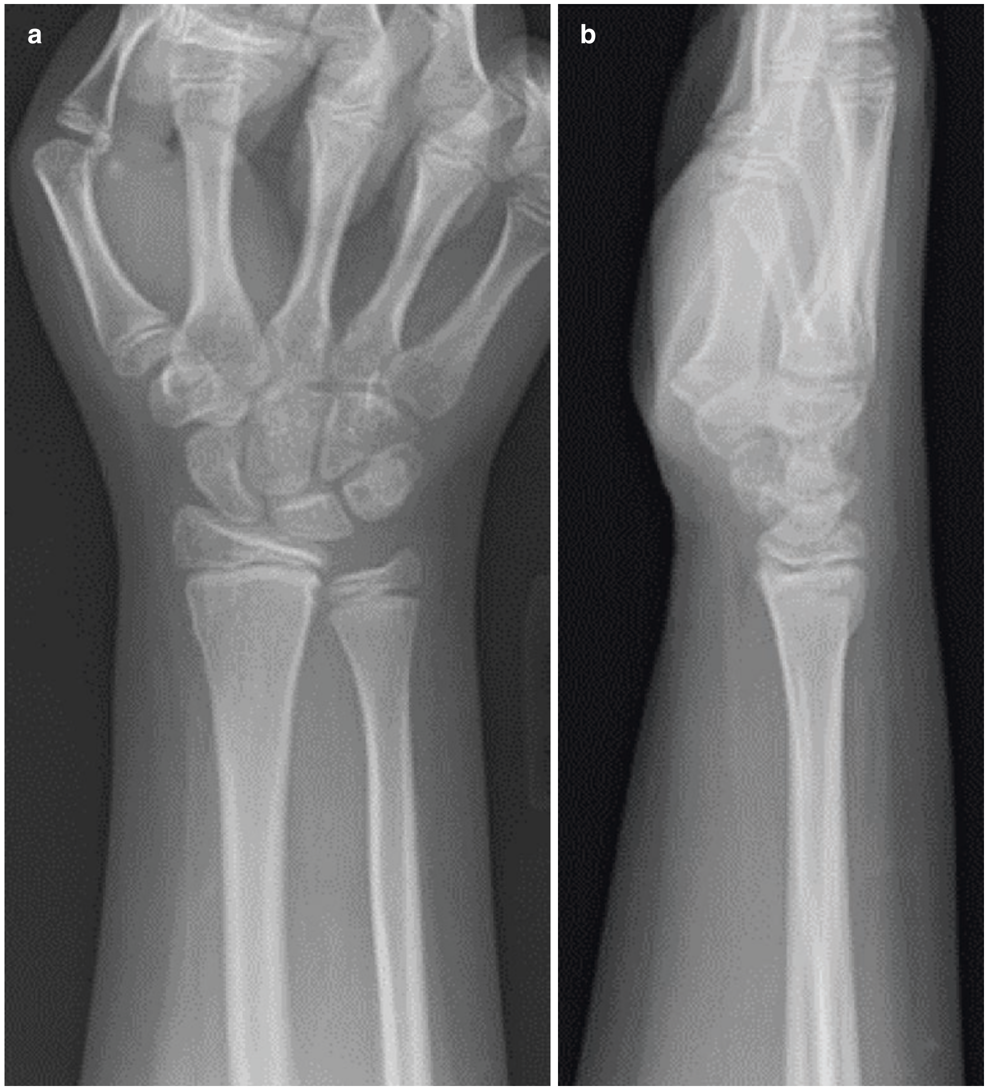

Fig. 17.14 Buckle fracture of the distal radius (**a**, **b**) in a 9-year-old female who tripped and had a FOOSH

(100% displacement with no angulation) is well tolerated and does not require surgery. All deformed distal radius fractures should be evaluated for compartment syndrome and posterior interosseous nerve injury. Distal forearm fractures that are not reduced usually heal in 4–6 weeks, while reduced fractures often take an additional 2 weeks to heal. Traditionally these reduced fractures have been treated in a long arm cast to limit supination and pronation, but there is some evidence that they can be managed successfully in a short arm cast. There is high-level evidence to demonstrate that buckle fractures of the distal radius can be treated successfully in a volar wrist splint for 3–4 weeks instead of a short arm cast. Physeal fractures should be treated in a short arm cast and then followed serially with X-rays for a year to monitor for physeal closure and positive ulnar variance.

Radiocarpal and Ulnocarpal Joint Dislocations

Mechanism of Injury

Radiocarpal and ulnocarpal dislocations occur when compressive, rotational, and shearing forces cause the carpus to dislocate in relation to the distal radius and ulna. These injuries are uncommon and are often due to high-energy injuries such as falls from a height or motor vehicle accidents [27]. In sports, these are more likely to occur in pole vault and motocross events (Fig. 17.17). Radioulnar joint dislocations often occur in a Galeazzi fracture as described above. They typically occur in a FOOSH with a hyperpronated forearm [5].

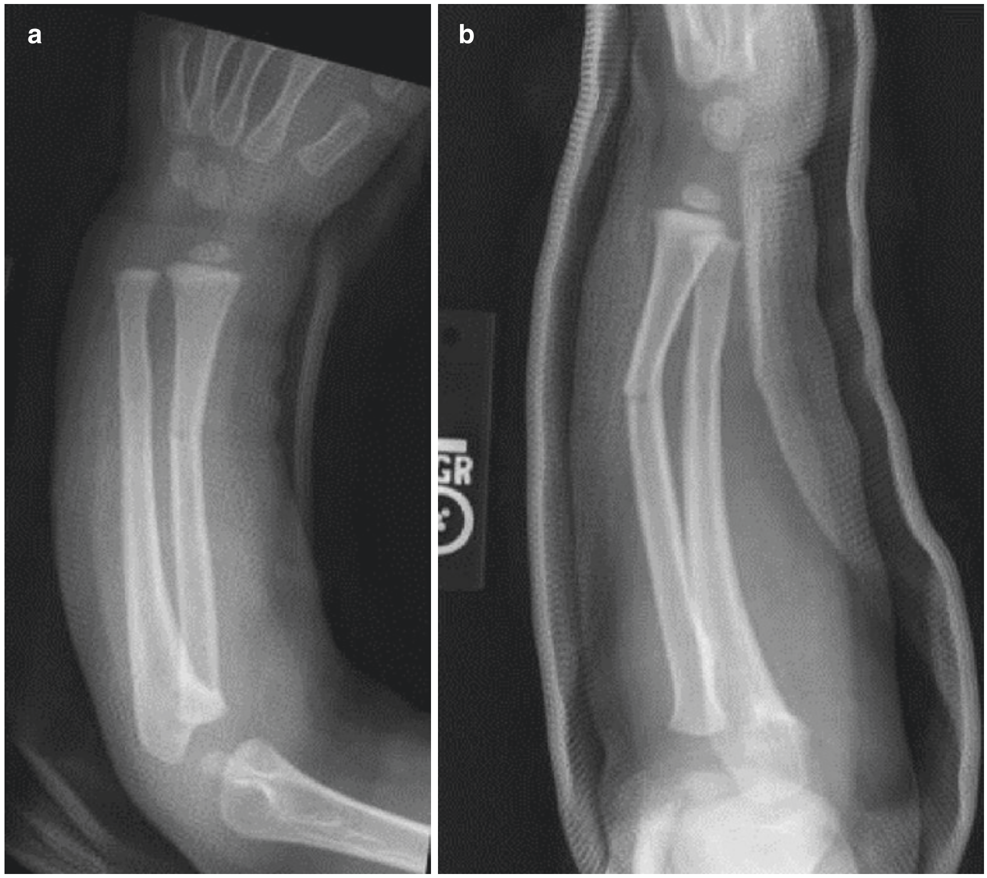

Fig. 17.15 Greenstick fracture of the radius and buckle fracture of the ulna (**a**, **b**) in a 2-year-old male who fell while playing

Epidemiology

Radiocarpal and ulnocarpal dislocations more commonly occur in men age 20–40 years old. They are very rare and account for 0.2% of all dislocations and 0.2% of all wrist injuries [27, 28].

Fracture Classification

There are two types of classification systems for radiocarpal dislocations. The Moneim classification is based on the presence or absence of injury to the intercarpal articulations. In type 1, the carpus dislocates as a unit from distal radius in a dorsal or volar direction. In addition, there may be an associated radial styloid fracture, but the intercarpal anatomy is intact. Type 2 is more complex and has an associated intercarpal fracture with ligamentous injuries [27, 28]. The second classification system is described by Dumontier and involves two groups [28]. Group 1 is a purely ligamentous dislocation with no radial fracture or only a small cortical avulsion fracture of the radius. Group 2 dislocations include a radial styloid fracture that involves at least one-third of the scaphoid fossa of the radius. Radioulnar dislocations are classified by the position of the distal radius: type 1 is dorsal displacement and type 2 is volar displacement [5].

Clinical Presentation

Patients with a radiocarpal dislocation typically present with pain, swelling, and a deformed appearing wrist. Dorsal dislocations are more common, but volar dislocations are more severe [29]. Because of the high-energy mechanism that typically causes the dislocation, patients often have open wounds and other injuries [28]. Patients may also have neurologic deficits, typically involving the median nerve, though occasionally the ulnar nerve is also injured [28]. They may also have vascular insufficiency due to arterial occlusion. The vascular insufficiency should be corrected by reduction as quickly as possible. Patients with a radioulnar dislocation almost always have a diaphyseal forearm fracture. Thus, closed attention should be paid to the DRUJ in any patient with a forearm fracture [5].

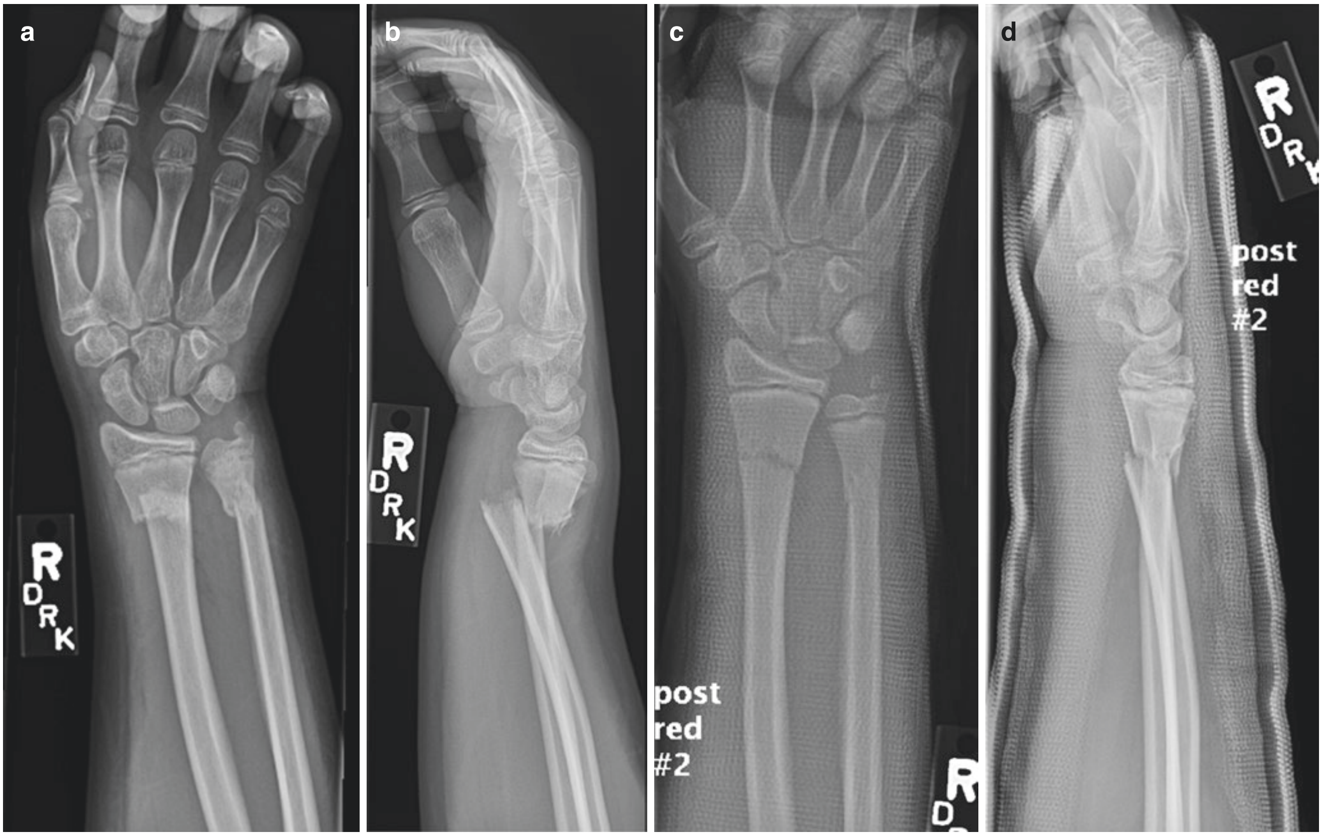

Fig. 17.16 A 13-year-old male who sustained a FOOSH injury while skiing (**a**, **b**). After closed reduction, he has improved alignment (**c**, **d**)

Imaging

Standard wrist X-rays including posteroanterior, lateral, and oblique views should be obtained in a suspected radiocarpal dislocation. The position of the lunate in relation to the radius on the lateral view determines the direction of dislocation [28]. Occasionally, stress radiographs are obtained to evaluate for stability; however, this is not routinely performed. Comparison views of the unaffected wrist can be useful in determining if the fractured arm has DRUJ disruption. A 2 mm widening of the DRUJ can suggest disruption of the joint [30]. CT typically is used for preoperative planning and can aid in evaluation of subtle fractures [28]. In addition, an MRI may be obtained to evaluate for ligamentous injuries, specifically the scapholunate and lunotriquetral ligaments [27, 29].

Initial Management

On the sideline, closed reduction may be attempted for radiocarpal or ulnocarpal dislocations if the medical facility is far, especially if there is neurovascular compromise. Reduction is performed by applying longitudinal traction to the wrist [28]. Because of the high-energy mechanism required to cause radiocarpal and ulnocarpal dislocations, patients typically have severe associated injuries that require a full evaluation in the emergency department (see Chap. 8 – Fracture Types and Definitions). Once the patient is stable and life-threatening injuries are addressed, closer evaluation of the stability of the wrist can be evaluated. If the bony alignment is normal and the wrist appears stable, a short arm cast can be placed for 6 weeks [29]. Patients with a radioulnar dislocation and Galeazzi fracture should be placed in a sugar-tong splint initially. They will typically need to be referred to orthopedics [5]; however, pediatric patients may be able to be treated without surgery.

Indications for Urgent and Nonurgent Orthopedic Referral

Although some patients have been treated with closed reduction and casting, there is a high risk for instability long term [28]. Therefore, both radiocarpal and ulnocarpal dislocations almost always require surgical fixation to restore wrist stability [27]. Specifically, patients with irreducible dislocations, an accompanying fracture, neurovascular involvement, or open injuries should be referred urgently to orthopedics [27].

Fig. 17.17 A 59-year-old male injured while mountain biking (FOOSH) and suffered an open ulnocarpal dislocation with distal radius fracture (**a**–**d**). After reduction (**e**–**g**), patient was urgently referred for a surgical fixation

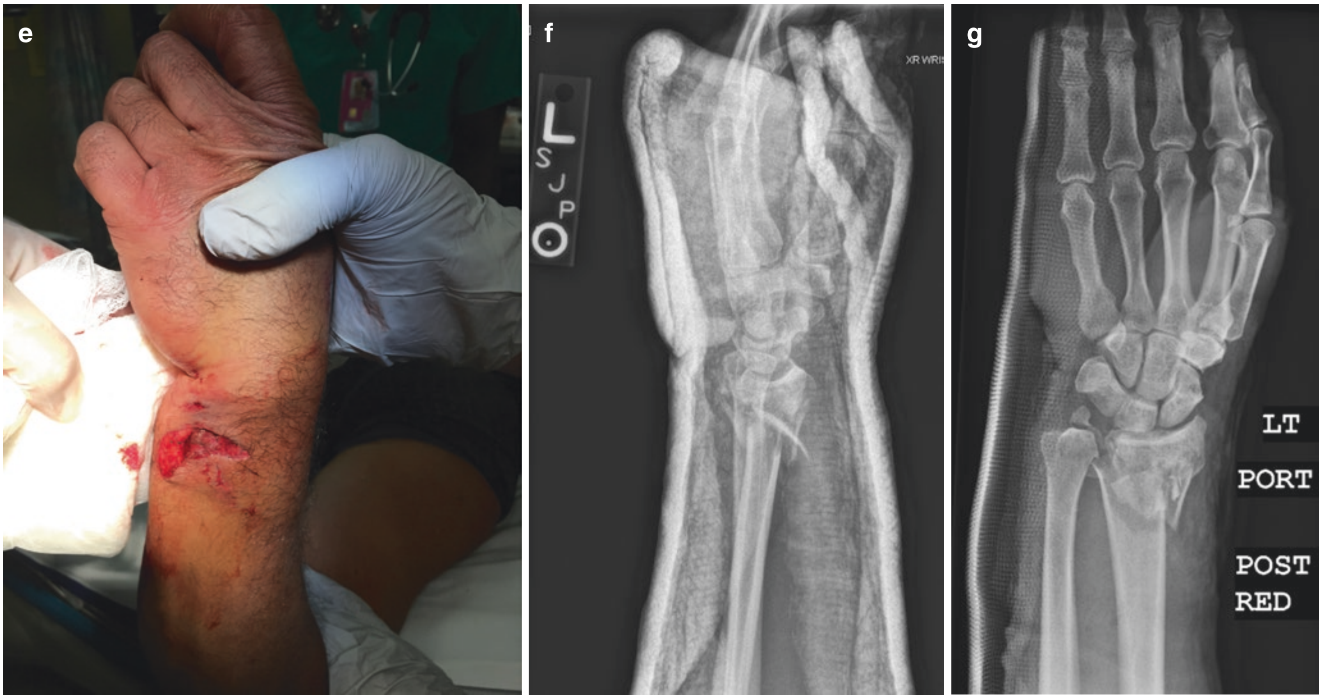

Fig. 17.17 (continued)

Follow-Up Care

If the patient is treated with nonoperative care, the patient should be placed in a short arm cast for 6 weeks. If the wrist is unstable after 6 weeks of immobilization, then the patient should be referred to orthopedics for surgical consultation. For patients who undergo surgery, follow-up depends on the type of surgery performed and the associated injuries.

Return to Sports

Full return to wrist function after radiocarpal and ulnocarpal dislocations typically takes 6 months. Because these injuries are so uncommon, there is very little evidence on return to sports. It should be noted that range of motion is often somewhat limited after radiocarpal dislocation, so aggressive PT is necessary to progress athletes back to sports.

Complications

For those patients who undergo conservative management, they still may have instability and radiocarpal collapse long term. If this occurs, surgical fixation is typically necessary. After radiocarpal dislocation, patients typically have a 30–40% decrease in the total of arc wrist flexion and extension [28]. This loss of wrist motion may occur due to prolonged immobilization and due to tightening of the intercarpal ligaments. This loss of motion is more common with open dislocations and those with nerve damage [27]. Arthritis may also develop over time and may require wrist fusion [27]. In three small case series, 11–66% of patients developed radiocarpal arthritis after radiocarpal dislocation [28]. Patients with open dislocation, complete radiocarpal ligamentous injuries, or nerve or intercarpal ligamentous injuries typically have worse outcomes [28]. If a DRUJ dislocation is not treated, patients may have chronic pain, weakness, and poor range of motion [5]. In addition, muscle-tendon entrapment can occur with more significant injuries [5].

Pediatric Considerations

It is unusual for children to have an isolated radiocarpal or ulnocarpal dislocation. It is much more common to break both the radius and the ulna or break one of the bones and dislocate the other. These patterns can be subtle and require a high index of suspicion. Galeazzi fracture with radioulnar dislocations can occur in children and can often be treated with conservative management, unlike in adults [5].

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18 Carpus

Kyros Ipaktchi, Omar Dimachkieh, and Sonia Chaudhry

Key Points

- Carpal injuries are commonly seen in upper extremity trauma and must be actively ruled out.
- With increasing understanding of the complex carpal pathophysiology, diagnostic and therapeutic modalities continue to evolve.
- Missed carpal injuries are often the source of adverse outcomes and progressive disability.
- Acute care provider must keep a high level of suspicion for these injuries and direct suspected carpal injuries to the early care of a hand surgical specialist.

Introduction

The carpus is a compound joint composed of two rows of bones which link the forearm to the hand and enables free positioning of the hand in space. The bony anatomy of the carpus consists of eight mostly cartilage-covered bones which are tightly bound to each other by intrinsic and extrinsic ligaments and capsular structures. The bony shape and alignment of the carpus plays an essential role in allowing movement around the four main planes, which are extension/flexion and radioulnar deviation as well as combined motion such as the dart throwing midcarpal joint movement. Traumatic and subsequent posttraumatic degenerative carpal conditions are commonly seen as isolated findings or in combination with distal radius and forearm fractures and are the source of significant upper extremity morbidity and functional disability.

K. Ipaktchi (✉)
Department of Orthopedic Surgery, Denver Health Medical Center, Denver, CO, USA
e-mail: Kyros.Ipaktchi@dhha.org

O. Dimachkieh
Department of Orthopaedic Surgery, UT Physicians, Houston, TX, USA

S. Chaudhry
Department of Pediatric Orthopaedic and Hand Surgery, University of Connecticut School of Medicine, Connecticut Children's Medical Center, Hartford, CT, USA

Anatomy and Pathophysiology

The proximal carpal row is composed of three bones: the scaphoid, lunate, and the triquetrum (Fig. 18.1a). These bones are dynamically linked via two major intrinsic ligaments: the scapholunate (SL) and lunotriquetral (LT) ligaments. Intrinsic ligaments originate and insert within the carpal bones as opposed to extrinsic ligaments, which connect forearm bones to the carpus. The proximal row assembly of bones and associated intrinsic ligaments has been described as a "self-stabilizing spring" holding the proximal row in harmonic position with the lunate perpendicularly (neutrally) positioned to the long axis of the radius. With intact proximal row intrinsic ligaments, the extension momentum exerted on the row by the triquetrum neutralizes the flexion tendency of the obliquely oriented scaphoid. The intercalated lunate will follow in traumatic SL or LT ruptures the rotation of the neighboring bone to which it still is attached. If the lunate is pulled into extension via the intact LT ligament in SL injuries, the midcarpal semilunar joint surface of the lunate will tilt dorsally on lateral views. This is called a dorsal intercalated segment instability (DISI) or deformity. Likewise, a LT ligament injury will result in a volarly rotated lunate via the unopposed scaphoid flexion momentum resulting in volar intercalated segment instability (VISI). The differential force momentum explains the anatomy of the SL and LT ligaments. The SL is thickest on its dorsal aspect to resist the volar flexion momentum of the scaphoid, and the volar LT ligament is thickest volarly to resist the extension vector of the triquetrum [1].

Volar to the triquetrum is the pisiform, which articulates dorsally with the triquetrum but is situated outside the carpal row (Fig. 18.1b). The pisotriquetral joint facet can give

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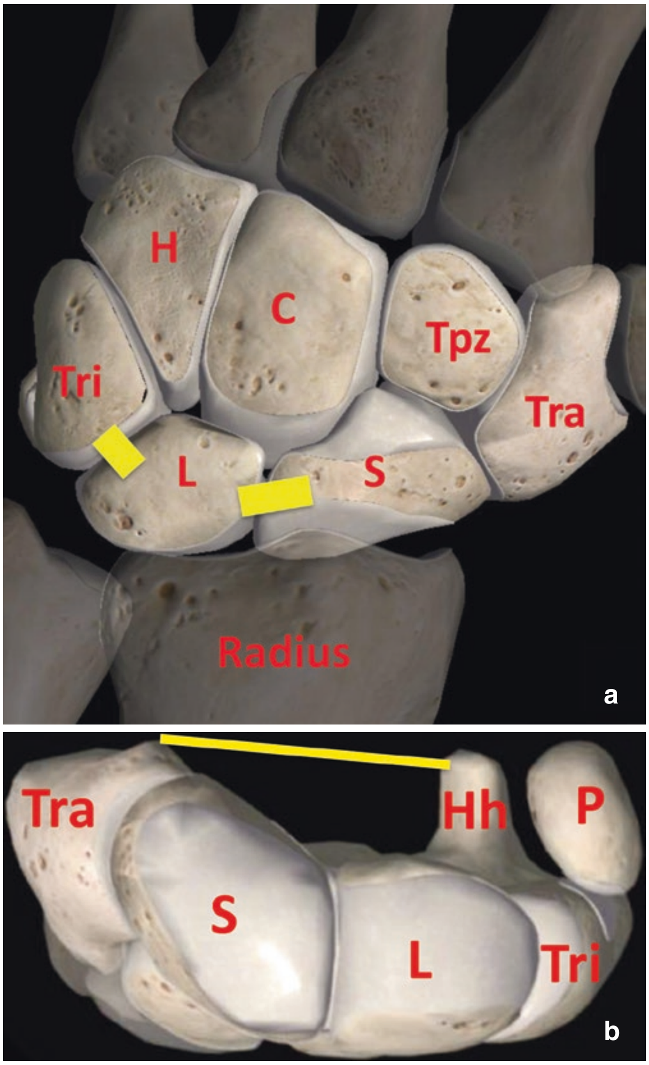

Fig. 18.1 (**a**) 3D model showing normal carpal anatomy. The proximal row consists from radial to ulnar of scaphoid (S), lunate (L), and triquetrum (Tri); the distal carpal row from ulnar to radial is formed by the hamate (H), capitate (C), trapezoid (Tpz), and trapezium (Tra). The scapholunate (SL) and lunotriquetral (LT) ligaments are outlined in yellow. (**b**) 3D carpal tunnel view showing in addition to the proximal row the trapezium (Tra), hook of hamate (Hh), and pisiform (P) which are sites of attachment for the flexor retinaculum which forms the floor of the carpal tunnel (yellow line)

rise to ulnar-sided arthritic hand pain. On the ulnar border of the triquetrum, the triangular fibrocartilage complex (TFCC) forms an extension of the radiocarpal joint surface. It spans to the ulnar styloid and helps stabilize the proximal located distal radioulnar joint. The distal carpal row includes from radial to ulnar the trapezium, trapezoid, capitate, and hamate. Unlike the more mobile proximal row, these bones are tightly bound and have minimal motion. The distal carpal row lies between the midcarpal joint, which connects it to the proximal row, and the carpometacarpal joints (CMCJ) linking the distal row to the metacarpal bones (Fig. 18.2).

The carpal bones collectively form a volarly concave arc, which forms the dorsal roof of the carpal tunnel and provides the radial (scaphoid tubercle and trapezoid) and ulnar (hook of hamate and pisiform) borders to the carpal tunnel and the attachment of the flexor retinaculum which forms the floor of the carpal tunnel (Fig. 18.1b).

Scaphoid Fractures

The scaphoid is the most frequently fractured carpal bone and accounts for up to 15–20% of wrist injuries and up to 70–80% of all carpal bone fractures [2]. This high injury prevalence can be in part explained by the exposed scaphoid position as the only carpal bone crossing the two rows, which make it especially susceptible to break in the common hyperextension and radial deviated hand position during falls on the outstretched hand (FOOSH). This mechanism loads both the scaphoid and scaphoid fossa of the radius and can lead to a scaphoid fracture. This is a type of injury that can be seen in snowboarding, soccer, rugby, or increasingly in mountain bike accidents.

More than 80% of the scaphoid is cartilage covered which is due to its important articular interaction with the radius in the radiocarpal joint and the lunate located medially in the proximal row, as well as the capitate and triquetrum medially and distally in the midcarpal joint [3]. The scaphoid can be divided into proximal, middle (waist), and distal 1/3rd segments, which differ both in terms of injury propensity and outcome. About 70% of fractures are seen in the scaphoid waist region (Figs. 18.3 and 18.4), 20% in the proximal third, and about 10% in the distal 1/3rd of the scaphoid [4].

In the pediatric population, distal pole fractures are the most common type (Fig. 18.5). For fracture healing and prognostic evaluation, the blood supply of the scaphoid is of special importance. The dorsal carpal branch of the radial artery entering through the dorsal ridge provides the main blood supply. About 80% of the blood supply is thus from distal and dorsal to proximal via "retrograde" blood flow. A waist fracture or proximal 1/3rd pole fracture can thus interrupt this important blood supply. Proximal 1/3rd fractures can undergo avascular necrosis in 33% and proximal 1/5th pole fractures can be expected to necrosis in 100% of cases. The distal superficial palmar arch branch to the distal scaphoid pole supplies the remaining 20% of scaphoid blood supply. This explains the good healing properties of distal as opposed to proximal scaphoid pole fractures.

Clinical Presentation

Tenderness in the anatomic snuffbox (Fig. 18.6), a radiodorsal depression between the tendons of the extensor pollicis

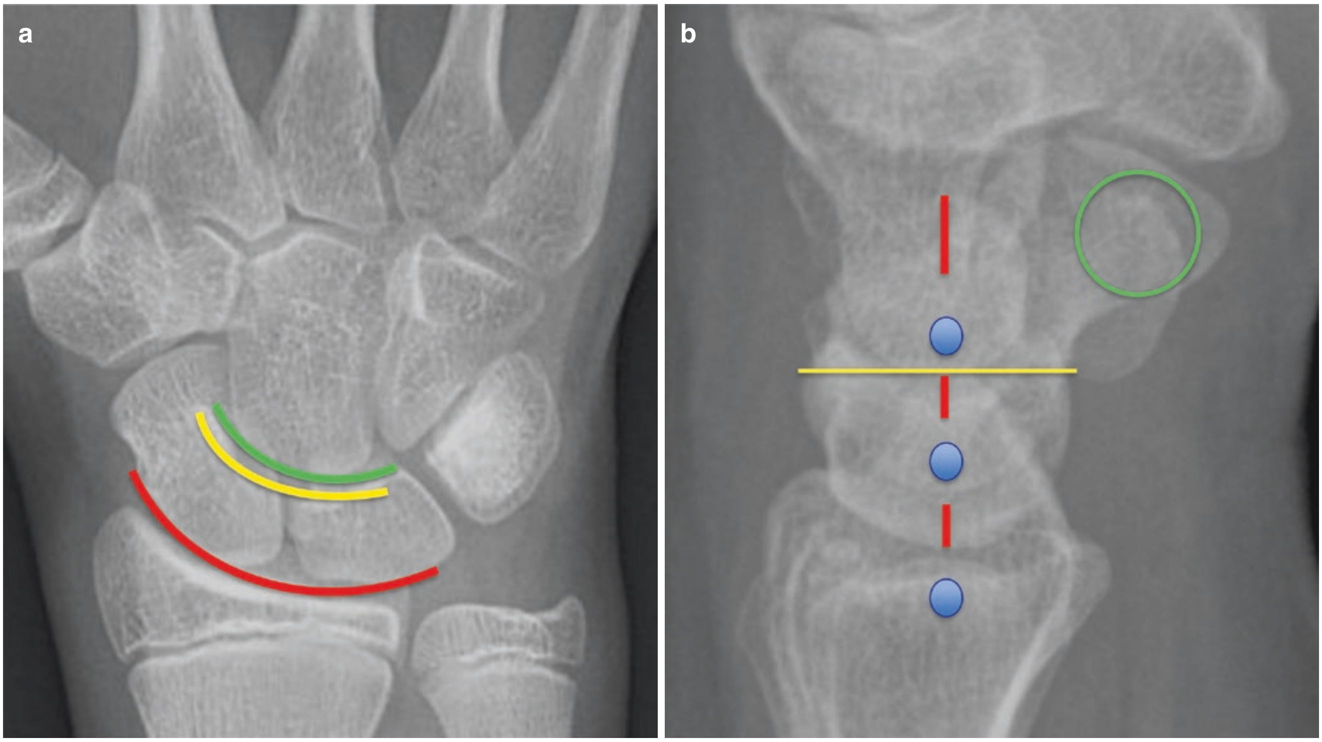

Fig. 18.2 (**a**) PA X-ray of a juvenile wrist showing normal carpal bony anatomy. Proximal and distal carpal rows outlined by Gilula arcs (red, radiocarpal joint; yellow and green, proximal and distal outline of midcarpal joint). (**b**) Correct positioned lateral view X-ray showing the distal scaphoid pole projected onto the pisiform contour (green ring). Collinear aligned radius, lunate and capitate centers (blue dots and red lines), the midcarpal lunate joint surface lies perpendicular (yellow line) to this

brevis radially and the extensor pollicis longus ulnarly, is a typical sign for scaphoid pathologies. In addition, distal volar tenderness over the distal scaphoid pole and pain to resisted pronation raises clinical suspicion for scaphoid fractures.

Imaging

Standard radiographs include wrist PA, lateral, and scaphoid views (taken in 30° wrist extension, 20° ulnar deviation, and 45° pronation) (Fig. 18.7a and Fig. 6.10, Chap. 6). Standard radiographs can be negative for fractures initially. With clinical suspicion, the patient is immobilized and repeat imaging is ordered 10–14 days later when initially occult fracture lines can appear due to bone resorption.

In the setting of a possible occult scaphoid fracture, bone scans can help diagnose scaphoid fractures with more than 95% specificity and 100% sensitivity at around 3 days after injury [5]. More commonly ordered MRI studies are highly sensitive for occult fractures immediately after injury. The MRI can help identify ligamentous injuries as well as evaluate proximal pole vascularity using T1 imaging sequences [6]. As average scaphoid thickness at the waist level measures only about 12 mm, under best of circumstances, a standard MRI with 3 mm slice thickness will capture only 3–4 images of a scaphoid waist fracture. Here CT scans can be very helpful given their faster capture rate and thinner scan slices, especially when assessing for bony union. CT scans should be ideally aligned to the scaphoid axis and no more than 1 mm thick (Fig. 18.7b).

Initial Management

The field management of suspected carpal injuries, particularly scaphoid fractures, must include a visual inspection for gross deformity and presence of skin disruption, which raises concern for fracture dislocation and open fractures respectively. This is followed by a documentation of the point of maximum tenderness, which can help formulate a preliminary differential diagnosis (e.g., snuffbox tenderness, midcarpal pain over the dorsum of the wrist). Neurovascular status must be examined and findings documented. Any signs of compromised sensation (e.g., traumatic carpal tunnel syndrome) and skin color, temperature, and capillary refill of the thumb and finger pulp, which normally is within 2 seconds after compression, raise concern for a neurovascular injury and demand urgent referral for workup. All wrist

Fig. 18.3 Middle (waist) scaphoid fracture in a 22-year-old male

Fig. 18.4 Middle (waist) scaphoid fracture in a 16-year-old male

injuries must be splint immobilized with slight elevation of the limb to heart level and referred for urgent X-ray workup.

Indication for Urgent and Nonurgent Orthopedic Referral

Most patients with carpal fractures in general and scaphoid fractures in particular should be referred to a hand specialist. As such referral to appropriate care is indicated for all suspected or diagnosed carpal injuries. Stable, nondisplaced fractures of the scaphoid waist as well as distal pole scaphoid fractures can be managed with thumb spica immobilization and a nonurgent referral within the first week to 10 days. Any displaced carpal fracture or fractures involving the proximal pole of the scaphoid should be referred urgently for evaluation and likely surgical care. All non-reducible carpal dislocations as well as open fractures and any fracture dislocation with concurrent neurovascular compromise must be triaged to same day urgent/emergent referral to receive timely reduction and operative care. Missed perilunate dislocations are among the most commonly missed and litigated upper extremity injuries in urgent/emergent care.

Nonoperative Management

Stable scaphoid waist fractures are amenable to conservative management. Assessment of fracture stability is essential. Transverse fractures are more stable than oblique or vertical patterns (Fig. 18.8). The majority of waist fractures can be managed conservatively. While short arm thumb spica cast immobilization is still commonly ordered to neutralize motion from the 1st metacarpal on the distal scaphoid pole, recent literature showed no difference in outcomes compared to regular short arm cast for minimally displaced scaphoid fractures [7].

Fig. 18.5 Avulsion fracture of distal pole of the scaphoid in a 17-year-old male as a result of an injury 3 months earlier

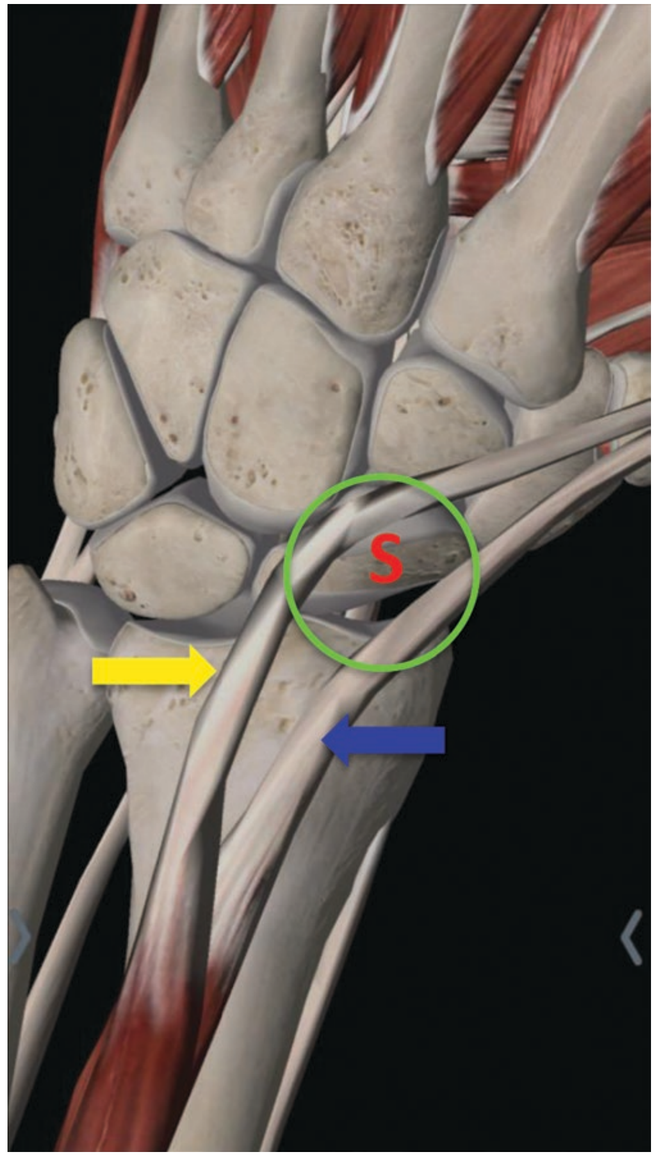

Fig. 18.6 Anatomic snuffbox (green circle) overlying the scaphoid waist (S). Borders of the anatomic snuffbox are medially the extensor pollicis longus tendon (yellow arrow) and laterally the extensor pollicis brevis and abductor pollicis longus tendon (blue arrow)

Operative Management

All unstable patterns with >1 mm displacement, >15° apex dorsal (humpback) deformity as well as interscaphoid angle of >40°, and a dorsally intercalated segment instability (DISI) of the lunate >15° are surgical indications. In addition, surgical treatment can be advocated to allow possible earlier return to work and play in minimally displaced fractures [8].

Open reduction is the standard for scaphoid fractures with significant humpback deformities and associated injuries (e.g., perilunate fractures). For the majority of minimally displaced fractures, a percutaneous interfragmentary compression screw fixation is indicated. The direction of screw insertion is commonly dictated by fracture position: proximal fractures are better approached from a dorsal approach and distal pole fractures from a volar distal approach. Ideally, a long screw placed in the center of the long axis of the bone is thought to provide the best stability.

Follow-Up Care

Waist fractures with less than 1 mm displacement are expected to heal in 90% of patients with conservative management [9]. For all patients with a suspicion of an initially occult fracture, immobilization is essential to reduce a nonunion risk from delayed immobilization. The duration of cast application depends on fracture location. Typically, the recommendation is for 12–16 weeks of immobilization for

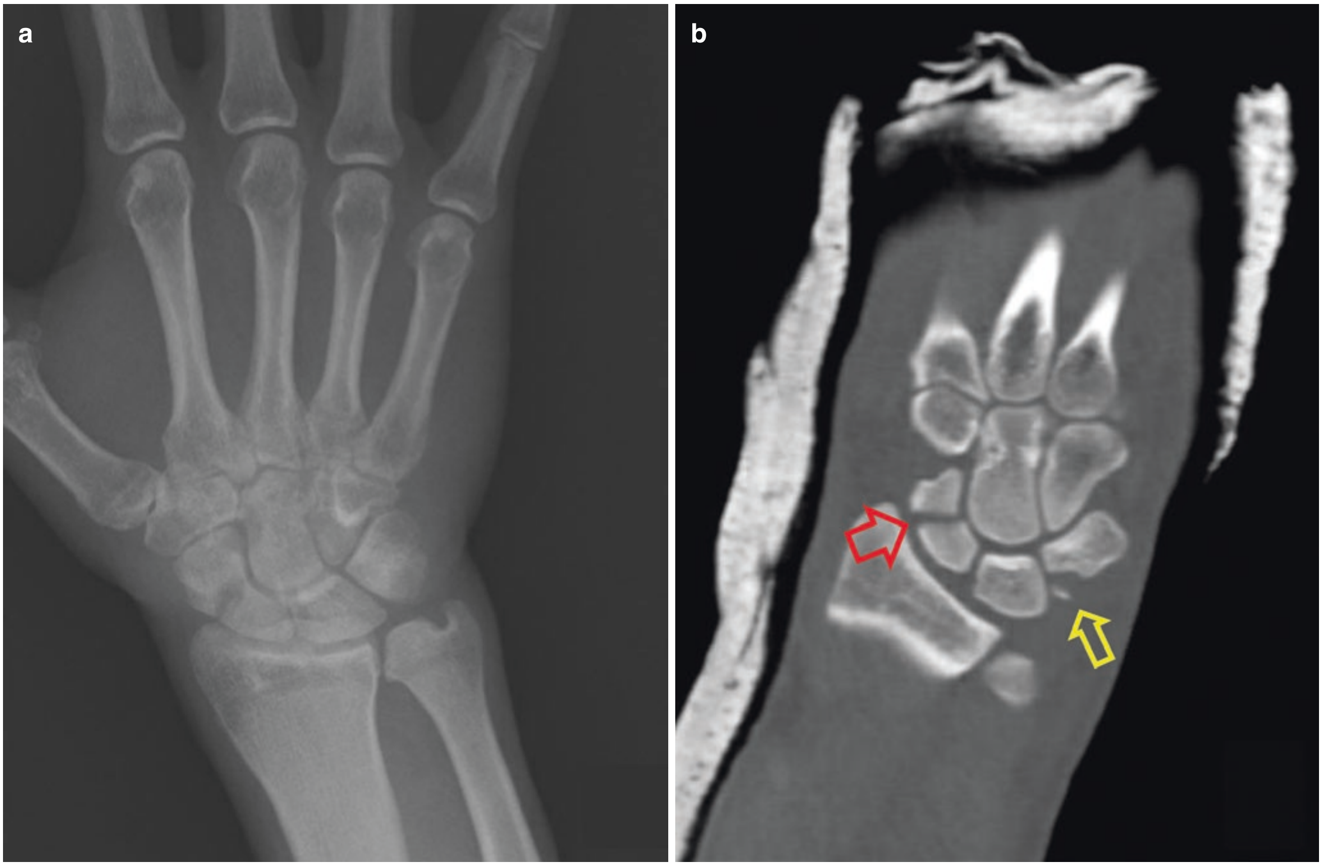

Fig. 18.7 PA X-ray of a midwaist scaphoid fracture (**a**). Coronal CT view showing both a midwaist scaphoid fracture (red arrow) and an associated occult triquetral fracture (yellow arrow) likely from a complex greater arc-type injury mechanism (**b**)

midwaist fractures and up to 5 months for proximal 1/3rd fractures.

Return to Sports

Patients should generally not return to sports activities as long as fractures are radiographically not united and clinically patients are still symptomatic. For scaphoid fractures, this is usually a 12-week interval. That said, for upper extremity injuries, patients can certainly be started on exercise bike cardio workouts with the wrist immobilized. Here bike handles should be elevated to avoid upper extremity loading as seen in race-tuned road bike setups.

Complications

Surgical complications include misplaced hardware with intra-articular screw penetration, which can also be due to collapsing bone around an initially well-placed screw. Given the tenuous blood supply and mechanical loading, nonunions (Fig. 18.9) are more often seen with scaphoid fractures and are managed by surgically addressing the contributing factors such as instability, bone loss, and lack of blood supply. This can call for augmentation of fracture stability with screw fixation, intercalated nonvascularized bone grafting, or vascularized bone grafts from the extensor retinacular system or as a free vascularized bone graft.

A long-term sequela of scaphoid nonunion is the progressive scaphoid nonunion advanced collapse (SNAC) pattern of wrist arthritis (Fig. 18.9). SNAC wrist follows a typical pattern of increasing arthritic changes around the scaphoid and increasing loss of carpal height, which is traditionally described in three stages.

Stage 1 demonstrates radial sided arthritis between scaphoid and radial styloid, stage 2 includes all changes of stage 1 plus scaphocapitate arthritis, and finally stage 3 describes periscaphoid arthritis.

Treatment of SNAC wrists has a similarly staged approach starting with radial styloidectomy for stage 1 disease and progressing to scaphoidectomy with 4-corner fusion or complete excision of all 3 bones of the proximal carpal row (proximal row carpectomy, PRC) for stages 2 and 3 (Fig. 18.9c). These motion-preserving procedures rely on the intact and arthritis-free lunate facet of the radius. For a successful PRC, the proximal capitate joint surface must be preserved and allow radiocapitellar motion. If

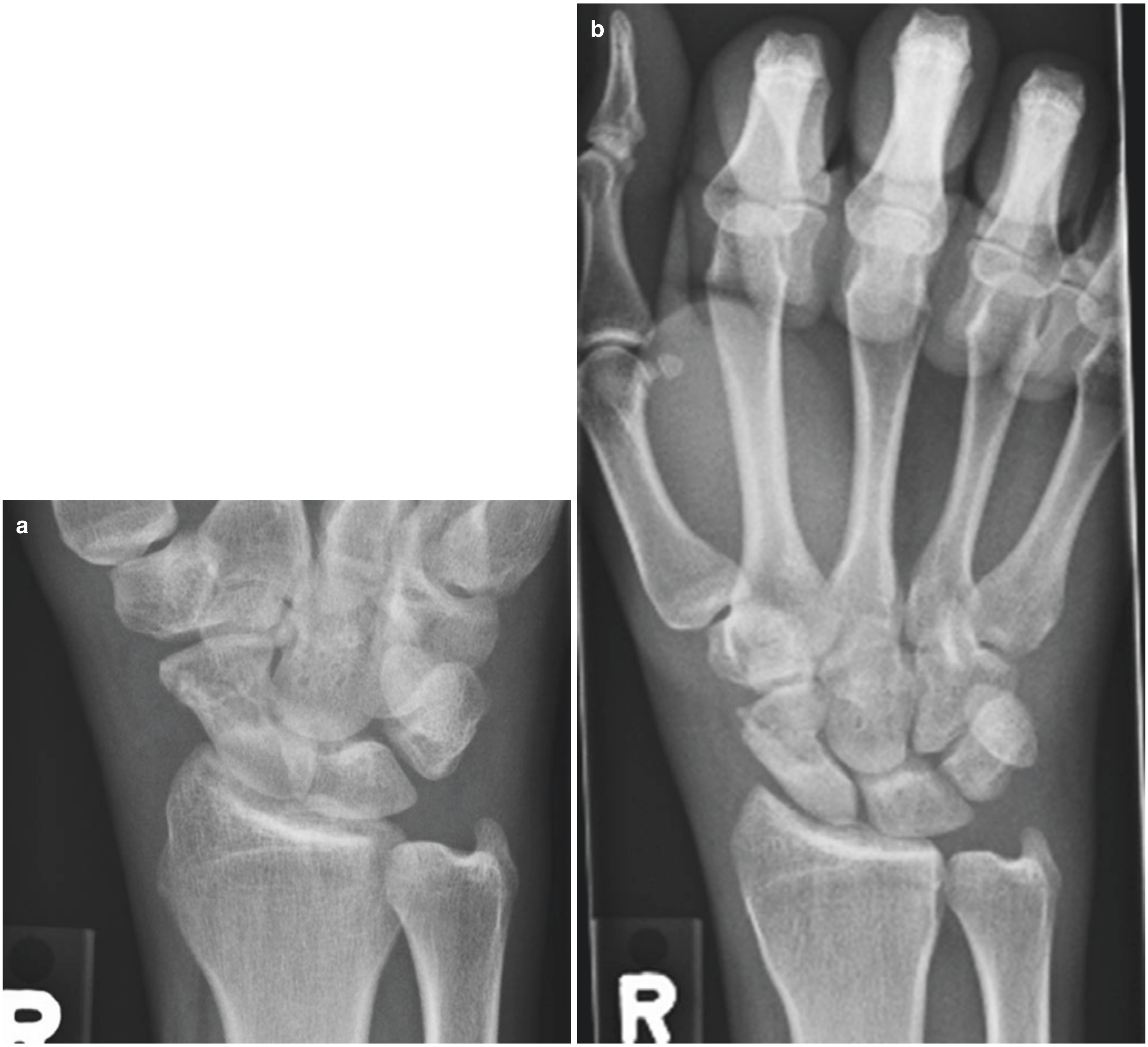

Fig. 18.8 Oblique scaphoid fracture in a 19-year-old male (**a**, **b**)

arthritic changes extend to the lunate facet of the radius or the lunocapitate joint, then both PRC and 4-corner fusions may not be successful and a wrist fusion should be considered.

Pediatric Considerations

Fracture of the carpal bones is uncommon in pediatrics, particularly in younger children. Scaphoid is the most commonly fractured carpal bone in pediatrics. However, fracture pattern in pediatrics is different than adult. Distal pole scaphoid fractures are more common in children than adults. Most pediatric scaphoid fractures are nondisplaced. Management and indications for referral are similar to adults.

Triquetral Fractures

Despite being the second most common carpal bone fracture, triquetral fractures are rare and appear often as dorsal chip avulsions visible in lateral wrist X-rays (Fig. 18.10). The mechanism is typically a fall on the extended and ulnarly deviated hand. Hyperflexion mechanisms can also result in triquetral fractures, which are a combination of radiotriquetral ligament avulsions and ulnocarpal loading and hamate compression.

These fractures can be concerning for possible dorsal intrinsic ligament injuries. Three sets of strong ligaments, which help stabilize the carpus, insert to the dorsal triquetrum: the dorsal intercarpal, the radiolunotriquetral, and the ulnotriquetral ligaments. Rarely do triquetral fractures ben-

Fig. 18.9 PA and lateral view X-ray of a chronic scaphoid nonunion with collapse (SNAC) wrist showing a mild additional SL widening and a marked DISI deformity in the lateral view (**a**, **b**). PA X-ray of the same wrist after a scaphoid excision and 4-corner fusion with staples of the lunate/capitate/triquetrum and hamate (**c**)

efit from surgical fixation, which could be indicated for open reduction and fixation in more than 2 mm displaced articular fractures. These injuries commonly present as ulno-dorsal pain located over the proximal carpal row. These injuries can be immobilized in a short arm splint/cast for 4–6 weeks.

Fig. 18.10 Triquetral fracture (arrow) in a 73-year-old female as a result of a FOOSH injury

Rare Carpal Fractures

Both pisiform and hook of hamate fractures are rare but need to be ruled out as a source of persisting ulnar and volar-sided pain with possible ulnar nerve irritation in the case of the hook of the hamate. Hook of the hamate fractures are seen by direct blows to the hypothenar eminence and can be seen in hockey, golf, and baseball where the bat or club can directly impact and compress the hook of the hamate. Persisting hypothenar pain and possible neurologic deficiency in the ulnar distribution as well as nonunions of these fractures can be best treated with fragment excision. Other types of hamate and trapezium fractures (Figs. 18.11 and 18.12) are rare. Occasionally seen lunate fractures can be found in high-energy injuries such as perilunate injuries. Lunate fractures found in lower-energy injuries with associated morphology changes can raise suspicion for an underlying avascular necrosis as in Kienbock's disease.

Carpal Instabilities

Current classifications of carpal instabilities differentiate instability patterns based on whether the instability affects and "dissociates" bones within a row or not. As such two major classes of carpal instabilities are described: the "carpal instability dissociative" (CID) and "carpal instability non-dissociative" (CIND) patterns. Examples for CID instabilities include scapholunate and lunotriquetral injuries, which dissociate the proximal carpal row [10, 11]. Non-dissociating instabilities include radiocarpal and midcarpal dislocations separating two rows. Combinations of the two groups can be seen in perilunate dislocations.

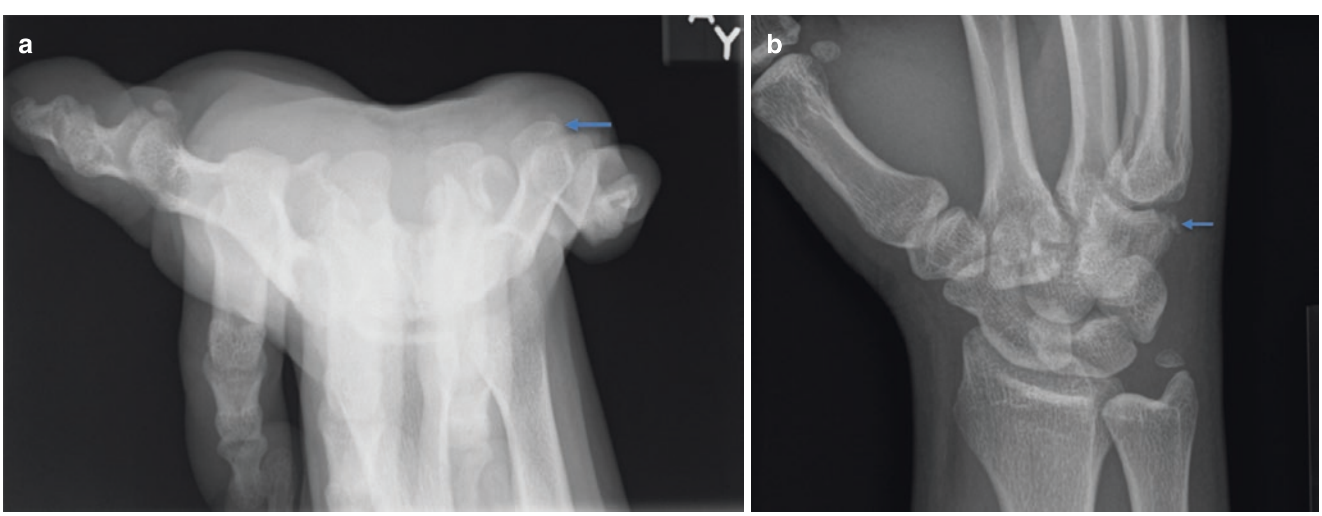

Fig. 18.11 Hamate fracture (arrows) in a 30-year-old male as a result of a punch (**a**, **b**)

Fig. 18.12 Trapezium fracture in a 34-year-old female

Scapholunate Injuries

SL injuries can be described as acute or degenerative injuries. Traumatic acute injuries are seen in approximately 20% of carpal fractures or intra-articular distal radius fractures. Degenerative SL injuries in the elderly have been described in up to 50% of geriatric patients.

Acute injuries usually occur in the setting of a fall on the outstretched and ulnarly deviated hand, leading to partial or complete tears of the SL ligament. These injuries are thus commonly seen in higher-energy falls such as in soccer and rugby as well as unprotected falls with skates. The SL ligament is composed of the strong dorsal and the thinner volar and proximal parts (Fig. 18.13a). As discussed above, complete SL tears can lead to a dorsal extended position (DISI) of the lunate based on the uncompensated pull of the triquetrum and hyperflexion of the scaphoid (Fig. 18.13b).

Similar to the SNAC wrist, a chronic SL injury can lead to progressive carpal collapse and characteristic stages of arthritic joint damage seen in scapholunate advanced collapse (SLAC) [12]. Chronic malpositioning of the carpal row leads to change of load transfer across the carpal bones and progressive degenerative breakdown of joint surfaces. In SLAC wrists the arthritic changes

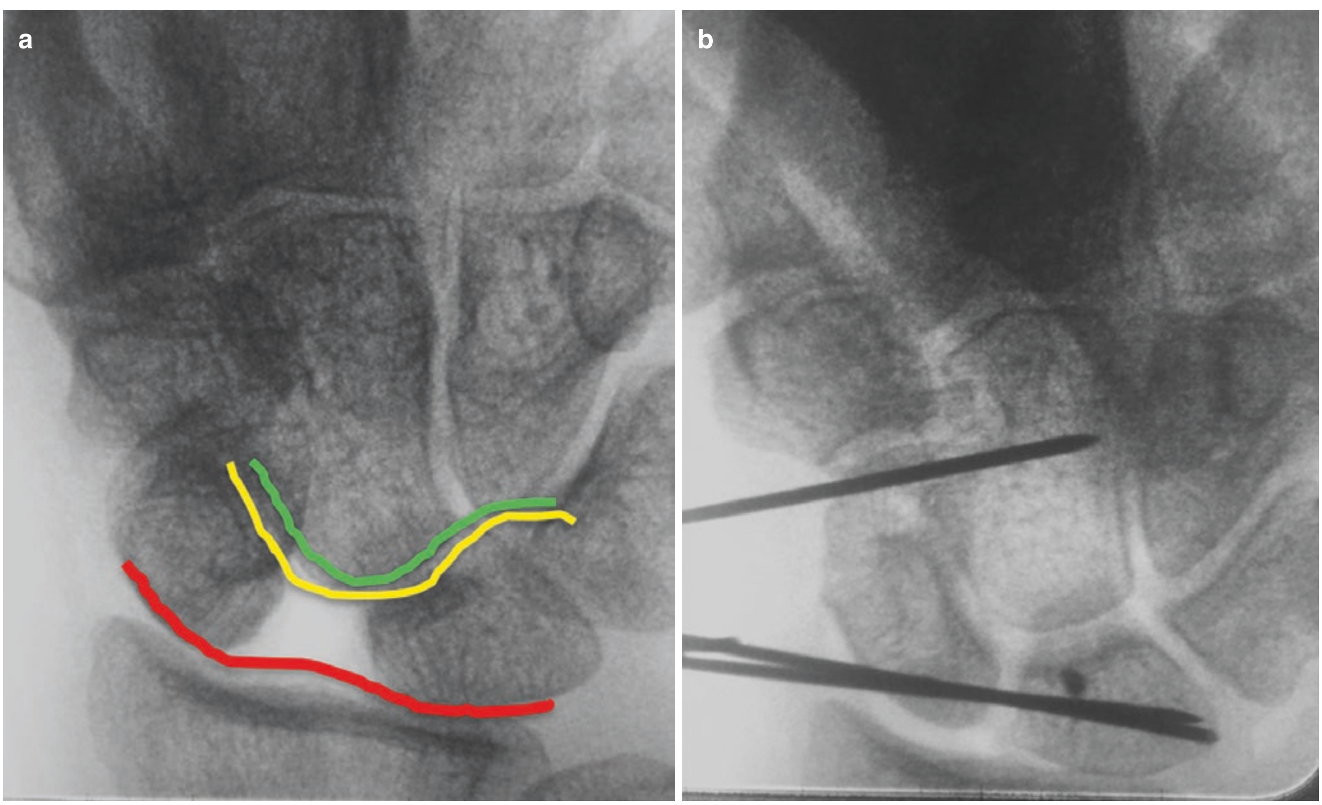

Fig. 18.13 PA X-ray of an acute scapholunate injury (**a**). Disrupted Gilula arcs, widened SL interval, and flexed scaphoid, which appear short and demonstrate a ring sign. The lunate shows typical contour changes due to dorsal (DISI) rotation. Postoperative view showing a reduced proximal row, K-wire stabilization of scaphocapitate joint, and SL interval transfixation. Anchor reinsertion of SL ligament on the lunate (**b**)

start at the radial styloid and progress over the scaphoid facet. Increasing deformity leads to capitolunate joint instability and finally dorsal displacement of the capitate head out of the midcarpal lunocapitate joint with loss of co-linear stacking of the wrist bones. These SLAC wrist changes are described in four stages. Stage 1 demonstrates arthritic changes between scaphoid and radial styloid, stage 2 shows an arthritic scaphoid facet of the radius, stage 3 includes in addition capitolunate joint arthritis, and finally stage 4 describes pancarpal arthritis (Fig. 18.14).

Clinical Presentation

Patients complain of dorsoradial pain, which can be exacerbated by forced wrist extension such as in push-ups. In the acute injury, local swelling over the dorsum of the wrist and the snuffbox area can be seen. On clinical examination, provocative testing of the rotatory stability of the proximal scaphoid pole can help establish the diagnosis. The Watson test describes a combined motion of volar pressure applied to the distal scaphoid pole and ulnar to radial deviation of the wrist. In this setting, the physiologic movement of volar flexion of the scaphoid during radial deviation leads to a dorsal shift of the unrestrained proximal pole in SL injuries, and often a painful click at the SL interval can be palpated as the proximal pole translates dorsally.

Imaging

In addition to standard PA and lateral wrist views, provocative clenched fist views (Fig. 6.11, Chap. 6) as well as views in radial and ulnar deviation can be helpful. SL dissociation leads to typical SL widening (>3 mm). On PA views the volar flexed scaphoid can show a dorsal ring sign. For the pediatric population, it is important to remember that the nonossified proximal pole of the scaphoid can falsely mimic an SL widening.

In addition to SL widening, PA X-rays will show upon close inspection a disruption of the physiologic harmonious proximal and distal carpal row alignment which can be outlined with tangential lines (Gilula arcs) along the distal radiocarpal joint as well as the proximal and distal sides of the midcarpal joint (Fig. 18.13a). Disruption of these virtual lines (similar to Shenton's line in the hip) should raise suspicion for a ligamentous and/or combined bony and ligamentous injury to the carpal rows [13].

While MRI can be a helpful tool for SL injury diagnostics, it is often overused and has a low sensitivity for SL tears. Many surgeons favor wrist arthroscopy as the gold standard for SL diagnosis.

Management

Immobilization and NSAIDs are still commonly prescribed modalities for nondisplaced acute SL injuries. Conservative

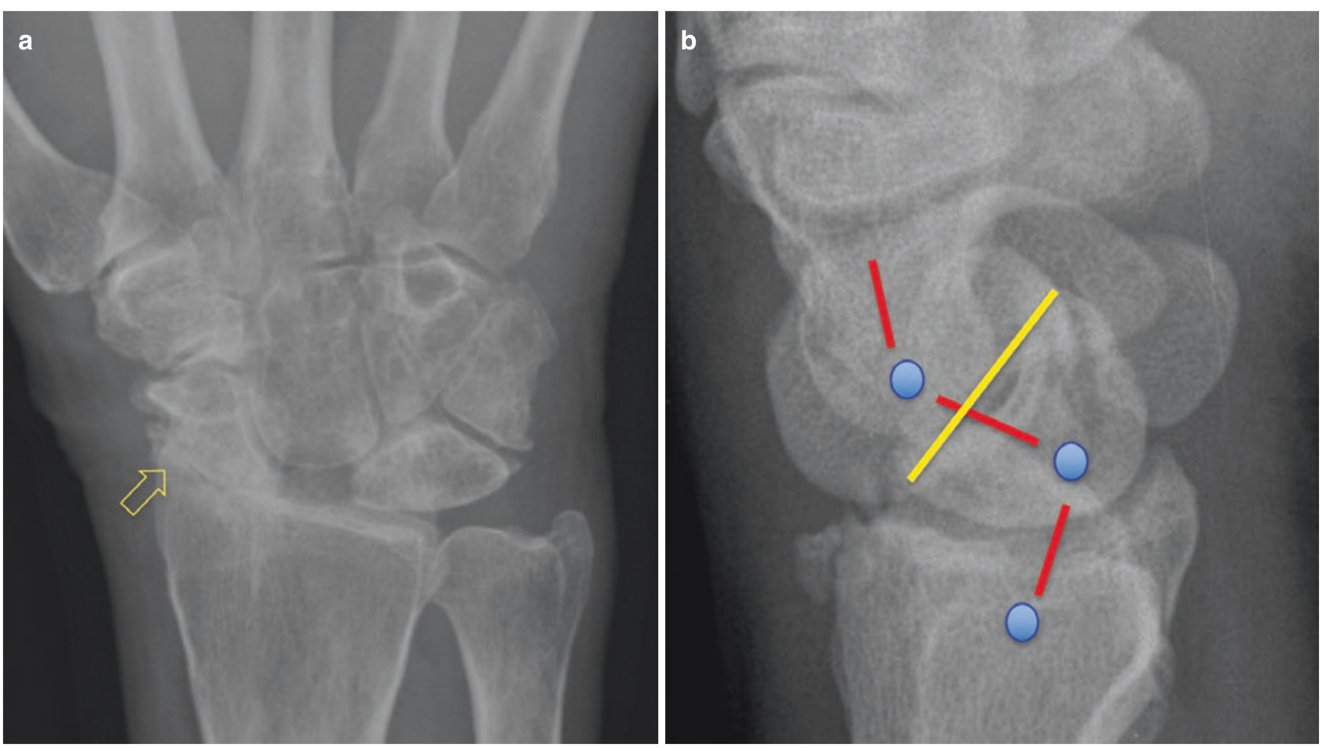

Fig. 18.14 PA X-ray of a stage 4 SLAC wrist with extensive radioscaphoid arthritis (yellow arrow) and pancarpal arthritis (**a**). Proximal capitate migration resulting in carpal collapse. Lateral view X-ray (**b**) shows typical "zigzag" collapse of the carpus with loss of collinearity (blue center markers and red axis lines) and DISI deformity of the lunate (yellow line)

management however can only be seen as appropriate for incomplete tears and may result in functionally suboptimal outcomes. Proper diagnosis and escalation of treatment with surgical SL ligament repair in the acute setting versus reconstruction if seen late (>6 month out) are the common recommendations.

Direct SL repairs using transosseous sutures and/or anchors and open versus closed reduction of the carpus and K-wire transfixation of carpal alignment for 8–10 weeks postoperatively with or without dorsal reinforcing capsulodesis are standard techniques (Fig. 18.13b).

For chronic injuries without signs of SLAC wrist arthritis, a modified Brunelli ligament reconstruction using the FCR tendon can help reconstruct the SL ligament. Often though, despite technically successful appearing repairs, secondary SL widening can be seen in follow-up imaging. This recurring SL widening does not necessarily result in functional compromise.

Once arthritic SLAC wrist changes are present, SL reconstruction is generally seen as not appropriate and instead partial carpal fusions versus proximal row carpectomies or wrist fusions are recommended (Fig. 18.14).

Perilunate Injuries

Perilunate injuries describe ligamentous or combined bony and ligamentous injuries around the lunate. This devastating injury pattern entails a complex disruption of the proximal row and midcarpal joint around the lunate and can result in a volar dislocation of the lunate into the carpal tunnel (Fig. 18.15). This is typically seen in high-energy hyperextension injuries such as falls from higher elevation and/or higher velocity (parkour, skateboarding). Despite the extensive intracarpal damage, perilunate injuries are unfortunately missed in up to 25% of initial presentations, which can result in adverse outcomes. This pathology is a common subject of upper extremity injury litigation [14].

Mechanism of Injury Is Sports

Perilunate injuries occur in high-energy-level trauma acting onto the extended and ulnarly deviated wrist. This can be seen in motorcycle crashes or higher-elevation falls onto the wrist. Bilateral injuries are possible and must be actively ruled out.

Classically, perilunate injuries have been described and staged as a clockwise injury progression around the lunate with progressive ligament failure as described by Mayfield [15]: Stage 1 starts with a SL rupture. Stage 2 is seen with distal displacement through the weak space of Poirier at the capitolunate joint and then circling back proximal and ulnarly in stage 3 with disruption of the LT ligament. Finally in stage 4, the lunate dislocates volarly into the carpal tunnel holding on by capsule and remnants of extrinsic ligaments [16].

Fig. 18.15 Lunate dislocation into the carpal tunnel in a 25-year-old male

This purely ligamentous injury pattern has been also described as a “lesser arc” injury, where the disruptive force circles through the perilunate ligaments as opposed to a more complex “wider” associated fracture dislocation pattern in which the injury transgresses through adjacent bones. This pattern has been in the past termed “greater arc” injuries and includes transscaphoid-perilunate injuries, transradial perilunate, and various combinations such as complex transscaphoid-transcapitate perilunate dislocations, etc. Figure 18.16a shows an uncommon transradial-transscaphoid translunate fracture with lunotriquetral ligament disruption and ulnar styloid fracture.

Clinical Presentation

Patient presents with swelling, wrist pain, and possible acute carpal tunnel syndrome which is more commonly seen in Mayfield stage 4 injuries where the lunate is displaced into the carpal tunnel.

Imaging

PA views show varying degrees of proximal row and Gilula arc disruptions; lateral views demonstrate a loss of linear alignment between distal radius, lunate, and capitate; and there can be a disruption of the capitolunate joint seen as well as possible associated fractures in greater arc injuries.

Management

All dislocations need an urgent closed reduction attempt, which usually entails in the emergency room 10–15 minutes of finger trap wrist distraction under hematoma block followed by manual reduction maneuver in the sedated and pain-controlled patient (Fig. 18.17). Reduction starts with manual

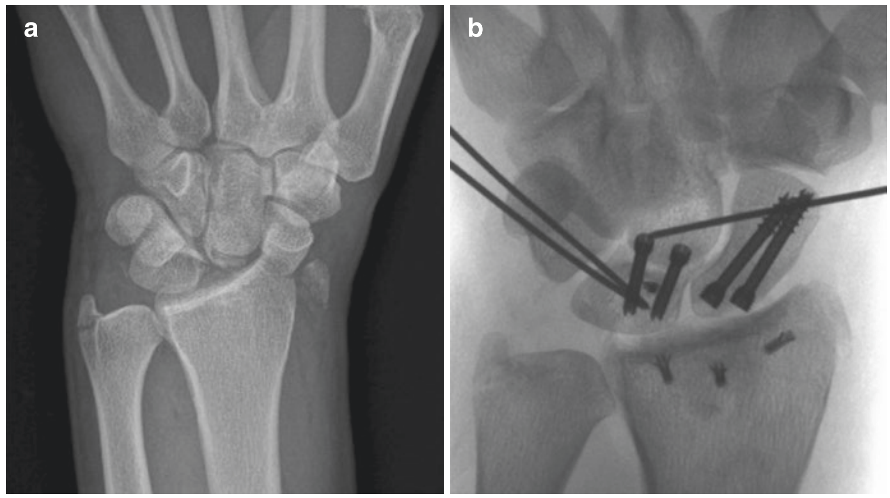

Fig. 18.16 PA X-ray of a complex transradial, transscaphoid-perilunate fracture with lunate fracture and LT ligament disruption and associated ulnar styloid fracture (**a**). Headless compression screw fixation of scaphoid and lunate fractures, reduction and transfixation of proximal carpal row, anchor fixation of LT ligament and dorsal extrinsic ligaments (**b**)

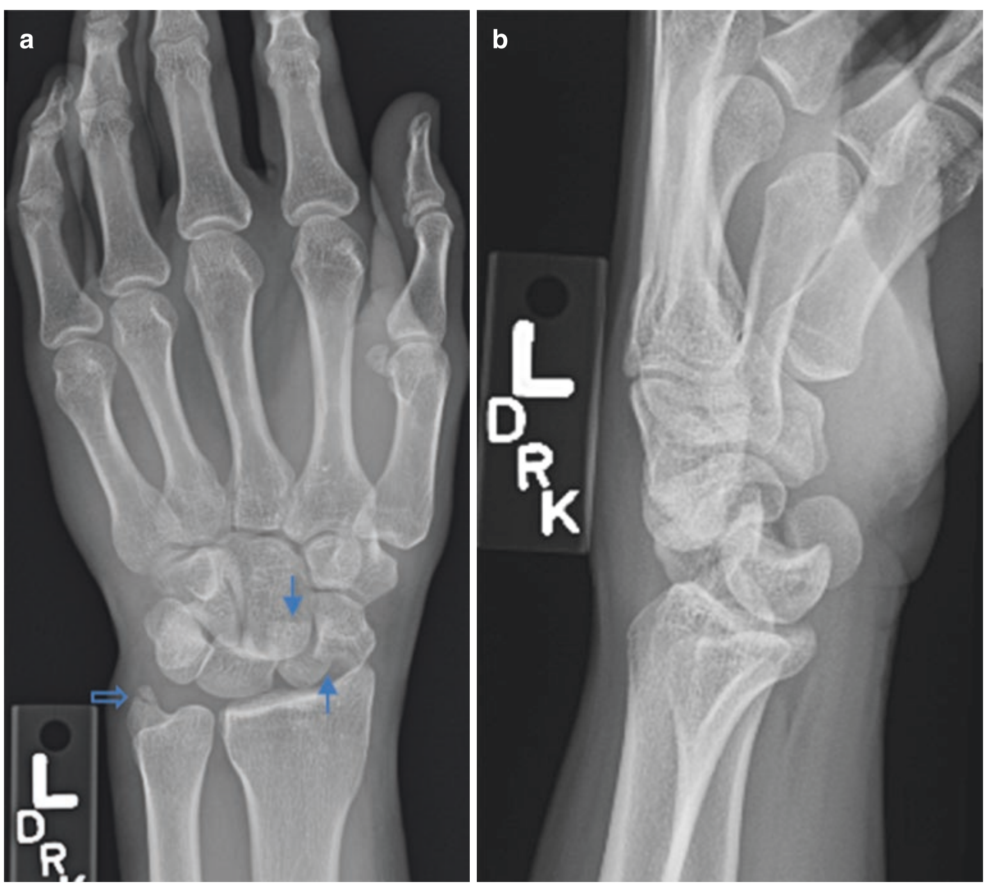

Fig. 18.17 PA (**a**) and lateral (B) X-rays of a complex displaced trans-scaphoid fracture (arrows), perilunate dislocation (best seen in **b**), and associated ulnar styloid fracture (open arrows) in a 32-old-year old male as a result of a fall during a downhill mountain biking injury. The perilunate dislocation and scaphoid fracture were reduced (**c**, **d**) in the nearby clinic. Headless compression screw fixation of scaphoid and reduction and transfixation of proximal carpal row (**e**, **f**)

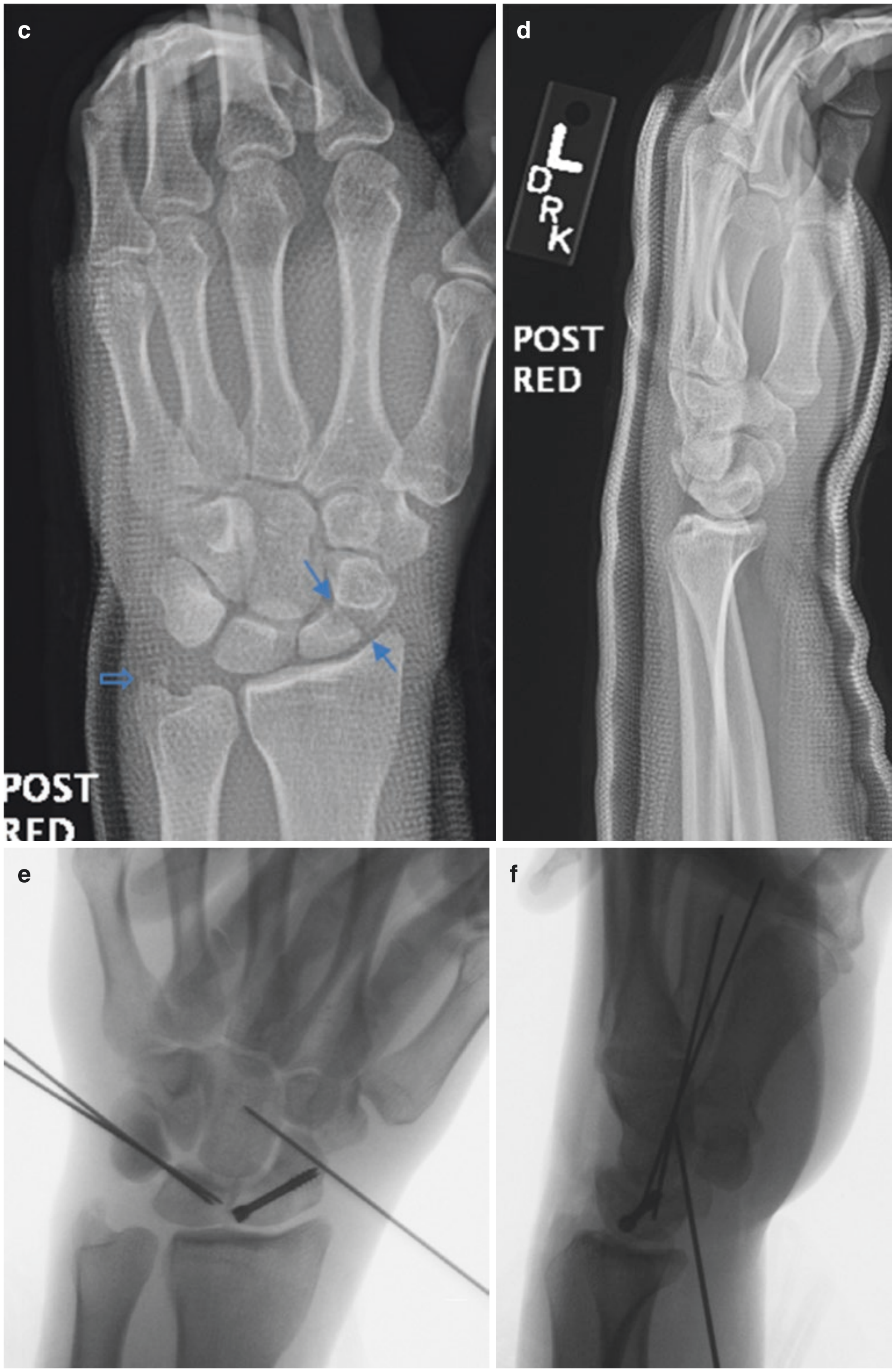

Fig. 18.17 (continued)

traction and hyperextension of the wrist to disengage the commonly dorsally displaced carpus under fluoroscopy. The lunate is during this maneuver digitally pushed dorsally into its normal position while at the same time taking the wrist into flexion to complete carpal reduction. Any successful closed reduction needs to be followed by a formal open repair of the injury to intrinsic ligaments and associated fractures within the following days or whenever the patient is stable to undergo the procedure (in the setting of a polytrauma). If closed reduction attempts fail due to instability or interposed tissues, then

at a minimum, urgent open reduction and possible carpal tunnel release are needed followed by full repair in same procedure or staged whenever patient is stable [17].

Surgical Strategy

Surgery aims to restore carpal alignment, repair disrupted ligaments, and reduce and stabilize associated fractures. Given the forces acting upon the proximal row as described above, the reduction, ligament repair, and bone fixation are commonly augmented by temporary transfixation pinning of the injured joints (Fig. 18.16b). An attempt of primary repair can still be offered in missed cases as soon as the patient presents but no later than 6–8 weeks after injury after which time period ligament repair may be unsuccessful due to scarring and retraction. In this setting, ligament reconstructions using autologous or allograft/synthetic material are offered as well as possible nonanatomic, motion-preserving procedures such as proximal row carpectomy and partial or complete fusions.

In greater arc injuries, headless compression screw fixation of associated carpal bone fractures is common practice, and bone anchors can be used to reinsert avulsed ligaments into their respective anatomic locations.

Postoperative immobilization in a short arm cast for approximately 10 weeks followed by progressive wrist range of motion exercises after transfixation wire removal is common rehabilitation recommendation. Lasting stiffness and posttraumatic arthritis can be expected in many patients.

Return to Sports

Patients with perilunate injuries can return to upper extremity involving sports activities only once fractures have healed, ligamentous stability has been restored, and the patient has regained, through extensive exercise and therapy program, a functional range of motion. This can entail more than 4–6 months. Permanent disability is likely.

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19 Metacarpus

Rebecca A. Myers, Kyle B. Nagle, and Morteza Khodaee

Key Points

- Metacarpal fractures may be displaced and angulated causing malrotation of fingers, which can cause increased morbidity and disability.
- First metacarpal fractures commonly occur at the base and are intra-articular, which require restored alignment to articular surfaces given its increased mobility.
- MCP and CMC dislocations are rare and often occur as a result of high-velocity trauma.
- Metacarpal fractures are the second most common pediatric fractures after distal forearm fractures.
- Most pediatric metacarpal fractures can be treated nonoperatively.

Introduction

Metacarpal fractures commonly occur in sport from FOOSH, direct force applied to the hand, or torsional forces applied to the fingers. Metacarpal fractures account for up to one-fifth of fractures below the elbow in general population [1]. Predominantly, metacarpal fractures occur in young males [2]. Risk of metacarpal fractures is higher in sports such as football, basketball, rugby, and baseball [1, 2].

R. A. Myers
Longs Peak Family and Sports Medicine, Department of Family and Sports Medicine, University of Colorado School of Medicine, Longmont, CO, USA

K. B. Nagle
Departments of Pediatrics, Orthopedics, and Rehabilitation, Seattle Children's and University of Washington School of Medicine, Seattle, WA, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Anatomy

There are five metacarpals. The thumb is known as the first, the index finger the second, the ring finger the fourth, and the little finger the fifth (Fig. 19.1). The bases of the second through fifth metacarpals articulate with the distal carpal row as well as adjacent metacarpals. Third and fourth metacarpal fractures are inherently more stable than first, second, and fifth metacarpal fractures given the splinting effect of the neighboring metacarpals.

Intrinsic hand muscles originate and insert within the hand and include lumbricals and interossei. Extrinsic muscles originate in the forearm and insert within the hand and include extensor digitorum communis, extensor carpi ulnaris, flexor digitorum superficialis, and flexor digitorum profundus. These muscle tendon forces can affect fractures by displacing fracture fragments. For example, dorsal and palmar interossei muscles originate on the metatarsal shafts and their forces cause the apex of many diaphysis and neck fractures to angulate dorsally.

Each metacarpal curves anteriorly and the unicondylar head is angled volarly. Metacarpal heads articulate with proximal phalanges at the metacarpophalangeal (MCP) joint. Collateral ligaments at MCP joints are the primary medial and lateral stabilizers. The ligaments lengthen when they are flexed while making a fist and shorten when they are extended while the hand is opened. Collateral ligaments are at risk of shortening and becoming stiff if they are immobilized in extension. Therefore, when immobilized, MCP joints should be flexed to 70°–90° of flexion and PIP and DIP joints in full extension in order to maintain intrinsic hand muscle length and prevent joint contractures.

The first metacarpal's structure and function are different from the rest of the metacarpals and therefore have additional fracture management considerations. The first carpometacarpal (CMC) is a biconcave saddle joint with increased biomechanical forces. Its intrinsic muscles include adductor pollicis, abductor pollicis brevis, flexor pollicis brevis, and

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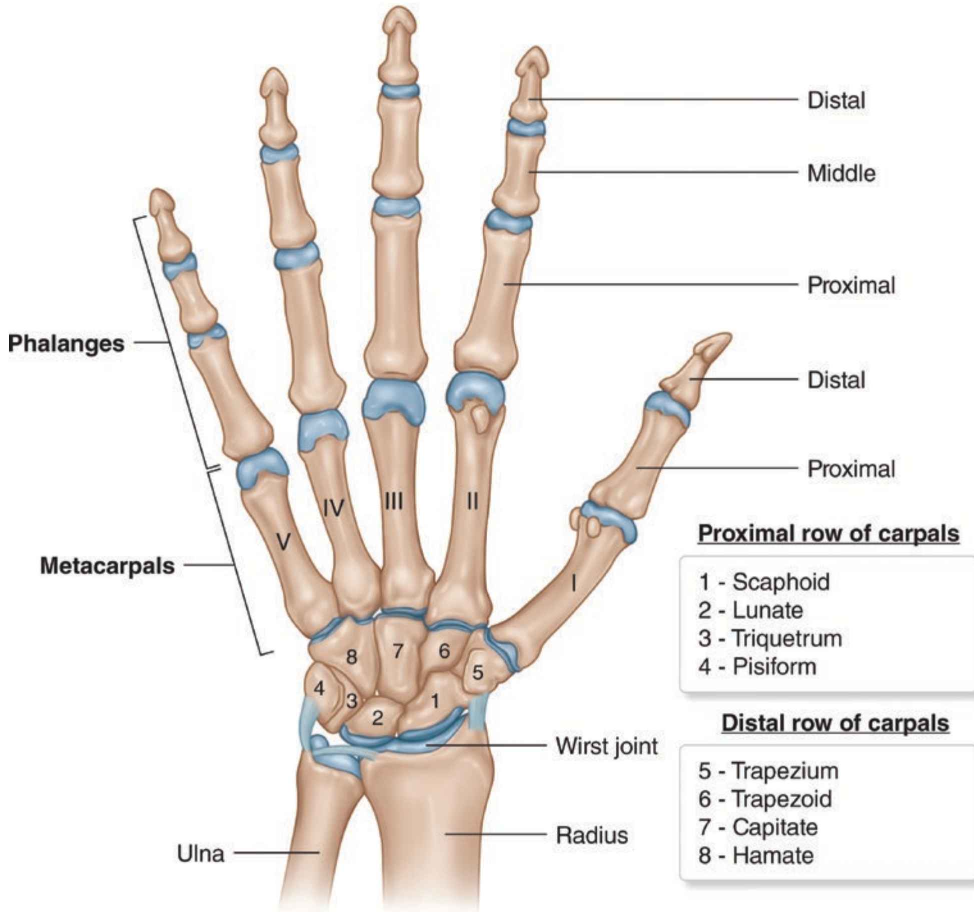


Fig. 19.1 Hand bone anatomy

opponens pollicis. Its extrinsic muscles include flexor pollicis longus, extensor pollicis brevis, and extensor pollicis longus. The web space between the first and second metacarpal needs to be maintained when immobilized to limit contractures of the intrinsic muscles.

General Evaluation Principles

After an injury, providers should assess range of motion of the wrist, hand, and fingers. Making a fist and then opening the hand and straightening the fingers give a general idea of hand function.

Rotation

Metacarpal fractures may cause rotation of fingers. When making a fist, the second through fifth fingers should point along an axis that converges on the scaphoid (Chap. 8, Fig. 8.4). The more proximal the metacarpal fracture, the more likely there will be malrotation. Therefore, it takes smaller displacement of a fracture at the base than at the neck to translate into digit malrotation. All metacarpal fractures, especially the second through fifth, need to be evaluated for malrotation.

Neurovascular Status

The arterial blood supply of the hand is from the radial and ulnar arteries that anastomose to form the deep and superficial palmar arches. The median nerve, ulnar nerve, and sensory branch of the radial nerve provide sensation to the hand. The median, radial, and ulnar nerves supply the hand's motor function. Median nerve function may be tested by touching and holding the thumb to the little finger. Radial nerve function may be tested by finger extension and the ability to give a thumb's up. Ulnar nerve function may be tested by finger abduction.

Second to Fifth Metacarpal Fractures

Fracture Classification

Metacarpal fractures are classified based on their location: head, neck, shaft (diaphysis), and base. Metacarpal head fractures may be intra-articular, comminuted, or an avulsion fracture from a collateral ligament (Fig. 19.2). These fractures may also involve the metacarpal neck (Figs. 19.3, 19.4, 19.5, and 19.6). Metacarpal neck fractures occur at the neck of the metacarpal and a boxer's fracture specifically refers to

Fig. 19.2 Fracture of the 3rd metacarpal head (arrow) in a 15-year-old male

a 5th metacarpal neck fracture (Figs. 19.3, 19.4, and 19.5). Metacarpal shaft fractures may be open or closed (Fig. 19.7). Fracture patterns may be transverse (Fig. 19.8), oblique, spiral, or comminuted. The apex of a transverse fracture is typically angled dorsally given interossei muscle attachments (Fig. 19.7). Oblique and spiral diaphysis fractures typically shorten and rotate. Base fractures may be extra-articular or intra-articular, displaced, and/or angulated given tendon and muscle attachment forces.

Base of thumb fractures have additional classification (see First Metacarpal Fracture section below). An intra-articular fracture of the base of fifth metacarpal is also known as reverse Bennett's fracture. In this fracture, the proximal, radial fracture fragment remains reduced via the carpometacarpal and intermetacarpal ligaments and joint capsule, while the metacarpal shaft is subluxed dorsally, proximally, and ulnarly by the extensor carpi ulnaris tendon.

Fig. 19.3 Fracture of the 5th metacarpal neck in a 49-year-old female

Mechanism of Injury in Sports

Metacarpal fractures occur when there is a direct force to the hand [1–4]. This can occur from the hand being stepped on during competition or when a heavy object falls onto the hand. These fractures may also occur when there are torsional forces to fingers [1–3]. Metacarpal neck and head fractures can also occur from an axial load to the metacarpal which most commonly occurs by punching an object [1–3].

Epidemiology

Metacarpal head fractures are rare [1, 3, 5, 6]. The most common metacarpal head fracture is the second metacarpal given there is less motion at its proximal carpal articulation when force is applied. The most common metacarpal fracture type is the metacarpal neck fracture as the volar aspect of the metacarpal neck is the weakest point of metacarpal [1]. Fifth metacarpal neck (Boxer's) fracture is the most common metacarpal neck fracture [1, 3, 5, 6]. Metacarpal shaft frac-

Fig. 19.4 Fracture of the 5th metacarpal neck in a 34-year-old male as a result of a fall while skiing (**a**). Postreduction radiograph shows better alignment (**b**)

tures are uncommon and can occur in isolation or in combination with other fractures (Figs. 19.9 and 19.10) or dislocations [1, 3]. Metacarpal base fractures are uncommon (Figs. 19.11, 19.12, 19.13, and 19.14). Intra-articular metacarpal base fractures are most common in the fourth and fifth digits and sometimes occur with concurrent CMC joint dislocations [1, 3]. Intra-articular fracture of the fifth metacarpal base is also called a reverse Bennett's fracture (Fig. 19.15).

Clinical Presentation

Swelling, ecchymosis, and tenderness are usually present at the fracture site. Wrist and MCP range of motion will be painful. Assess for malrotation of fingers and neurovascular status. Assess deep palmar arch blood supply and median and ulnar nerve function if injury involves second or third metacarpal. Assess ulnar artery and ulnar nerve function if injury involves fourth or fifth metacarpal. Careful inspection for abrasions and lacerations especially over the MCP joint is recommended as it may require wound irrigation and exploration.

In metacarpal neck fractures, the MCP joint may be less prominent due to the head being directed volarly due to intrinsic muscles pulling it into the flexed position. It may be difficult to extend the MCP joint. Pseudoclawing may occur if fracture is markedly angulated [2, 3, 5]. Pseudoclawing is when the MCP joint extends while PIP joint flexes when trying to extend MCP joint.

Imaging

Obtain PA, lateral, and oblique X-ray views of the hand. If occult fracture is suspected, recommend obtaining CT of hand to further evaluate.

Fig. 19.5 A volarly angulated 5th metacarpal neck fracture in a 15-year-old male (**a**, **b**). Nine years later (age 24), he suffered a 4th metacarpal base fracture (arrows) with associated posterior 5th CMC joint dislocation (**c**, **d**). Normal alignment is achieved postoperatively (**e**, **f**)

Fig. 19.5 (continued)

Fig. 19.6 Fracture of the 3rd metacarpal neck in a 40-year-old male with mild callus formation

Initial Management

On the sideline, assess and document the neurovascular status. If there is hand deformity, bony tenderness, limited function due to pain, weakness, or numbness, the athlete should not be returned to competition. Recommend ice, elevation, compression, and immobilization with splint until imaging can be done.

Nondisplaced fractures may be placed in a splint along the dorsal and volar aspect of the fracture with the wrist in 30° of extension, the MCP joint in 70°–90° of flexion, and PIP and DIP joints in extension [1, 3, 7]. Ulnar gutter splints are optimal for fourth and fifth metatarsal fractures. Similarly, fractures of the second or third metatarsals can be immobilized with a radial gutter splint. Depending on the location of the displaced metacarpal fractures, closed reduction may or may not be indicated. Closed reduction is performed as nonoperative management of some displaced metacarpal fractures or as the initial management to preserve the neurovascular status prior to surgery [7]. Most published studies on closed reduction techniques are about 4th or 5th metacarpal neck fractures [7]. Regional or hematoma blocks

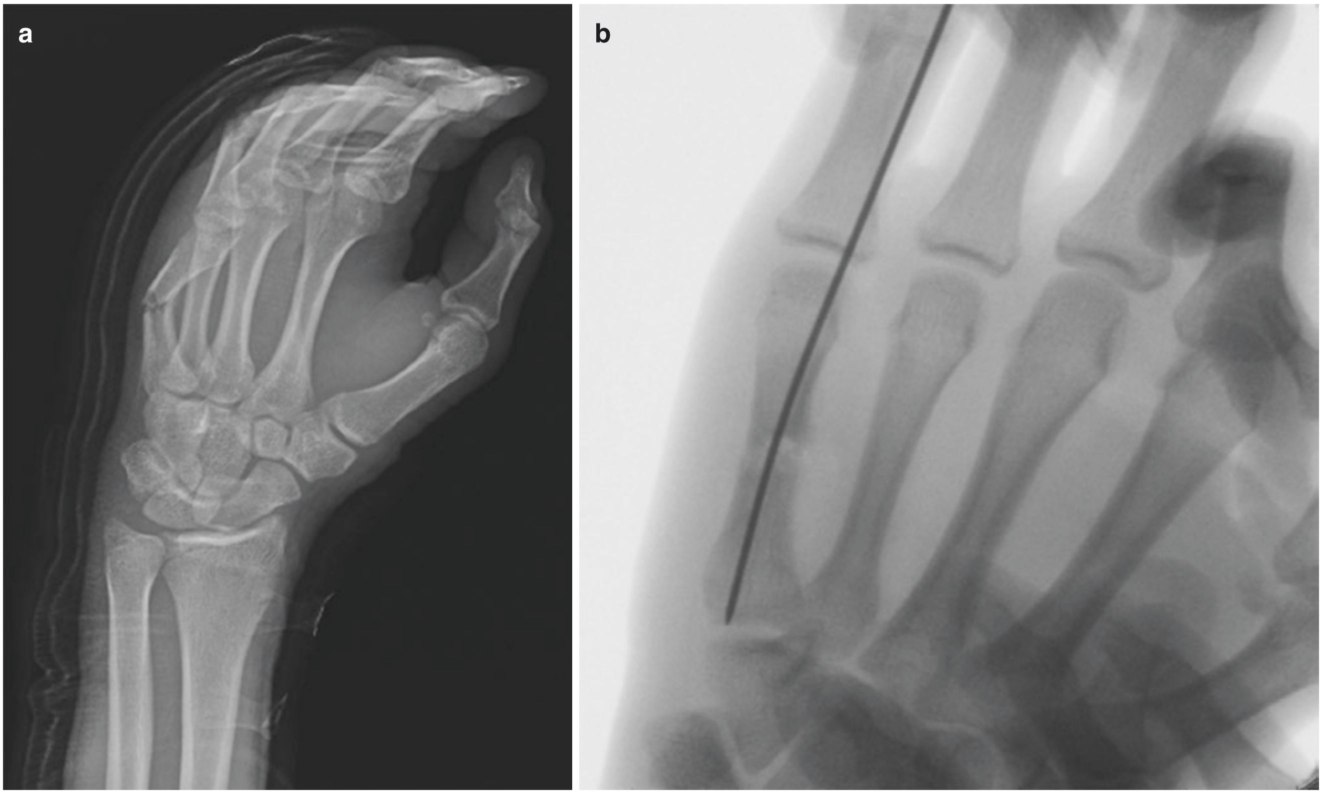


Fig. 19.7 Volarly angulated transverse fracture of the 5th metacarpal shaft in a 16-year-old male (**a**). Normal alignment is achieved postoperatively (**b**)

Fig. 19.8 Displaced transverse fracture of the 2nd metacarpal shaft in a 24-year-old male as a result of a fall while snowboarding (**a**, **b**)

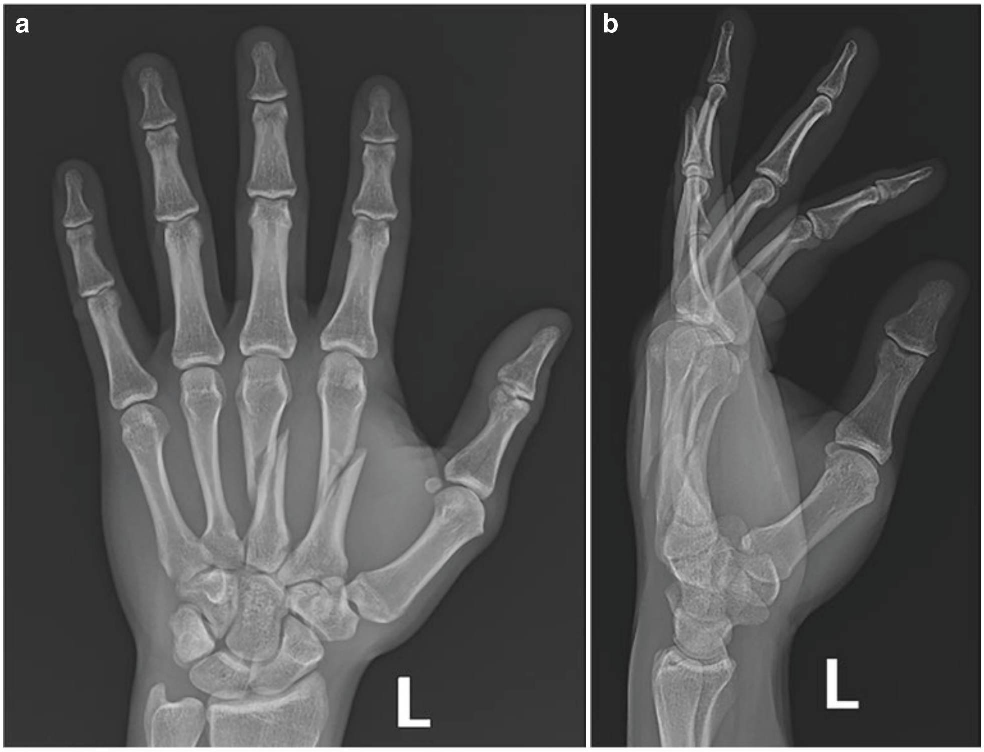


Fig. 19.9 Displaced spiral fractures of the 2nd and 3rd metacarpal shafts in a 25-year-old male as a result of a fall while skiing (**a**, **b**)

typically provide adequate anesthesia. Traction with finger traps may help with the procedure, but they are not always necessary. Dorsally directed pressure should be applied to the distal fragment of the affected digit while in 90° of MCP joint flexion [7]. The proximal metacarpal part should be stabilized or directed volarly [7]. Ultrasound can be used to ensure articular surface of metacarpal is aligned. The hand should be immobilized in a splint similar to nondisplaced fractures above. Postreduction radiographs should be obtained.

Indications for Urgent and Nonurgent Orthopedic Referral

Immediate referral is only indicated if there is an open fracture or there is compromised neurovascular status. Open fractures may require delayed closure if contamination is present.

Decision-making for nonoperative versus operative management of metacarpal fractures depends on many factors such as location of the fracture, intra- or extra-articular type, degree of angulation and rotation, amount of shortening, and the sport and/or occupation of the patient. In general, metacarpal fractures that are more distal and on the ulnar side of the hand are better tolerated and less likely to need surgical interventions [1–3, 5–7].

Fractures that cannot be reduced or maintain reduction will likely need surgical intervention. Malrotation of fractures is best evaluated clinically [5]. A few degrees of malrotation can cause significant deformity at the level of the finger [1, 3, 5]. Due to the possible significant decrease in grip strength and pseudoclaw deformity, metacarpal shaft and neck fractures with significant dorsal angulation (10° at the 2nd and 3rd, 20°–30° at the 4th, and 40° at the 5th metacarpals) should be referred to hand surgery [1, 3, 5]. Any shortening of more than 5 mm or with an articular step-off of more than 1 mm should be referred to hand surgery as well [1–3, 5, 7]. Another indication for surgical referral is the presence of multiple metacarpal fractures or association with MCP or CMC joints dislocations [1–3, 5, 7].

Metacarpal Head Fractures

Most metacarpal head fractures are intra-articular; therefore, referral to a hand surgeon is appropriate [1, 2, 5, 6]. Small avulsion fractures with stable MCP joints can be managed nonoperatively [1, 2].

Metacarpal Neck Fractures

The majority of metacarpal neck fractures can be managed nonoperatively [1, 2, 5, 6, 8]. In general, metacarpal neck fractures with dorsal angulation up to 10° at the 2nd, 20° at the 3rd, 30° at the 4th, and 40° at the 5th can be managed nonoperatively [1, 2, 5, 6, 8].

Fig. 19.10 Displaced spiral fractures of the distal 3rd, 4th, and 5th metacarpal shafts in a 29-year-old male as a result of a fall while skiing (**a**, **b**)

Metacarpal Diaphysis (Shaft) Fractures

Dorsal angulation is less tolerated in metacarpal shaft fractures compared to the neck fractures [1, 2, 5, 8]. This is mainly due to more displacement of metacarpal shaft fractures compared to the metacarpal neck fractures [1, 2, 5]. In general, metacarpal shaft fractures with dorsal angulation up to 10° at the 2nd, 20° at the 3rd, and 30° at the 4th and 5th can be managed nonoperatively [1, 2, 5, 8]. These general rules may not apply to athletic population as optimal function is usually required to perform at the highest level [1, 2].

Metacarpal Base Fractures

Extra-articular base fractures can be treated similar to metacarpal shaft fractures [1]. Intra-articular fractures occur mainly at the 4th and 5th metacarpal bases and they are often unstable [1–3]. Nondisplaced intra-articular fractures with less than 25% joint involvement can be managed nonoperatively, but it is still advised to refer them to hand surgery [1]. Base fractures of the 4th and 5th metacarpals can be associated with CMC joint dislocations (often posterior), so clinicians should carefully evaluate this with lateral X-ray view [1–3].

Follow-Up Care

If managed nonoperatively, it is advised to place a cast 3–5 days after the edema has subsided. Depending on the type of fractures, a follow-up within 2–4 weeks with repeat X-rays is recommended. Early gentle ROM is advised to prevent joint stiffness. Functional and protective braces are appropriate at this stage as they allow for ROM exercises. Athlete can be cleared after clinical and radiographic healing

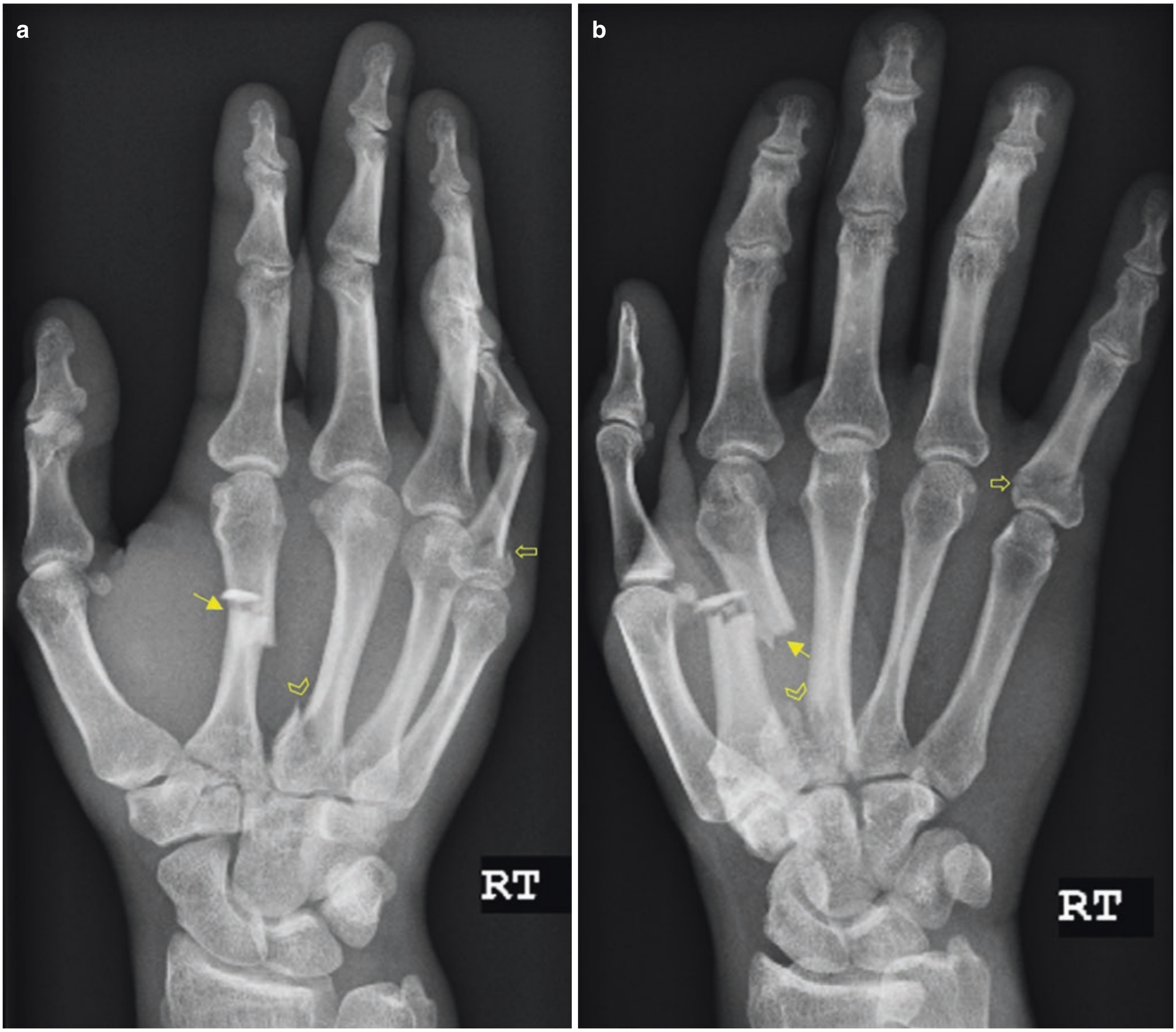


Fig. 19.11 Displaced comminuted transverse fracture of the 2nd metacarpal shaft (arrows), intra-articular fracture of the 3rd metacarpal base (arrowheads), and intra-articular fracture of the 5th proximal phalangeal base (open arrows) in a 32-year-old male as a result of a mountain bike injury (**a**, **b**)

is achieved [1, 2]. In general, athletes postoperatively can start gentle ROM and hand therapy as early as 5–7 days [1, 2].

First Metacarpal Fractures

Fracture Classification

Most first metacarpal fractures occur at the base. First metacarpal shaft fractures can be categorized and managed similar to the other metacarpal shaft fractures (Fig. 19.16). There are three types of first metacarpal base fractures in adults (Fig. 19.17). Type I also known as Bennett's fracture (described by Edward Bennett in 1882) occurs when there is an intra-articular fracture, and a proximal volar fracture fragment stays attached to the trapezium by the anterior oblique ligament (Fig. 19.18). The remaining shaft of the metacarpal is pulled proximally, dorsally, and radially by the abductor pollicis longus (Fig. 19.17). Type II also known as Rolando's fracture (described by Silvio Rolando in 1910) occurs when there is a comminuted intra-articular fracture at the base (Fig. 19.19). Rolando's fracture can be comminuted (Fig. 19.20) with three or more pieces (called by some "comminuted intra-articular") [1, 9].Type III occurs when there is an extra-articular fracture at the proximal shaft of the metacarpal. Type IIIA is a transverse fracture (Fig. 19.21) and Type IIIB is an oblique fracture (Fig. 19.22).

Mechanism of Injury in Sports

Base of first metacarpal fractures occur when there is an axial load to a partially flexed thumb, which can occur when punching with a clenched fist or falling onto a flexed thumb [1, 9, 10]. Fractures may also occur from a fall onto the hand causing hyperflexion or hyperabduction to thumb [1, 9, 10].

Fig. 19.12 Extra-articular fracture of the 4th metacarpal base in a 32-year-old male

Epidemiology

First metacarpal fractures account for one-fourth of all metacarpal fractures [11]. The majority of these fractures occur at the metacarpal base [11]. Fracture of the base of the first metacarpal accounts for about 5% of hand fractures [1, 9, 11]. First metacarpal fractures are relatively uncommon injuries in sports [1, 9–11]. The exact prevalence of thumb injuries in athletes is unknown, but it seems to be more common in sports when the thumb is exposed to direct trauma such as football, volleyball, and sports with high risk of falls [1, 9, 11].

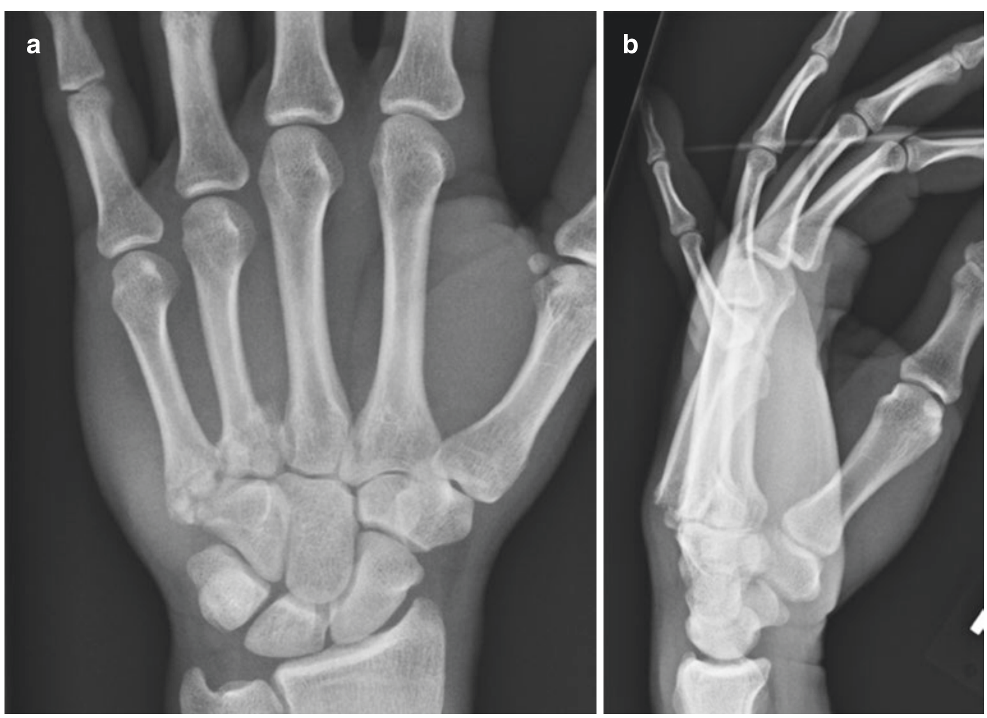
Clinical Presentation

Patients usually present with pain and swelling at the base of thumb and radial wrist. ROM of the thumb is usually decreased.

Imaging

Obtain AP, lateral, and oblique X-ray views of the thumb. If occult fracture is suspected, recommend obtaining CT to further evaluate.

Fig. 19.13 Comminuted intra-articular fracture of the 4th and 5th metacarpal base (**a**, **b**)



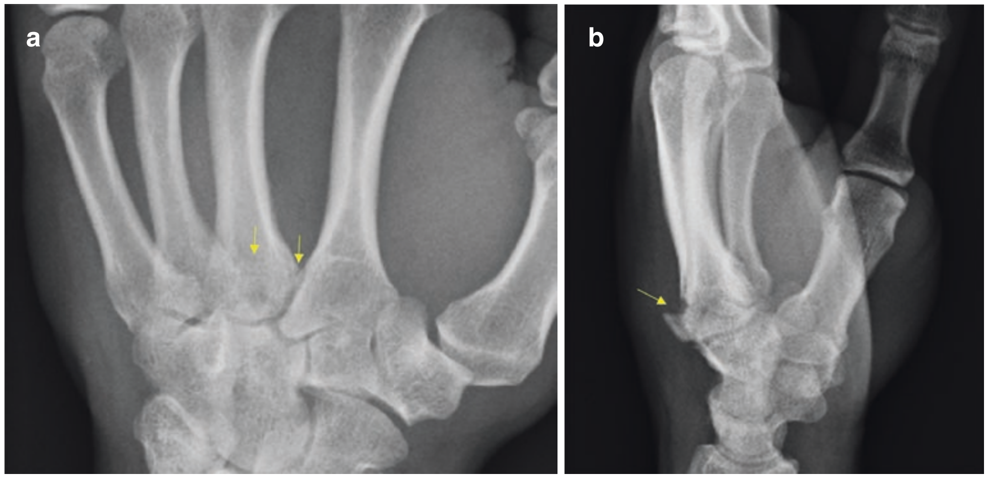


Fig. 19.14 Mildly displaced fracture (arrows) of the 3rd metacarpal base in a 31-year-old male as a result of a mountain bike injury (**a**, **b**)

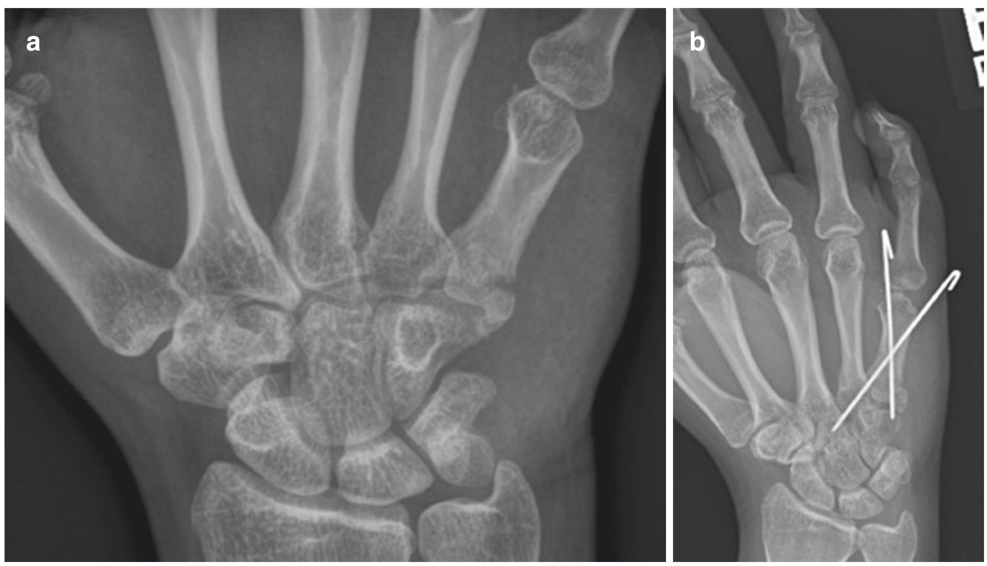


Fig. 19.15 Intra-articular fracture of the 5th metacarpal base (reverse Bennett) in a 36-year-old female (**a**). The fracture was stabilized by pinning (**b**)

Initial Management

First metacarpal head, neck, shaft, and extra-articular base fractures should be managed similar to the second to fifth metacarpal fractures principles [1]. Assess neurovascular status. Ice, elevation, compression, and immobilization should be applied until imaging can be obtained. Nondisplaced extra-articular fractures and intra-articular fractures are placed in a thumb spica splint with the wrist in 30° of extension, MCP joint in neutral position to avoid contracture of the first web space, and interphalangeal (IP) joint free or in extension [1, 9–12]. Displaced extra-articular fractures with more than 30° of angulation need to be reduced [1, 9–12]. Perform a hematoma block at the fracture site. Finger traps are helpful to dis-impact the fracture prior to reduction. Apply traction to the thumb while applying pressure over the apex of the fracture [1, 9, 11]. Hyperextension of the MCP joint should be avoided. Evaluate reduction with ultrasound or X-ray to ensure articular surface of metacarpal is aligned. Immobilize in thumb spica splint with wrist in 30° of extension, MCP joint in neutral position to avoid contracture of the first web space, and IP joint free or in extension [1, 9, 11].

Fig. 19.16 Intra-articular oblique fracture of the 1st metacarpal neck in an 18-year-old male (**a**, **b**)

Indications for Urgent and Nonurgent Orthopedic Referral

Patients with intra-articular fractures should be referred in order to restore alignment to articular surfaces of the first CMC joint given its mobility [1, 9–12]. Both operative and nonoperative management of mildly displaced intra-articular fractures are practiced with evidence more in favor of operative options [1, 9–12]. Displaced extra-articular fractures that cannot be adequately reduced should also be referred [1, 9, 11].

Follow-Up Care

Athletes with thumb spica cast or splints should be re-evaluated for clinical and radiographic healing in 2–3 weeks and 5–6 weeks [1, 11]. Early ROM exercises are advised as early as 5–10 days after screw fixation or after 4 weeks with pinning [11]. Athletes usually can return to sports in 2–6 weeks depending on their treatment and sport [1, 11].

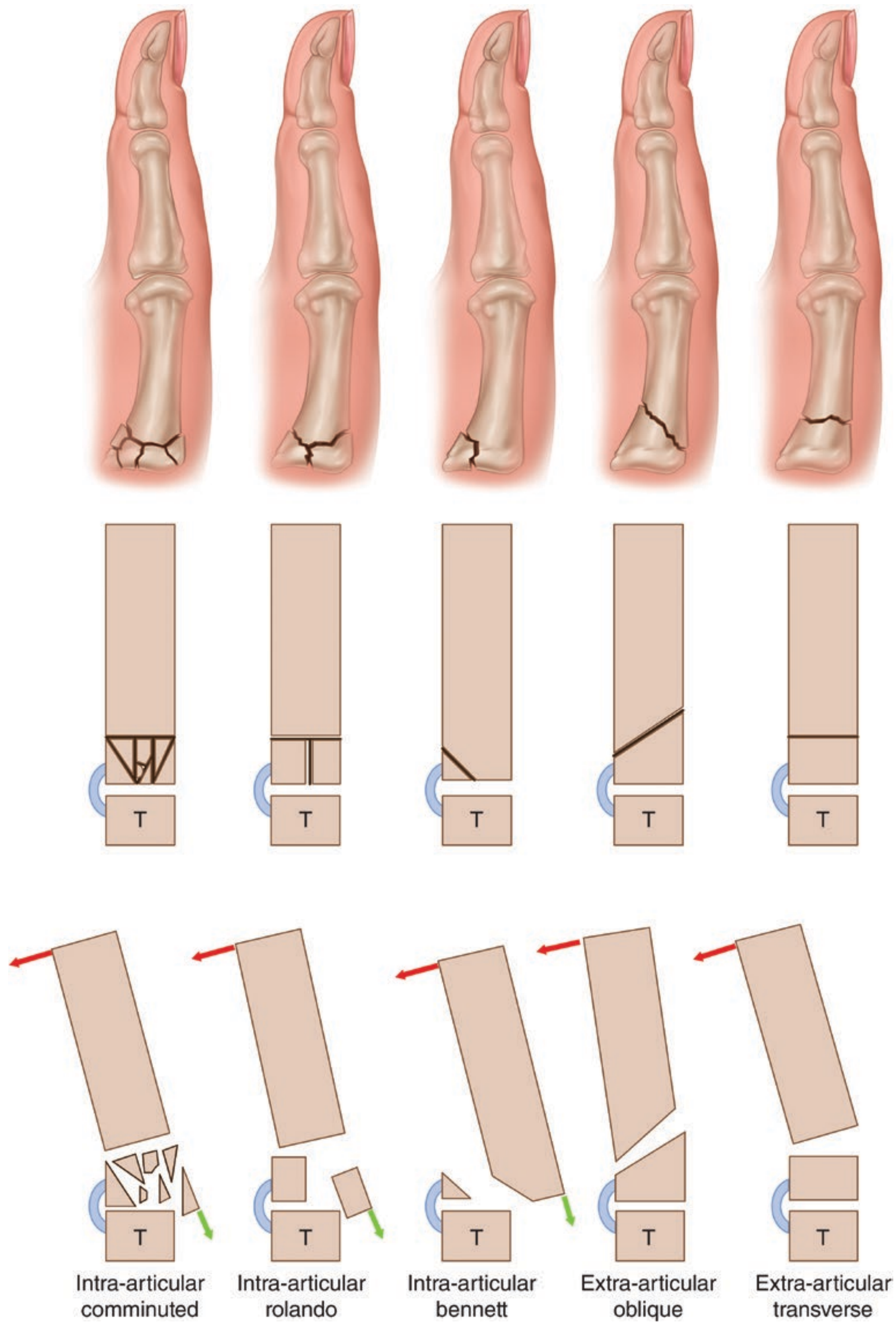
Complications

Joint stiffness from prolonged immobilization can be avoided with early gentle ROM [1, 11]. Intra-articular fractures increase risk of post-traumatic osteoarthritis. Malunion and malrotation are uncommon in athletes [1, 11].

Carpometacarpal Dislocations

Carpometacarpal (CMC) dislocations are rare and most often occur as a result of a high-velocity blow directly to the joint in sports such as skiing and cycling [13]. The third and fourth CMC joints are more stable with strong ligamentous attach-

Fig. 19.17 Classification of the base of the 1st metacarpal fractures. The blue line represents the oblique posteromedial ligament. Displacements of the intra-articular fractured part of first metacarpal in relation to trapezium (T) under the effect of the abductor pollicis longus (green arrow) and internal thenar muscles (red arrow)



ments and neighboring metacarpals acting as splints compared to the increased mobility of the first, second, and fifth CMC joints which cause the latter to be more vulnerable to dislocation. As such, dislocations most frequently occur at the first and fifth CMC joints (Figs. 19.23 and 19.24) [14, 15]. Usually the proximal metacarpal shaft is displaced dorsally. Due to significant soft tissue swelling, these dislocations can be difficult to assess clinically. A high degree of suspicion should be maintained in the context of significant swelling and tenderness at the CMC joints. If a dislocation is suspected, neurovascular status should be assessed due to the close proximity of neurovascular bundles and relatively common associated injury. Radiographic findings can be subtle, with overlapping metacarpals and carpals on the AP view and displacement more easily seen on the lateral view (Fig. 19.23). It is not uncommon for a fracture of the carpal bones to accompany a CMC dislocation. These fractures tend to be unstable and should be referred to a hand surgeon for reduc-

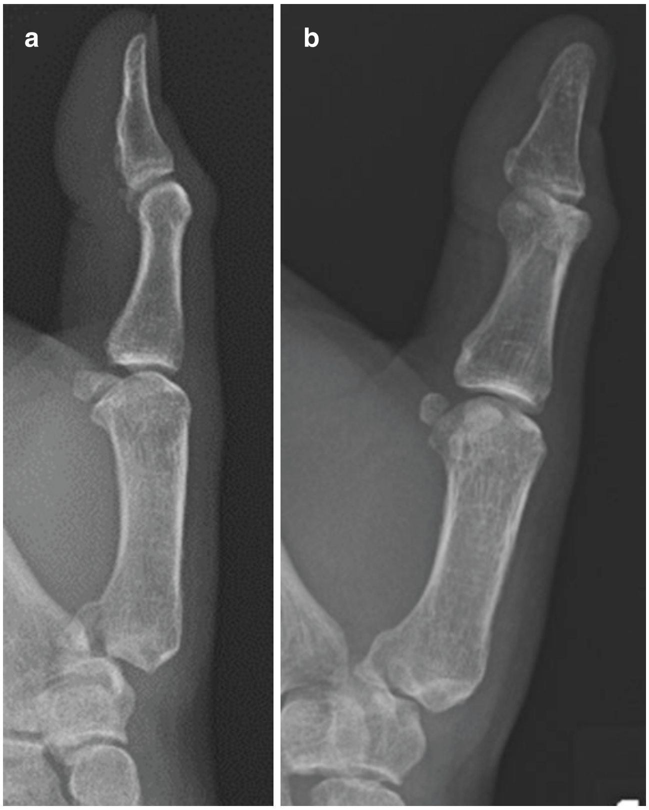


Fig. 19.18 Bennett's fracture of the 1st metacarpal base in a 45-year-old male (**a**, **b**)

Fig. 19.19 Rolando's fracture of the 1st metacarpal base in a 42-year-old female

tion and stabilization [14–16]. A volar splint or thumb spica splint is appropriate temporizing splinting methods.

Metacarpophalangeal Dislocations

Dislocations of the MCP joints are not common. Dislocations of the finger MCP joints most commonly occur dorsally as a result of a hyperextension injury causing disruption of the volar plate. They usually present with a hyperextension deformity, swelling, and pain over the joint [17]. With mild injuries, the MCP joint may only be subluxed, hyperextending 60°–90°. These injuries can usually be treated with closed reduction. More severe injuries involve full dislocation of the MCP joint without contiguity of the joint surfaces (Fig. 19.25). Reducible dislocations are called simple, while irreducible dislocations are called complex [17]. Unfortunately, the volar plate often interposes between the joint surfaces making closed reduction difficult (Fig. 19.25).

Reduction may be performed with the wrist and PIP joints in flexion. Hyperextension of the MCP joint is maintained, while volar pressure is applied to the base of the phalanx. Traction should be strictly avoided to prevent entrapment of the volar plate [17]. Splinting in slight flexion for 1–2 weeks followed by buddy taping with range of motion exercises is appropriate. Alternatively, buddy taping can be started immediately. Treatment should continue until full strength and range of motion is attained. Occupational therapy may be needed for recalcitrant cases. Referral to hand surgery should be considered for complex cases and for cases that cannot maintain reduction, or if there is an associated displaced fracture fragment or a fragment involving more than 20% of the joint surface [17].

Lateral and volar dislocations are much less common and involve injury to the collateral ligaments and volar plates. They usually present with tenderness and swelling over the collateral ligaments or volar aspect of the joint. They may have an associated avulsion fracture present. If no significant fracture is present, splinting in 60° of flexion for 3 weeks followed by 3 weeks of buddy taping can result in good outcomes [17]. If the fracture is displaced more than 2 mm or involves more than 20% of the joint surface, or significant instability persists, referral to a hand surgeon should be considered. Thumb MCP dislocations are treated similarly to finger MCP dislocations (Fig. 19.26) [17]. However, after reduction, care should be taken in assessing stability of the UCL, clinically and radiographically [18]. Treatment should then follow guidelines for a UCL injury [17].

Ulnar Collateral Ligament Injury

The most common ligament injury to the first MCP joint is a sprain or disruption of the ulnar collateral ligament (UCL), also known as skier's thumb (Figs. 19.27 and 19.28) [13, 18]. This injury is of particular note, as grip strength and stability rely upon this ligament's integrity. This injury occurs from forced abduction of the thumb. It commonly presents with pain and swelling over the ulnar aspect of the thumb MCP joint. Palpation elicits tenderness over the UCL area and may be associated with a palpable firm lump if a Stener lesion is present [13, 18]. Stener lesion is the displacement of the torn UCL above the adductor aponeurosis (Fig. 19.29) [13, 18]. Radiographs should be obtained before any stress testing is

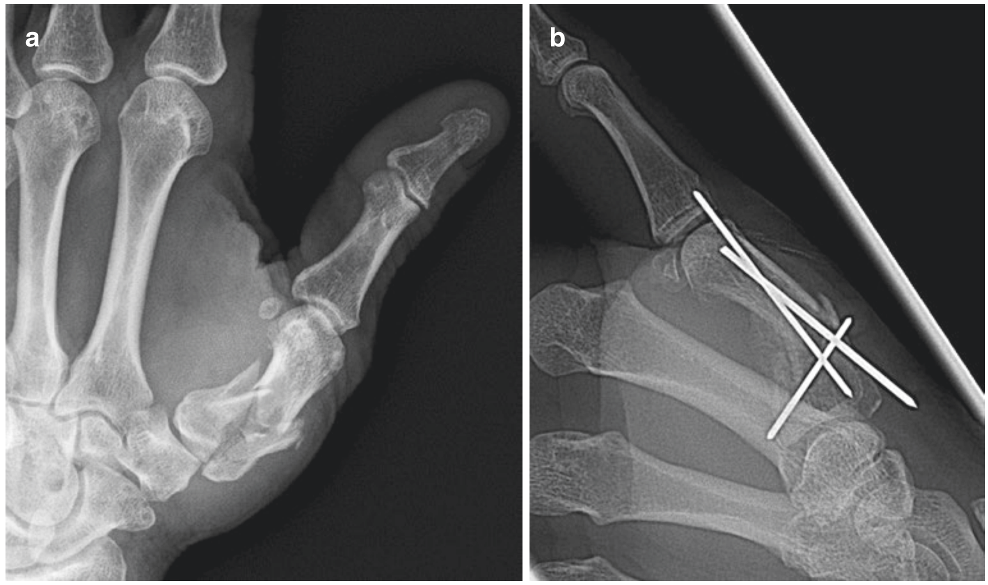


Fig. 19.20 Comminuted and intra-articular fracture of the 1st metacarpal base in a 66-year-old male (**a**). Postsurgical radiograph shows better anatomical alignment (**b**)

Fig. 19.21 Displaced transverse fracture of the 1st metacarpal base in a 20-year-old male as a result of a fall while skiing (**a**, **b**)

Fig. 19.22 Displaced, oblique, and comminuted fracture of the 1st metacarpal base in a 52-year-old (**a**, **b**)

performed. There can be an associated avulsion fracture of the distal end of the UCL (Figs. 19.27, 19.28, and 19.29). In acute settings, if there is an avulsion fracture present, stress testing should not be performed as this may lead to an iatrogenic Stener lesion [18]. If no Stener lesion is clinically present and no avulsion fracture is present on radiographs, stress testing of the UCL can be performed (Fig. 19.28). Greater than 30° abduction or greater than a 15° increase relative to the contralateral thumb indicates instability of the joint and the patient should be referred to a hand surgeon for consideration of reconstruction [18]. If there is less than 30° abduction or less than 15° increased abduction, then conservative management is appropriate [13, 18]. Casting in a thumb spica cast for 3 weeks followed by 3 weeks in a thumb spica splint allowing for gentle range of motion exercises is an appropriate regimen [13, 18].

Pediatric Considerations

Metacarpal fractures in pediatric population are relatively common [19, 20]. Accounting for 40% of hand fractures, metacarpal fractures are the most common hand fractures in pediatrics [20]. One important difference between pediatric and adult metacarpal fractures is the presence of open physes in children. Practitioners should remember that the physes of the second through fifth metacarpals occur at the distal end of the metacarpal at the metacarpal head, while the first metacarpal physis is at the base of the metacarpal. Similar to metacarpal fractures in adults, pediatric metacarpal fractures can be grouped by fracture location. Pediatric metacarpal fractures are more common among boys [21].

Metacarpal base fractures occur infrequently in pediatrics [20]. Fractures of the base of the second through fifth metacarpals may be buckle-type fractures or complete fractures. Reverse Bennett's fractures involving the base of the fifth metacarpal can occur and are usually considered unstable [20]. Management of metacarpal base fractures is similar to management in adult patients. Due to their stability, buckle fractures can be treated with an appropriate gutter splint, a moldable thermoplastic brace, or a short arm cast if needed. Displaced fractures or fracture dislocations of the carpometacarpal joint should be splinted and referred to a hand surgeon [20].

Metacarpal neck and shaft fractures in the pediatric population are similar in presentation, diagnosis, and treatment to adult fractures (Fig. 19.30) [20]. Metacarpal shaft fractures

Fig. 19.23 Posterior 4th and 5th CMC dislocation in a 22-year-old male (**a**, **b**)

often occur as a result of a rotatory force, with a spiral and oblique pattern to the fracture. As such, these fractures should be carefully examined for evidence of any rotational malformation. The remodeling potential for pediatric metacarpal shaft fractures is less than fractures of the metacarpal neck and decreases as the fracture moves proximally [20]. The acceptable amount of angulation for metacarpal neck fractures is 10°, 20°, 30°, and 40° for the second through fifth metacarpals, respectively. Metacarpal shaft fractures of greater than 10° of the second or third metacarpal or 20° of the fourth or fifth metacarpals should prompt consideration for reduction [20]. Traditionally, treatment has rested upon immobilization with radial or ulnar gutter splints or casts. There is some recent evidence that hand-based splints or casts may be as effective as ulnar gutter splints in children in treating metacarpal neck fractures [20].

Fractures of the metacarpal heads, particularly of the fourth and fifth digits, are most often Salter-Harris II fractures [20]. The collateral ligaments of the MCP joint also attach to the epiphysis and these can be avulsed along with a small fracture from the epiphysis (Fig. 19.24: epiphyseal avulsion fracture from metacarpal head). Nondisplaced or minimally displaced Salter-Harris II fractures or avulsion fractures of the collateral ligaments can be treated conservatively in a gut-

Fig. 19.24 Lateral 1st CMC joint dislocation in a 24-year-old male as a result of a snowboarding injury (**a**). Postreduction radiograph reveals anatomic alignment (**b**)

ter splint (radial or ulnar) with the MCP joints flexed to 90° and the PIP and DIP in full extension with the wrist slightly extended [20]. Displaced fractures should be referred to a hand surgeon for consideration of operative reduction. Immobilization is advised for 3 weeks followed by gentle mobilization while avoiding falls or contact injuries. Returning to sport after 6 weeks, if the fractures are clinically and radiographically healing well, is appropriate [20].

Similar to fractures in adults, first metacarpal fractures in children are treated separately from other metacarpal fractures. It is important to remember that the first metacarpal physis is at the proximal end at the base of the metacarpal. Commonly, first metacarpal fractures are Salter-Harris II with a metaphyseal fracture on the ulnar aspect (more common) or radial aspect (Fig. 19.31). Less commonly, a Salter-Harris III fracture (pediatric Bennett's) can occur, usually

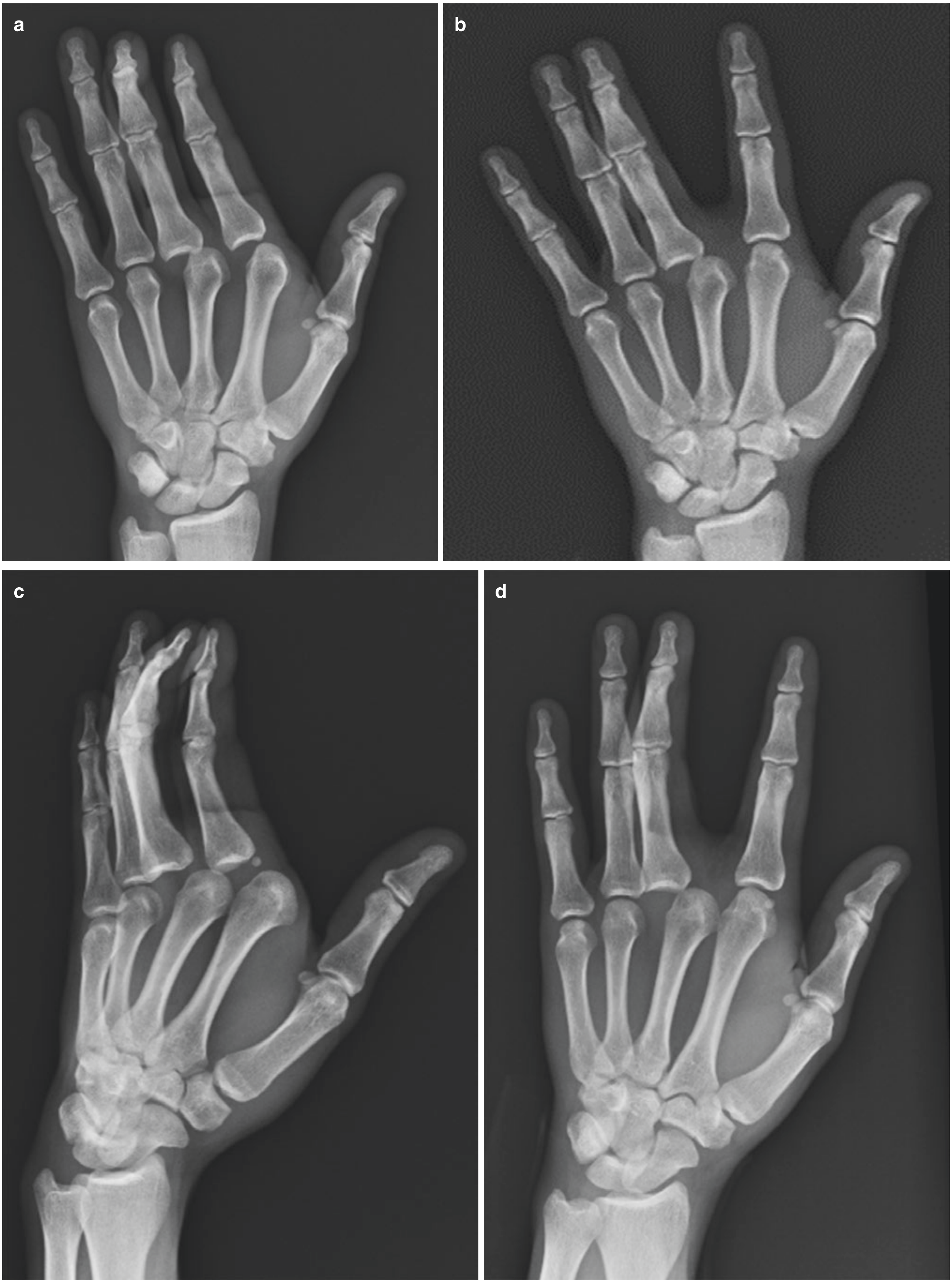


Fig. 19.25 Left posterior 2nd and 3rd MCP joint dislocation in a 25-year-old male skier (**a**, **c**, **e**). Only 2nd MCPJ was reduced with closed reduction (**b**, **d**)

Fig. 19.25 (continued)

with the epiphyseal fragment from the ulnar aspect of bone. The ulnar epiphyseal fragment may retain its normal position relative to the trapezium; however, the shaft of the metacarpal may displace radially and proximally. It is important to evaluate these fractures from multiple radiographic angles as there can be significant displacement and angulation of the metacarpal shaft that is visible only on one view. Transverse shaft fractures and stable Salter-Harris II fractures may be treated with a thumb spica cast for 3 weeks, followed by a thumb spica splint for 3 weeks, removing twice daily for range of motion exercises [20]. Unstable Salter-Harris II fractures or Salter-Harris III or Salter-Harris IV fractures should be splinted in a thumb spica splint and referred to a hand surgeon [20].

Return to Sports

Early range of motion after immobilization is very important for metacarpal fractures. Hand occupational therapist may be needed to regain range of motion, fine motor skills, and grip strength. Treatment should be individualized depending on type and location of injury, occupation, sport, and hobbies of patient. It typically takes 4–6 weeks for

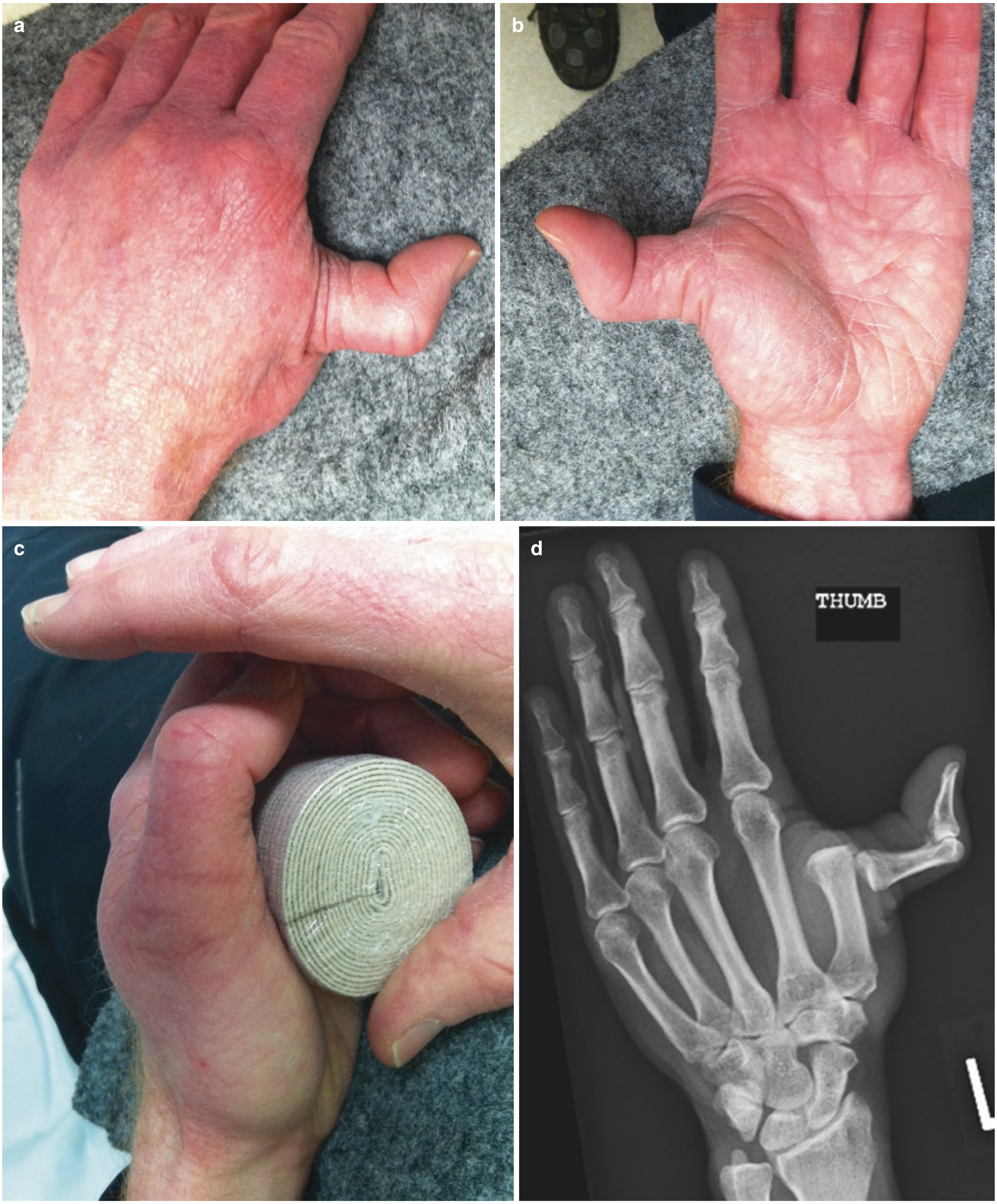


Fig. 19.26 Dislocation of the 1st MCP joint in a 57-year-old skier (**a**, **b**, **d**, **f**). Postreduction images reveal anatomic alignment (**c**, **e**, **g**)

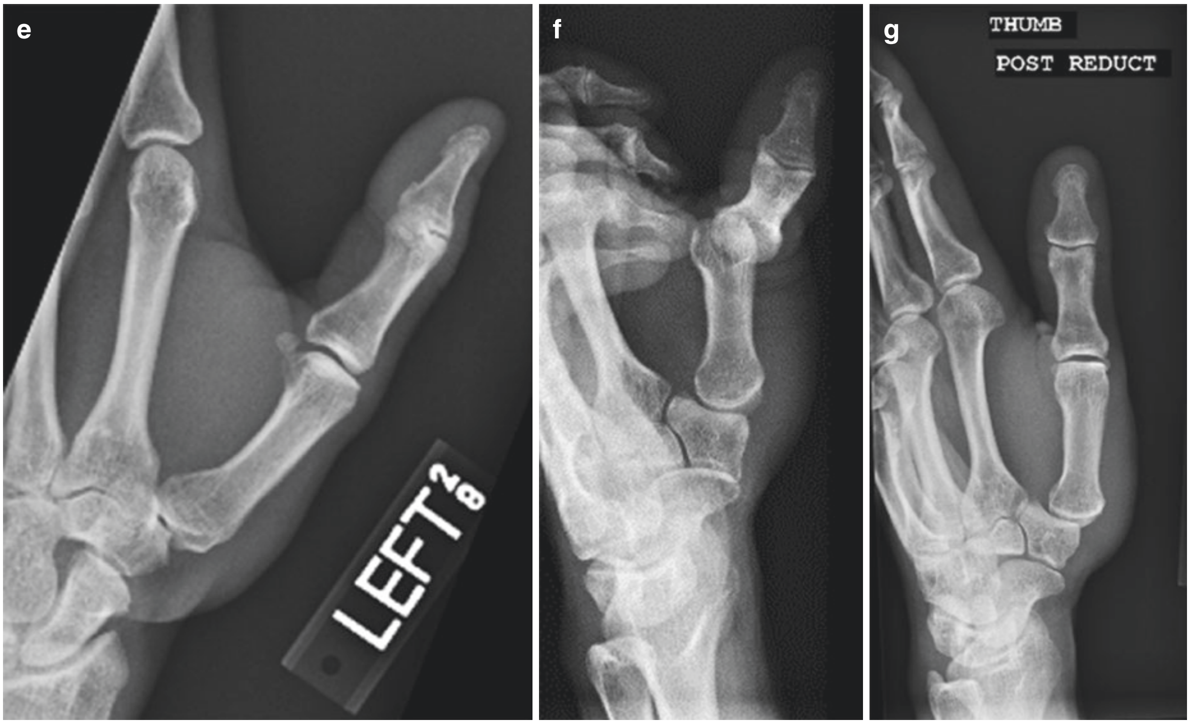


Fig. 19.26 (continued)

Fig. 19.27 Left 1st proximal phalanx avulsion fracture (arrow) due to a UCL sprain (skier's thumb) in an 18-year-old male as a result of a ski injury

clinical evidence of fracture healing and near-complete range of motion. Depending on fracture stability, the patient may return to activity sooner with cast in position of function (hold a ski pole). Cast will need proper padding if playing a contact sport. Functional and protective braces may be used for additional protection after cast is removed.

Practical Considerations

Bite wound over the MCP joint and metacarpal head from a fist fight has a high risk for infection and should not be sutured. The wound should be treated like an open fracture and the area needs to be explored, irrigated, and cultured if necessary.

If possible, remove rings immediately after injury prior to hand and finger swelling. Remove jewelry prior to imaging.

Complications

Metacarpal fractures can result in nonunion or malunion. Intra-articular fractures have increased risk of developing post-traumatic osteoarthritis. Prolonged immobilization may cause chronic stiffness, ligament shortening, and joint capsule contracture. A healed unacceptably dorsal angulated shaft fracture may cause irritation of the corresponding extensor tendon, prominent metacarpal head within the

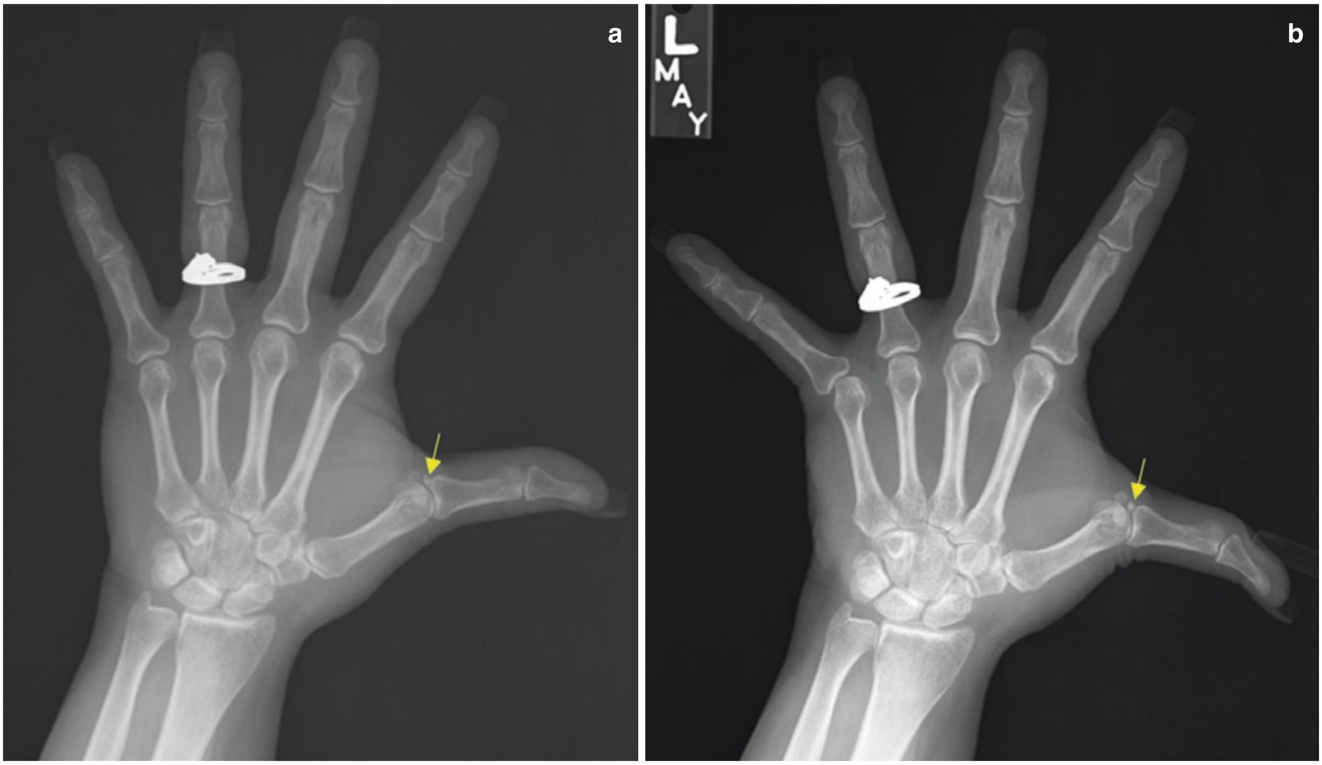


Fig. 19.28 An old left thumb injury in a middle-aged female. X-rays showed a small avulsion fracture at the 1st MCP joint (**a**). Stress view (**b**) confirmed laxity of the UCL of the 1st MCP (skier's thumb)

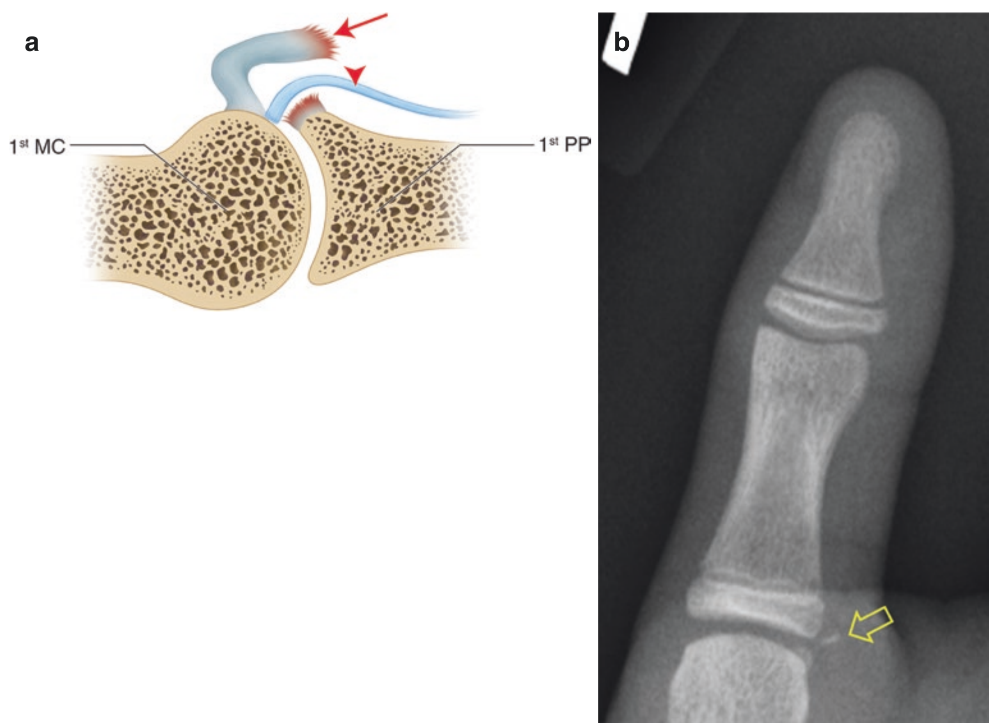


Fig. 19.29 Illustration of a displaced full-thickness 1st UCL tear (arrow) displaced above the adductor aponeurosis (arrowhead), Stener lesion toward 1st metacarpal (MC) (**a**). Avulsion fracture (open arrow) of the proximal part of the 1st proximal phalanx (PP) in an 11-year-old male (**b**)

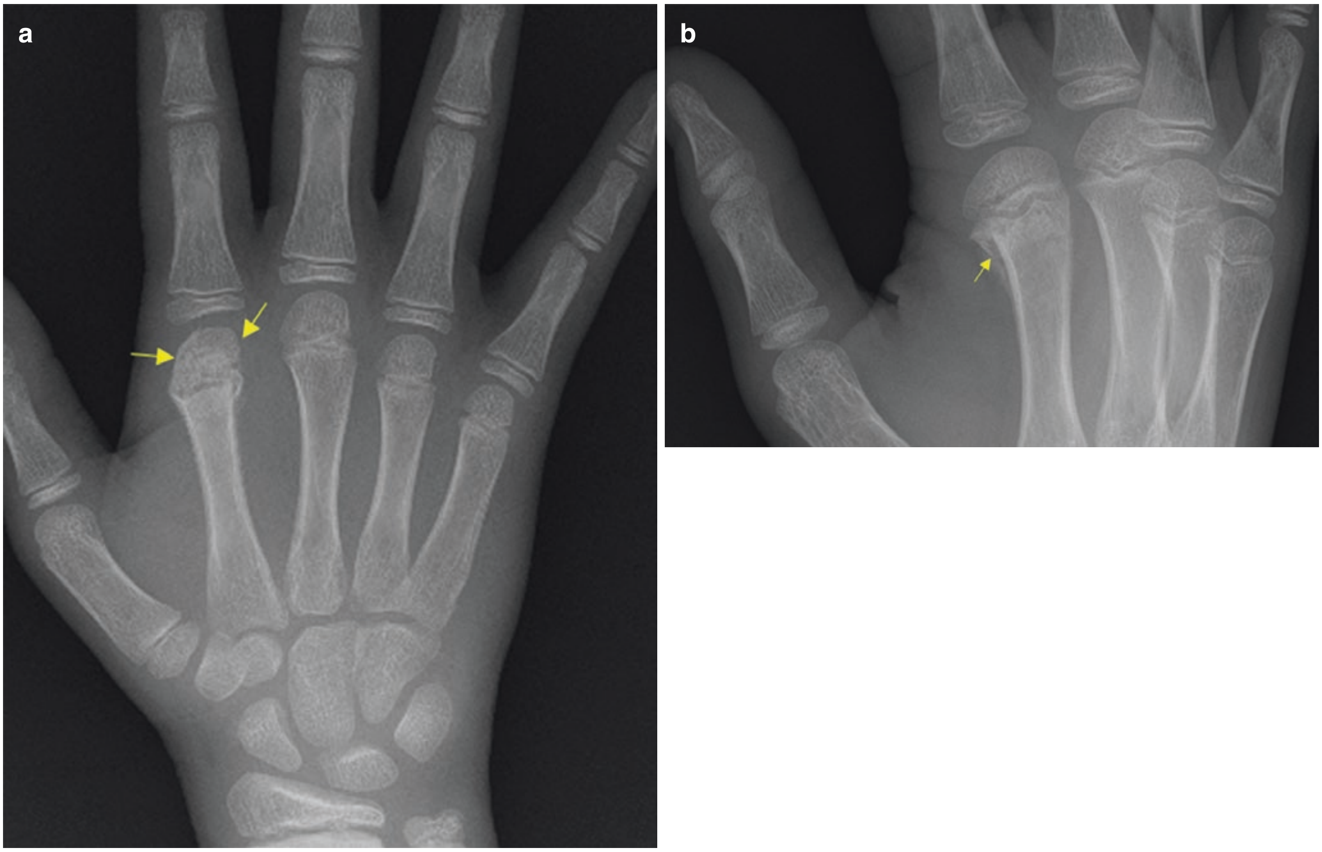
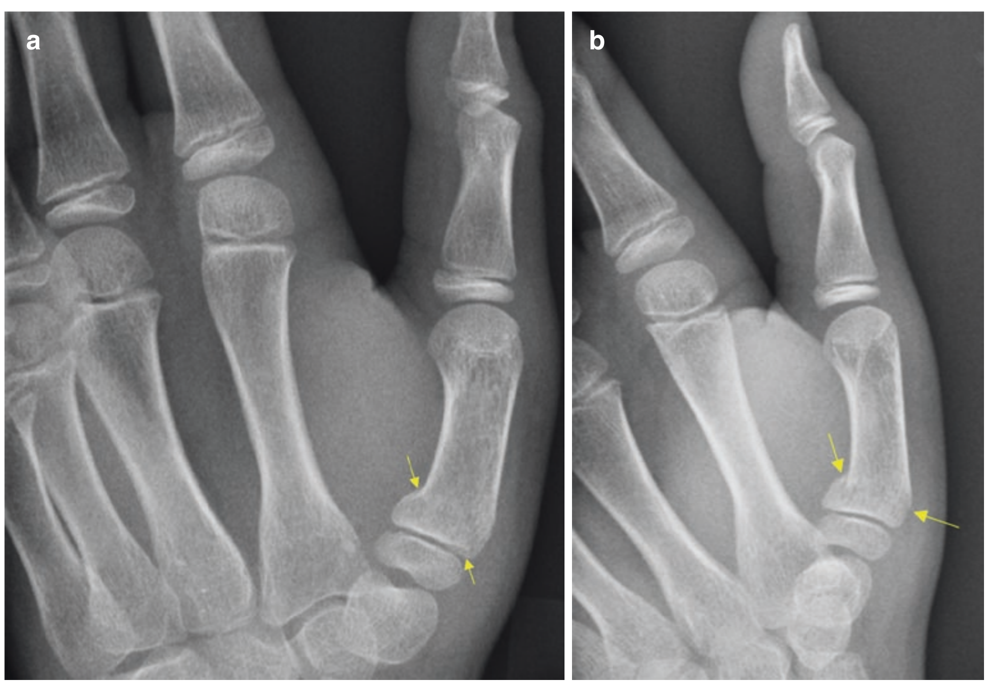


Fig. 19.30 Salter-Harris IV fracture of the 2nd distal MC (arrows) in an 11-year-old male (**a**, **b**)

Fig. 19.31 Salter-Harris II fracture of the 1st proximal MC (arrows) in a 12-year-old male (**a**, **b**)



palm, and pseudoclaw deformity. There are ten compartments within the hand; therefore, metacarpal fractures can increase the risk of compartment syndrome. However, this is very rare.

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Finger

20

Drew Ashby, Greg Gutierrez, and Morteza Khodaee

Key Points

- Detailed history of mechanism and manner of injury will guide the physical examination and differential diagnosis.
- Pediatric injuries can include growth plate injury as well as fracture.
- Orthopedic referral should be done for open fractures, joint disruption, irreducible fractures, significant displacement, and significant neurovascular injury.
- Use a relative rest approach to treating finger fractures, especially in active patients.

Introduction

Finger injuries are common in sports. Fingers are exposed and vulnerable at the end of the upper extremity. Understanding what is injured, and what could potentially be injured, is important to prevent deformity, dysfunction, and other long-term consequences such as osteoarthritis. These injuries are variable and consist of finger fractures and dislocations, ligament and tendon injuries, soft tissue injuries, nail and nail bed injuries, and amputations. The exact prevalence of finger injuries in sports is unknown. Finger injuries are more common in high-speed sports and sports requiring hand use such as skiing, baseball, lacrosse, and boxing.

D. Ashby
Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

G. Gutierrez
Department of Family Medicine, University of Colorado School of Medicine, Denver Health, Winter Park Medical Center, Winter Park, CO, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Fractures

Finger fractures are common in both sports- and non-sports-related injuries. They account for approximately 10% of all fractures presenting for care [1]. About 30% of phalangeal fractures in adults and adolescents occur because of sports [2]. Fractures of finger phalanges are the most common fracture in basketball, snowboarding, hockey, karate, ice skating, horse riding, and skiing and second most common fracture in football and rugby [1]. The first and fifth rays were the most commonly injured fingers [2]. Notably, they also account for a 25% of all missed fractures in the emergency room [3]. All finger fractures should be evaluated for infection and non-union in 1–3 weeks. Most hand and finger fractures do not require formal rehabilitation after healing of the fracture site. If, however, the fracture involved the joint, or required surgical intervention to repair, rehabilitation may be required to regain strength and range of motion. In any case, if range of motion and strength fails to return to appropriate levels within a reasonable amount of time after fracture healing, physical therapy intervention may be indicated to restore function [4, 5].

Proximal and Middle Phalanx Shaft (Diaphysis) Fractures

Fracture Classification

In general, other standard long bone fracture categories can be applied to these phalangeal shaft fractures. Shaft fractures are usually extra-articular fractures (Figs. 20.1, 20.2, 20.3, and 20.4). They can be transverse (Fig. 20.2), oblique (Figs. 20.3 and 20.5), and spiral. Occasionally, an oblique shaft fracture can extend into a proximal or distal joint (Figs. 20.5, 20.6, and 20.7).

Mechanism of Injury in Sports

Fall, direct blows, axial, bending, or twisting forces to the finger are usually the cause of these fractures. Crush injuries

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Fig. 20.1 Displaced fracture of the shaft of the 2nd proximal phalanx in a 36-year-old male

can also be responsible for these fractures, particularly the comminuted fractures.

Epidemiology

Phalangeal fractures are the most common fracture of the hand. The proximal phalanx of the third finger is more commonly fractured compared with the middle or distal phalanges [6].

Clinical Presentation

Athletes usually present with deformity given flexor and extensor forces. Pain with ROM, ecchymosis, edema, and tenderness are typical with phalangeal fractures. A complete neurovascular examination should be performed.

Diagnosis

PA and lateral radiographs with added oblique especially if articular surface may be involved are generally adequate. If clinical concern warrants, CT can be obtained to evaluate in more detail.

Initial Management

Although small amounts of angulation may be acceptable in the general population, near anatomic alignment may be necessary for athletes [6–9]. Postreduction, non-displaced, and stable fractures can be managed nonoperatively with

Fig. 20.2 Transverse fracture of the left 2nd proximal phalanx in a 38-year-old female (**a**, **b**)

Fig. 20.3 Mildly displaced oblique fracture of the 4th proximal phalanx shaft as a result of a mountain bike injury in a 41-year-old female (**a**, **b**)

Fig. 20.4 Fracture of the 5th middle phalanx shaft in a 25-year-old male snowboarder (**a**, **b**)

immobilization of the affected digit and at least one adjacent digit in 70°–90° of MCP joint flexion and with the PIP and DIP joints in extension for 1–3 weeks [6–10].

Indications for Orthopedic Referral

Unstable, irreducible, multiple digits, and open fractures and fractures associated with tendon lacerations should be referred to orthopedic surgery [6–10].

Follow-Up Care

Clinical and radiographic evaluation is recommended in 1–3 weeks. At this point splinting and buddy taping can be applied for few more weeks [6–10].

Return to Sports

Athletes can return to play in a protective splint as soon as the pain permits. Obviously, this depends on their position in specific sports and ability to function with the protective splint [8–10].

Complications

Unstable or irreducible fractures can lead to significant finger deformity and hand function impairment unless surgically treated [6, 8–10]. Nonunion and infections are rare in athletic population.

Fig. 20.5 Intra-articular and mildly displaced fractures of the distal shaft of the 4th middle phalanx as a result of a mountain bike injury in a 46-year-old female (**a**, **b**)

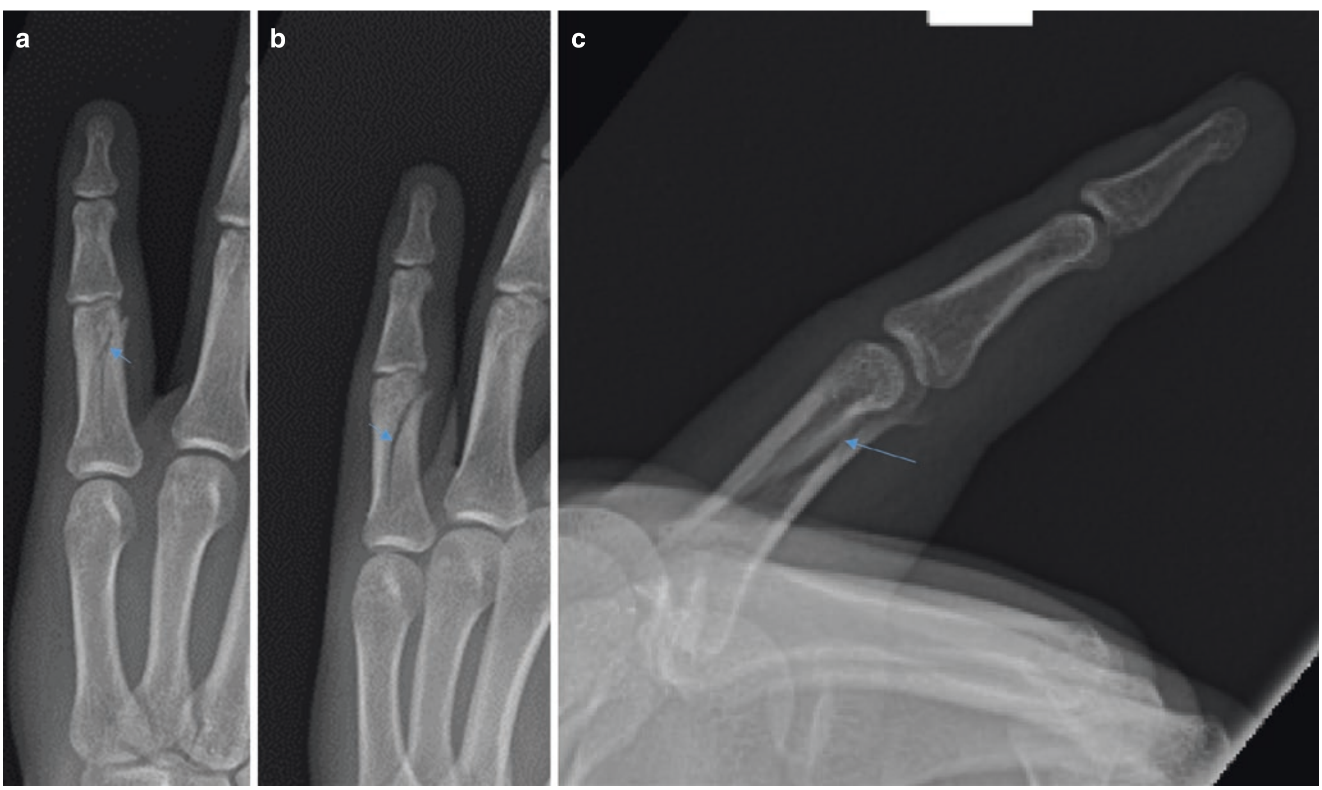

Fig. 20.6 Fracture of the 5th proximal phalanx shaft (arrows) in a 27-year-old male skier (**a**, **b**)

Fig. 20.7 Oblique fracture of the 4th middle phalanx shaft in a 30-year-old male mountain biker (**a**, **b**)

Pediatric Consideration

The further the fracture site is from the physis, the more the potential for decreased bone remodeling (Fig. 20.8). Salter-Harris II is the most common type of pediatric fractures (Fig. 20.9). Therefore, surgical treatment may be the optimal treatment for pediatric patients who have a displaced phalangeal neck fracture [11]. Salter-Harris III (reported in climbing athletes) and Salter-Harris IV and V epiphyseal fractures of the middle and proximal phalanx should be referred to a hand surgeon [12].

Proximal and Middle Phalanx Intra-articular Fractures (Base and Condylar)

Intra-articular fractures can be at the base or condylar parts of the phalanges.

Fracture Classification

Intra-articular base fractures can be classified into collateral ligament avulsion fractures, compression (plateau) fractures, and vertical shear shaft fractures [13]. Condylar fractures can be divided into type I (non-displaced and stable) (Figs. 20.10 and 20.11), type II (unstable and unicondylar) (Figs. 20.12 and 20.13), and type III (unstable and bicondylar) [4, 5, 8].

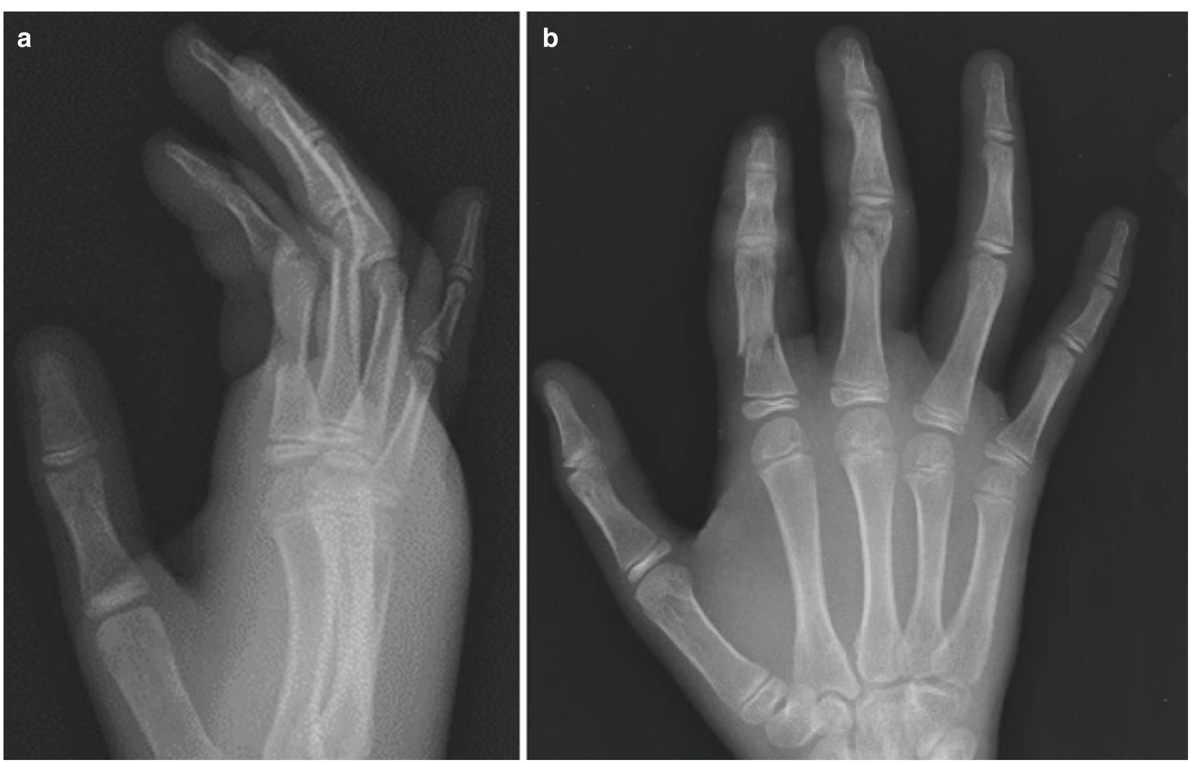

Fig. 20.8 Displaced fractures of the 2nd proximal phalanx shaft and distal part of the 3rd proximal phalanx as a result of a mountain bike injury in a 12-year-old boy (**a**, **b**)

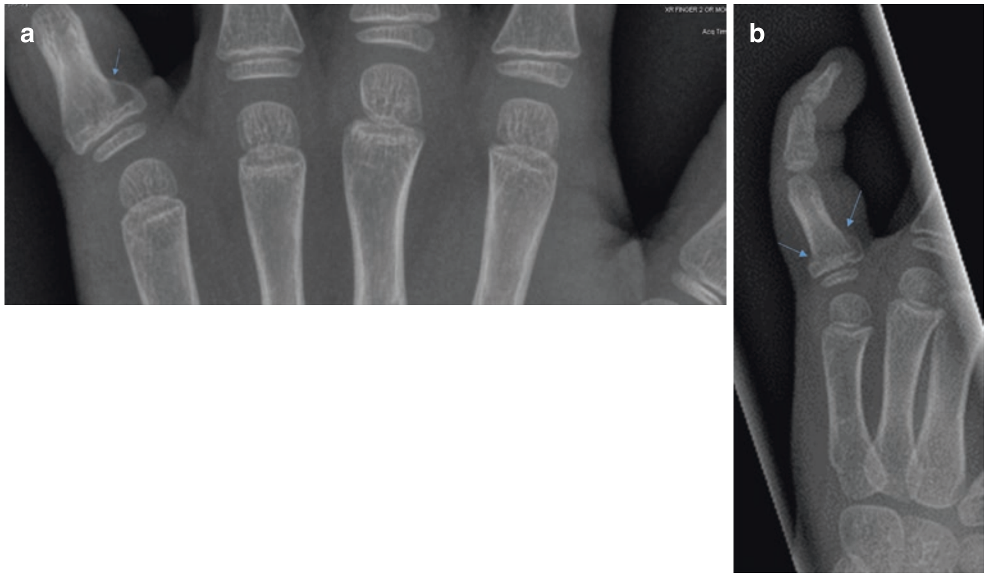

Fig. 20.9 Salter-Harris II fracture (arrows) of the 5th proximal phalanx in a 7-year-old boy (**a**, **b**)

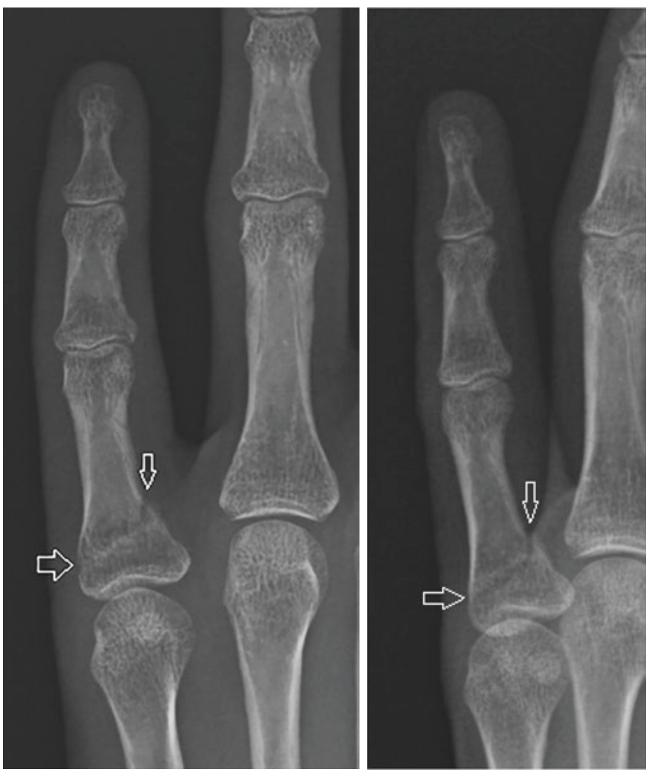

Fig. 20.10 Fracture of the base of the 5th distal phalanx (arrows) in a 26-year-old female snowboarder (**a**, **b**)

Fig. 20.11 Right 3rd proximal phalanx condylar fracture (arrow) in a 67-year-old male

Mechanism of Injury in Sports

Proximal phalangeal base fractures result from a finger being abducted beyond the acceptable limits of the MCP joint usually from sports injuries or a FOOSH [4, 5, 13]. Middle phalangeal base fractures are usually due to hyperextension, hyperflexion, or lateral deviation force on an outstretched finger (e.g., basketball and volleyball injuries), or from a FOOSH [4, 5, 13]. Condylar fractures are usually the result of direct axial compression, avulsion forces, or subchondral shearing [4, 5, 13].

Fig. 20.12 Intra-articular fracture (arrow) of the base of the right 5th proximal phalanx in a 50-year-old female

Epidemiology
Generally, finger fracture location prevalence is not noted; however, Salter-Harris II fractures of the proximal phalanx base are the most common type of finger fracture in the pediatric population [14].

Clinical Presentation
Proximal base fractures generally present after axial impact on extended finger [4, 5]. Athletes are usually unable to move PIP or DIP joint of the affected digit [4, 5]. Patients with condylar fractures generally present with a painful PIP or DIP joint with an associated soft tissue injury [5].

Diagnosis
Condylar fractures can be misdiagnosed as a subluxation or dislocation [8]. Therefore, plain radiography should be used to fully evaluate any suspected digital dislocations and fractures. Plain radiographs are generally adequate following physical examination. However, CT imaging should be considered in cases of high clinical suspicion without clear signs of fracture on X-ray [4, 5, 15].

Initial Management
Non-displaced intra-articular base or condylar fractures can be managed nonoperatively [7, 8]. All intra-articular type 2–3 condylar fractures are unstable and should be splinted and referred to orthopedic surgery [4, 5, 7, 8]. In general, intra-articular base fractures with more than 30% of joint involvements are unstable. Mildly displaced stable intra-articular base fractures may be managed with initial reduction [5, 7, 8]. This can be attempted using a regional, hematoma, or ring block, followed by splinting [5, 7, 8]. Extension block splinting is recommended for any stable fracture and dislocation of the articular surface [5]. Close follow-up with plain radiography is essential to assess for fracture stability [5, 7, 8].

Indications for Orthopedic Referral
Any unstable intra-articular base or condylar fracture should be referred to an orthopedic surgeon [4, 5, 7, 8]. Urgent referral is only indicated if there is any neurovascular compromise or there is any open fracture component.

Follow-Up Care
Athletes who are treated nonoperatively should be assessed first weekly and then biweekly for any displacement during immobilization until the clinical and radiographic union is achieved [4, 5, 7, 8].

Return to Sports
Depending on the sport and the position played in the sport, athletes may return to play with a protective brace/splint or when clinical union has occurred [4, 5, 7, 8].

Complications
Chronic pain, stiffness, and secondary arthritis are possible complications [8]. Fractures that are unstable or displaced have high rates of flexion contracture [4].

Pediatric Consideration
Orthopedic referral is recommended for any joint involvement (Figs. 20.14 and 20.15) or unstable fractures to limit loss of function [4, 5, 8, 16].

Distal Phalanx Fractures

Fracture Classification
Distal phalanx fractures are classified into tuft, shaft, and articular fractures. Articular fractures can be small as an avulsion fracture (Fig. 20.16). Tuft fracture is when there are small fractured bone fragments off the distal edge of the distal phalanx

Fig. 20.13 Displaced, intra-articular, and comminuted fracture of the 5th middle phalanx base in a 39-year-old male (**a**, **b**)

(Figs. 20.17 and 20.18). These fractures are often associated with fingertip soft tissue injuries (e.g., nail bed injuries). Seymour fractures are displaced distal phalangeal fractures that involve the physis with an associated nail bed laceration [17].

Mechanism of Injury in Sports

Distal phalanx fractures generally occur with crush injury, direct blow from a stick, or collision with another player [8, 18, 19]. In the thumb, crush injury is the most common mechanism [4].

Epidemiology

The distal phalanx is the most common location for sports-related hand fractures [2]. The most common type of distal phalanx fracture is the tuft fracture [19].

Clinical Presentation

Athletes usually present with distal finger tenderness, swelling, discoloration, decreased range of motion, numbness, weakness, and bleeding. It is not unusual to have associated nail and nail bed injury [4, 18].

Diagnosis

Following clinical examination, plain radiography is used for the diagnosis [4, 7, 18].

Initial Management

Distal phalanx fractures are usually stable and can be treated with simple immobilization of the DIP joint with a splint for 2–4 weeks (2–3 weeks in pediatric patients). Early DIP ROM exercises are recommended as pain permits [4, 7, 19].

Fig. 20.14 Distal 5th middle phalanx fracture (arrows) in a 10-year-old boy (**a**, **b**)

Unstable shaft and displaced fractures should be reduced before splinting [7, 19, 20]. Nail bed injuries and subungual hematomas should be explored and treated appropriately or referred to a hand surgeon [7, 19, 20]. Seymour fractures and fractures with concurrent nail bed injuries should be treated as an open fracture [7, 17, 19–21].

Indications for Orthopedic Referral

Depending on the comfort level of the treating physician, an orthopedic referral may be necessary if there is any significant soft tissue injury. In addition, irreducible and unstable fractures and those with more than 30% articular involvement should be referred to an orthopedic surgeon [4, 7, 17, 18, 21].

Follow-Up Care

Athlete should be evaluated in 2 and 4 weeks. Plain radiography of an asymptomatic athlete is not necessary as the bony union may occur months later.

Return to Sports

After 2 weeks of immobilization for nonoperative fractures, the athlete can return to sports with protective splint as tolerated [4, 7, 17, 18, 21].

Complications

Infection among healthy athletes is very rare. As most of these fractures are healed by fibrous union, radiographic bony healing may not occur for a long time [7]. Sensory loss and paresthesia in the fingertip may be present up to 6 months after many of these injuries [4, 19, 21]. In rare cases, the DIP joint may develop arthritis requiring fusion and symptomatic osteonecrosis of the distal fragment requiring excision [4, 21].

Pediatric Consideration

In general, phalangeal fractures in pediatrics heal faster compared to similar fractures in adults. Tuft fractures in pediatric patients are at risk for growth arrest with physeal injuries and require thorough evaluation and possibly an orthopedic referral (Fig. 20.19). Seymour fractures may be mistaken for bony mallet. Orthopedic consultation may be necessary [17].

Thumb Fractures

Fractures of the thumb phalanges (Fig. 20.20) have no distinct difference in treatment from fractures of phalanges of digits 2–5 [22]. Tuft fractures of the thumb should be splinted for 4 weeks with early range of motion and rehabilitation [4, 22]. For transverse shaft fractures, careful evaluation should be taken as they might be unstable secondary to pull of the FPL on the proximal fragment. For proximal fractures, less than 20° of angulation in the lateral plane is acceptable and can be managed with immobilization. Displaced transverse fractures are usually stable after closed reduction and can be managed with splinting [4, 22]. Splinting non-displaced fractures of the proximal phalanx for up to 4 weeks, followed by early range of motion, is a reasonable treatment course [4, 22].

Fig. 20.15 Salter-Harris III fracture of the 1st proximal phalanx in a 15-year-old boy as a result of a snowboarding injury (**a**). The fracture was surgically fixed (**b**, **c**)

Finger Joint Dislocations

The PIP and DIP joints can sublux (Fig. 20.21) or dislocate as a result of injuries. The most common finger dislocation occurs at the PIP joints (Figs. 20.22, 20.23, 20.24, and 20.25), followed by DIP (Fig. 20.26) and first IP (Fig. 20.27) joints. The dislocations are almost always dorsal (Figs. 20.27 and 20.28), but lateral (Figs. 20.24 and 20.25), volar (Fig. 20.26), and complex (Figs. 20.22 and 20.23) dislocations can occasionally happen. Some dislocations may be associated with fractures particularly with volar plate fractures (Figs. 20.21 and 20.27).

Mechanism of Injury in Sports

Finger dislocations occur during hyperextension or hyperflexion of the finger or with an axial load to the finger. This type of injury mechanism is common with many sports-related activities. For example, this type of mechanism happens easily while attempting to catch a ball or hold on to another player such as in football. In one study, the ring and long fingers were the most affected by athletes [23].

Epidemiology

Finger injuries account for a large portion of emergency room visits, with lacerations and fractures being the most common diagnosis. The prevalence of dislocations as the primary presenting problem appears to be about 2–3%; however, the incidence in sports-related injuries remains unclear as many of these injuries appear to be treated outside emergency department care [24].

Clinical Presentation

Patients present to a variety of care settings including urgent care, emergency care, and primary care locations. They will present with a painful digit with deformity and loss of motion. They can occur at the PIP, DIP, or first IP joints [20].

Fig. 20.16 Avulsion fracture of the dorsal and proximal part of the 4th distal phalanx (arrow) in a 42-year-old female

Diagnosis

Generally, initial clinical examination with 3-view plain radiography is appropriate for diagnosis [20]. Often time a dislocation may not be obvious in AP view.

Initial Management

Dorsal finger dislocations should be managed with reduction and immobilization. PIP reduction should be attempted on the sideline if possible [20]. This is generally accomplished by applying traction to the distal phalanx and volar pressure on the middle phalanx at the PIP joint [19, 20, 23]. If pain limits reductions, a digital block with local anesthetic should be used to allow for timely reduction. Following reduction, X-rays should be completed to confirm position and the finger should be splinted for 1–3 weeks with early range of motion and buddy taping for another 1–2 weeks [19, 20, 23]. For volar or lateral dislocation, immediate reduction with the above method is recommended (traction and counterpressure either dorsal or medial, respectively) with 6 weeks of splinting in extension to prevent long-term complications [20].

Indications for Orthopedic Referral

Urgent orthopedic referral and evaluation are necessary for any irreducible or open dislocations [20]. Athletes with comminuted fracture-dislocations, with more than 25% of joint involvement, or significantly displaced fractures should be seen by orthopedic surgery within a few days [20, 23].

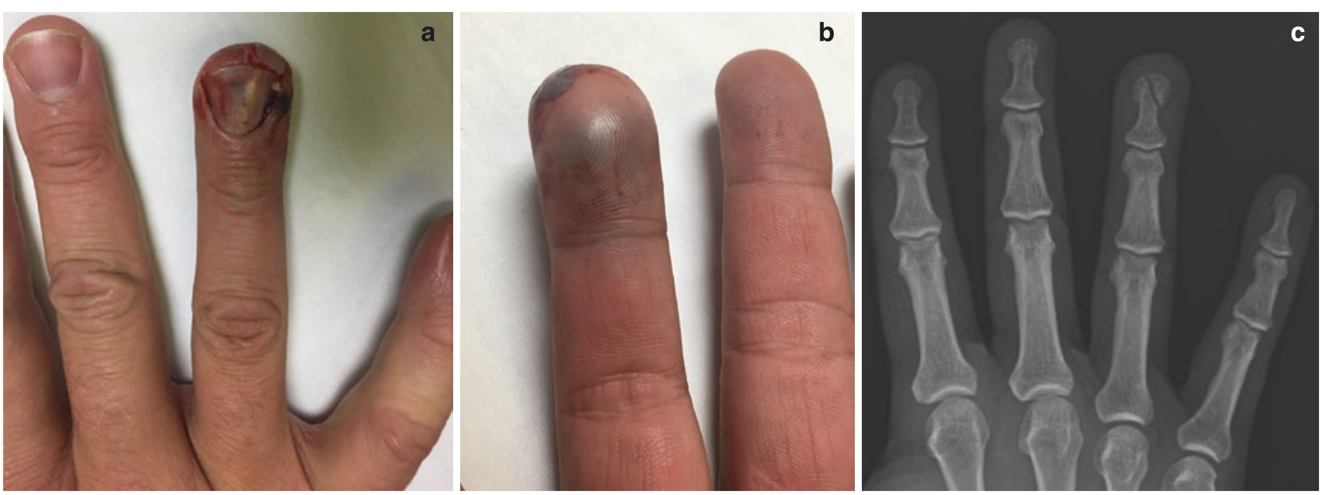

Fig. 20.17 Distal (tuft) fracture of the 4th distal phalanx in a 46-year-old male as a result of a tree strike while riding a snowmobile (**a**–**c**)

Fig. 20.18 Laceration and tuft fracture of the 2nd finger as a result of a snowmobile injury in a 28-year-old female (**a–d**). The laceration was repaired with loose sutures (**e**)

Follow-Up Care

Early range of motion has been shown to improve post-dislocation function. Full gentle flexion is recommended at 1 week. Full range of motion exercises should be started at about 3 weeks [20].

Return to Sports

Athletes can return to play postreduction with appropriate immobilization and protection of the affected finger. If this is not feasible due to functional limitation, then they can return with buddy taping in 3–4 weeks postreduction [19, 20].

Complications

Finger dislocation can result in limited range of motion, loss of function, or chronic pain if there is a delay in reduction or inappropriately treated [19, 20, 23].

Pediatric Consideration

Dislocations are not very common in pediatric population (Fig. 20.28). If there is a concern for physeal plate disruption (Salter-Harris III, IV, V), orthopedic referral is indicated [16, 19, 20, 23].

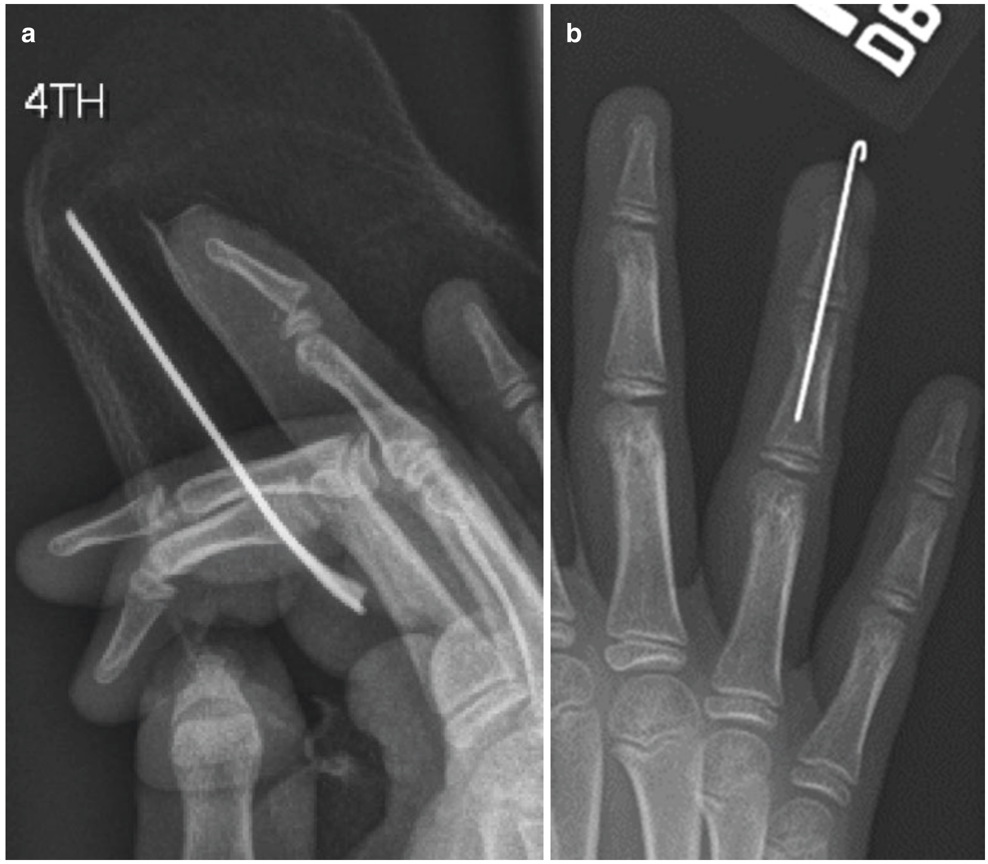

Fig. 20.19 Displaced Salter-Harris II fracture of the 4th distal phalanx (**a**) in a 10-year-old boy, treated with pinning (**b**)

Fig. 20.20 Comminuted and intra-articular left 1st proximal phalanx fracture in a 42-year-old male as a result of a ski fall

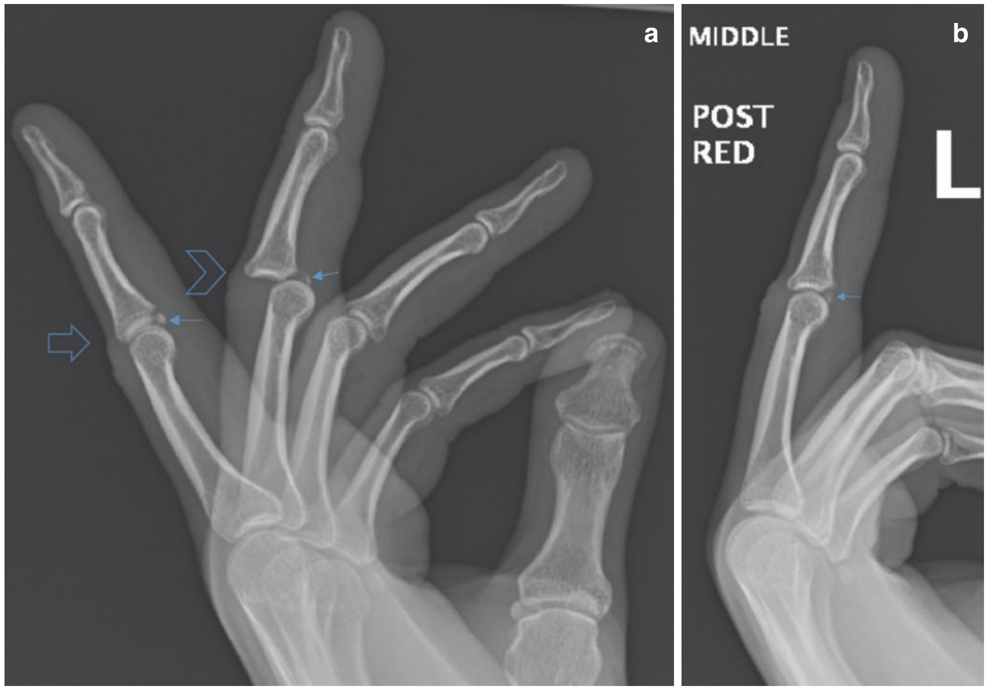

Fig. 20.21 Posterior subluxation of the 2nd and 3rd (arrowhead) PIP joints in a 31-year-old male mountain biker (**a**) with volar plate fractures (arrows). The 2nd PIP was reduced spontaneously before the X-ray was taken (open arrow). The 3rd PIP was reduced (**b**)

Tendon and Ligament Injuries

Sports-related finger tendon and ligament injuries are common. Injuries to tendons and ligaments should be managed with 1 week of external support and edema control followed by protected mobilization and tendon gliding [4]. Clinical examination is generally sufficient; however, X-ray can be utilized when there is concern for avulsion or other fractures. Progressive supervised rehab is recommended and usually lasts for 8 weeks post injury. Most fractures involve some soft tissue injury given the location of soft tissue structures and bony structures in the fingers [4, 25].

Mallet Finger

Mechanism of Injury in Sports

Mallet finger occurs with an axial loading followed by hyperflexion/hyperextension injury to the DIP joint of the finger. In sports, it is generally caused by high impact to the affected digit usually related to impact to the hand from a ball [25–28].

Epidemiology

Mallet finger is a common injury especially in young to middle-aged athletes [25–28]. Overall incidence was 33.2 per 100,000 with a prevalence of 9.3% of all tendon and ligament lesions in the body and an incidence of 5.6% of all tendinous lesions in the hand and wrist [29].

Classification

Multiple classifications exist for mallet finger injury. There is limited consensus on the clinical utility of these classifications [26–28]. Generally, Doyle's or Tubiana's classifications are most commonly used and are based on fracture location, size, and articular surface involvement [27].

Clinical Presentation

Athletes generally present with a flexion deformity of the DIP joint (Fig. 20.29), painful swollen fingertip, and a history of high-energy impact or crush injury to the affected hand. Inability to perform active extension at the DIP joint is a common finding [26–28].

Diagnosis

Clinical history combined with standard 3-view X-ray of the affected digit to evaluate for avulsion fractures is usually adequate [26–28].

Initial Management

Management of mallet finger injury including the thumb is a full-time splint that keeps the DIP joint in mild hyperextension (<10°) for 6–8 weeks (6 for bony mallet fingers, 8 for tendon injuries without bony avulsion) [26, 28, 30, 31]. This is usually followed by a 4-week period of part-time (night) splinting, although recent studies have not shown this to change functional outcomes [26, 30, 31].

Fig. 20.22 Posterior-ulnar dislocation of the 5th PIP joint in a 20-year-old male snowboarder (**a**, **c**, **e**). Postreduction images confirm anatomical alignment (**b**, **d**, **f**)

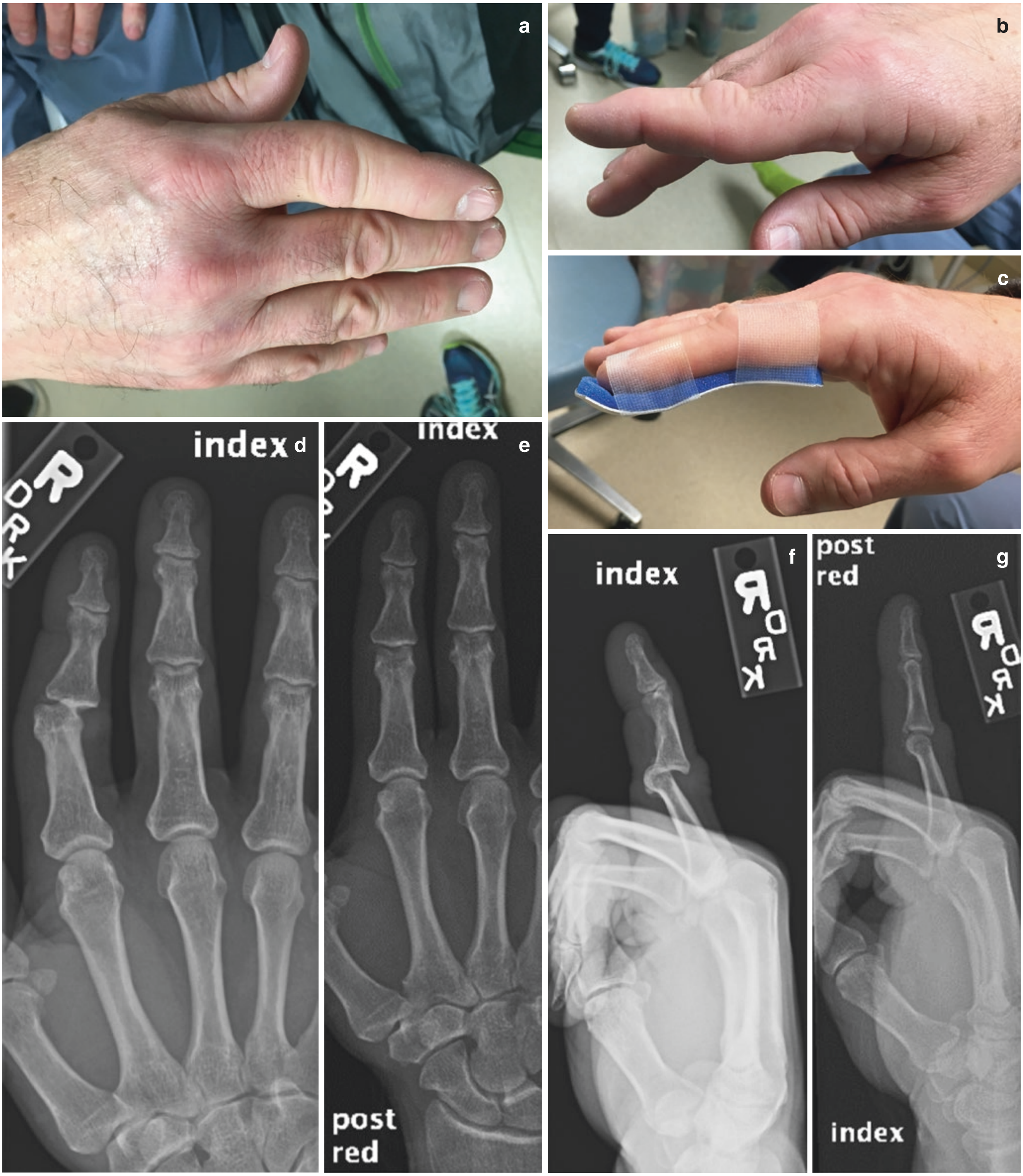

Fig. 20.23 Posterior-ulnar 2nd PIP joint dislocation in a 54-year-old male as a result of a fall while skiing (**a**, **b**, **d**, **f**). Postreduction images also revealed volar avulsion fracture (**c**, **e**, **g**)

Fig. 20.24 Ulnar dislocation of the 3rd PIP joint in a 30-year-old male mountain biker (**a**). Anatomical alignment was achieved postreduction (**b**)

Indications for Orthopedic Referral (Urgent and Nonurgent)

Generally, there is no need for urgent referral, unless there is an open fracture. However, referral to a hand surgeon is recommended for any failed nonsurgically treated injuries, irreducible bony avulsions, or fracture involving >1/3 of the articular surface [26, 27, 30, 31].

Follow-Up Care

Follow-up radiography usually is not necessary, unless there is concern for nonunion. Athletes should be evaluated clinically in 4 and 8 weeks post injury [26–28].

Return to Sports

In general, athletes can return to sport when they are asymptomatic, have functional active range of motion, and demonstrate a good grip strength (>80% relative to the uninjured side) [32].

Complications

Neglecting mallet fingers can lead to permanent fingertip disfigurement (swan neck deformity), dorsal DIP pain and inflammation, and restricted DIP extension [27].

Flexor Digitorum Profundus Avulsion Fracture (Jersey Finger)

Mechanism of Injury in Sports

Flexor digitorum profundus (FDP) avulsion fracture generally occurs when grasping an opponent's jersey as it is quickly pulled away (Fig. 20.30). The FDP tendon is avulsed off the volar aspect of the distal phalanx due to forced hyperextension during active flexion. Injuries can involve tendon or tendon and a bony fragment [25, 32].

Fig. 20.25 Radial dislocation of 2nd PIP joint in a 42-year-old female mountain biker (**a**, **c**, **e**). Postreduction images conform the volar plate avulsion fracture (**b**, **d**, **f**)

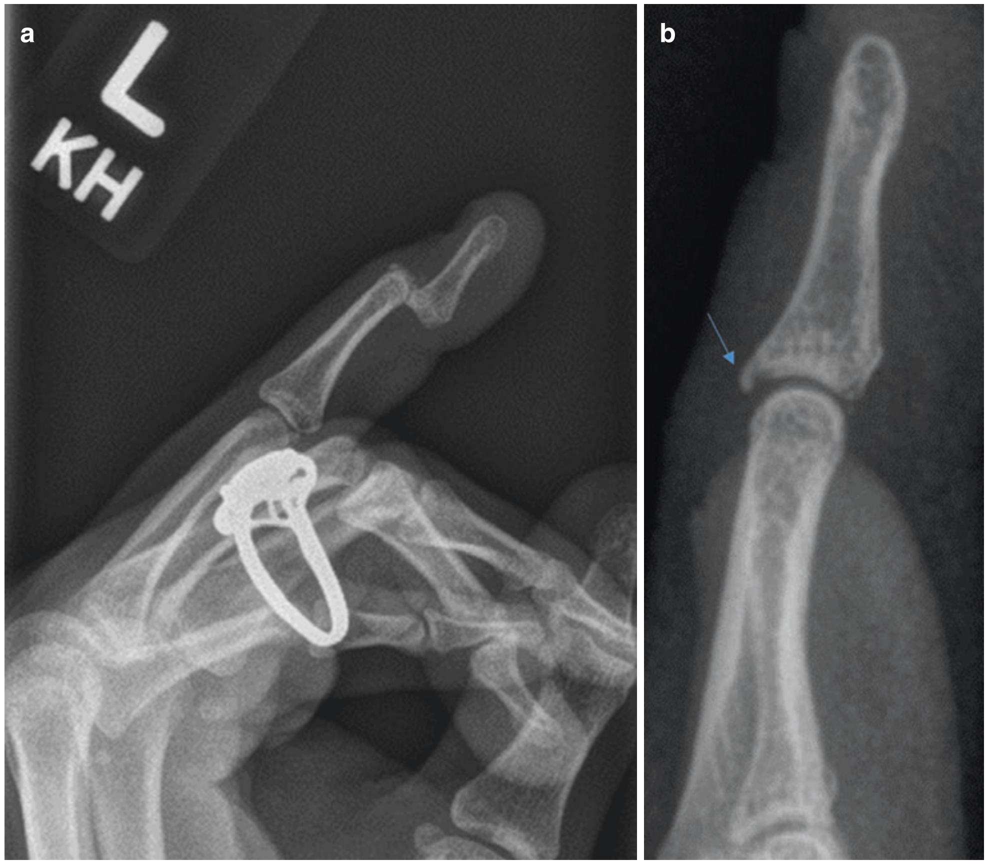

Fig. 20.26 Anterior dislocation of the 4th DIP joint in a 20-year-old mountain biker (**a**). Postreduction image (**b**) revealed a small avulsion fracture (arrow)

Classification

There are three types of FDP injuries. Type 1 occurs when the tendon retracts into the palm and the long and short vincula are both ruptured. These types of injuries can lead to compromised tendon nutrition and are the most severe injuries [25, 29, 32]. Type 2 injuries, which are the most common, are defined by tendon contraction to the DIP joint, preserving the vinculum and most of the blood supply, and can be successfully treated up to months post injury [29, 32]. In type 3 injuries, the tendon is avulsed off the bone and has a bony fragment preventing tendon retraction past the middle phalanx [29, 32].

Epidemiology

Injury to the FDP tendon is relatively common although incidence of specific FDP avulsion injury pattern is not well documented. Zone 1 FDP injuries (avulsion of the FDP at its insertion into the distal phalanx) occur about 6% of all tendinous injuries to the hand and wrist and 22% of all finger tendon injuries [29].

Clinical Presentation

Athletes present with a painful swollen digit notably for the inability to make a fist with loss of flexion at the DIP joint of the involved digit. If the tendon has avulsed, sometimes a bony fragment is notable on exam [20, 25, 32, 33].

Diagnosis

Three-view plain radiography is useful for evaluation of other osseous injuries and can identify type 3 injuries. MRI or ultrasound is necessary to evaluate the level of tendon retraction and should be obtained to confirm clinical diagnosis [20, 25, 32, 33].

Initial Management

Due to tendon retraction, operative management is necessary for all types of FDP avulsion injuries [20, 25, 32, 33]. In the meantime, the finger should be placed in a finger splint.

Indications for Orthopedic Referral

All suspected cases should be immediately referred. If a type 1 injury has occurred, repair is urgent, whereas type 2 and 3 injuries can have delayed repair [20, 25, 32, 33].

Follow-Up Care

Follow-up postoperatively to evaluate for normal routine healing is recommended and physical therapy is indicated to return the finger to normal function [20, 32, 33].

Return to Sports

Athletes can be expected to return to play in 8–12 weeks following surgical repair and should have normal range of

Fig. 20.27 Dislocation of the left first IP joint as a result of a fall while cycling (**a**, **c**, **e**). Postreduction picture (**b**) and X-rays (**d**, **f**) reveal anatomic alignment and small avulsion fracture of the volar plate (arrows)

motion with minimal pain with 80% of contralateral grip strength prior to resuming their sport [20, 32, 33].

Complications

Complications are noted especially with delayed tendon repair and can result in adhesion, contracture, chronic pain, and loss of grip strength in the affected digit [20, 32, 33].

First MCP Ulnar Collateral Ligament Injury (Skier's/Gamekeeper's Thumb)

Mechanism of Injury in Sports

This generally occurs with hyperabduction of the extended thumb when it receives extra valgus stress. A fall while skiing and holding the pole is the typical mechanism in skiers,

Fig. 20.28 Posterior dislocation of the 5th PIP as a result of a soccer injury in a 12-year-old boy 2 weeks earlier (**a**, **c**, **e**). Postreduction images confirm the anatomical alignment (**b**, **d**, **f**)

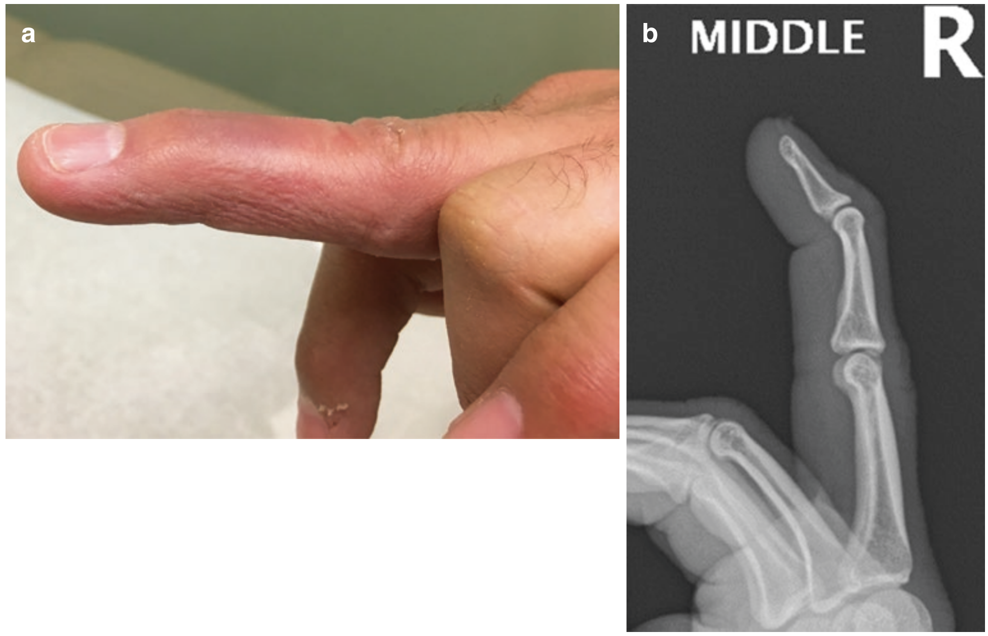

Fig. 20.29 Left 3rd mallet finger in a 40-year-old male (**a**). X-ray of a 38-year-old male with 3rd mallet finger (**b**)

but this can be seen in other sports where sticks are used (e.g., hockey and baseball) [34].

Epidemiology

This is a common injury with approximately 86% of all injuries to the base of thumb, resulting in about 200,000 cases per year in the United States [34].

Clinical Presentation

Patients generally present with pain, swelling, and ecchymosis on the ulnar side of the injured thumb [34, 35]. In acute settings, if there is no clinical or radiographic concern for an avulsion fracture and Stener lesion, clinical and radiographic stress test can be performed (see Chap. 19) [34, 35]. The UCL of the injured thumb can be tested by first holding the MCP in extension and applying valgus stress to the phalanx. Next, the MCP should be tested with the thumb in 30° of flexion. It is important that the thumb of the investigator is placed on the radial side of the MCP joint to apply counterpressure to prevent possible rotational effects and further injury. Laxity should be compared to the contralateral digit as this can vary widely between individuals [34, 35].

Diagnosis

The diagnosis is usually made clinically in combination with plain radiography [34, 35].

In rare cases, if there is high concern for injury with equivocal examination and X-ray, MRI can help with the diagnosis [34].

Initial Management

Initial management is based on severity of UCL injury. For partial tears, conservative management is utilized where the MCP joint is immobilized with a thumb spica splint or cast [34, 35]. The IP joint should remaine free. Immobilization for up to 4 weeks with repeat clinical evaluation for healing is generally sufficient for partial tears. Generally, if the MCP joint is stable on initial evaluation, nonoperative treatment is sufficient [34, 35].

Indications for Orthopedic Referral

For complete tears where the MCP joint is unstable or the UCL has avulsed with a bony fragment (Stener lesion), orthopedic referral is recommended [34, 35].

Follow-Up Care

Clinical evaluation for improvement of laxity is recommended in 4 weeks. When laxity improves, physical therapy for strengthening is recommended [34, 35].

Return to Sports

This depends on the sports and the position played in the specific sports. In general, athletes can return to play with a functional or protective brace as long as the symptoms are resolved with the ability to perform the specific sports. Athletes can return to play without a brace when laxity is resolved and range of motion has normalized. This usually occurs between 8 and 12 weeks [34, 35].

Complications

Without appropriate immobilization and/or repair, chronic stiffness, impaired range of motion, joint instability, and pain have been noted. Occasionally neuropraxia from radial nerve has been observed but generally resolves with healing [34].

Fig. 20.30 Left middle finger injury as a result of playing flag football in a 36-year-old male (**a**). Plain radiography revealed comminuted fracture of the third distal phalanx with about 4 mm of distraction of the anterior and posterior fracture fragments and flexor digitorum profundus tendon avulsion fracture (**b**, **c**). This was repaired using pullout sutures and a silicon button (**d**, **e**) and pinning of distal phalangeal fracture **f**, **g**)

Pediatric Consideration

Generally, pediatric cases are treated in the same manner; however, if there is concern for physeal disruption, orthopedic consultation may be necessary. Children with Salter-Harris III, IV, or V fractures should be referred to orthopedic surgery [34].

Fingers Collateral Ligament Injuries

Collateral ligament sprains (jammed finger) are very common in sports. They are more common at the PIP joint than DIP joints [25]. Athletes complain only of localized pain. On physical examination, there is usually localized edema and tenderness at the affected collateral ligament and worsening pain with stress varus or valgus test (while the affected joint is flexed to 30° and the MCP is flexed to 90°). Complete rupture of the collateral ligaments are uncommon. Surgical referral rarely indicated only for cases with instable joints [25]. Management is usually achieved by buddy taping. Athletes can return to sports as long as they can tolerate the symptoms [25].

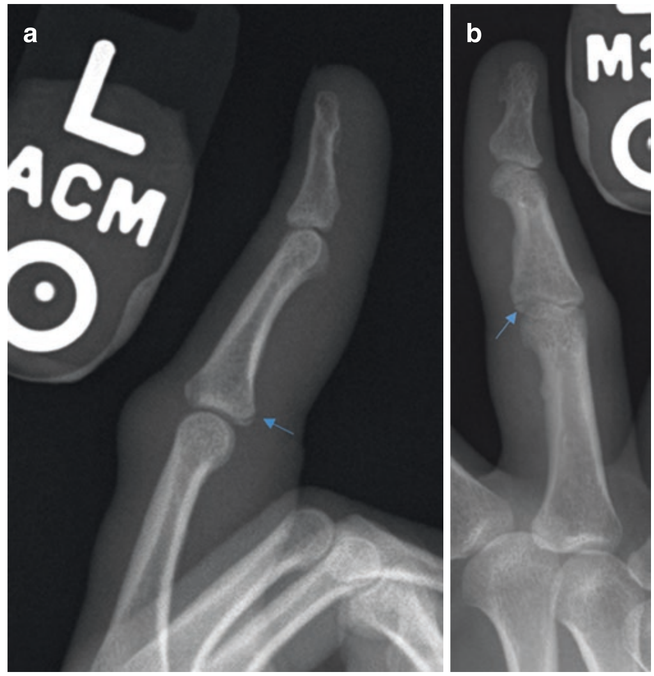

Fig. 20.31 Volar plate fracture (arrows) of the 3rd proximal phalanx in a 33-year-old female (**a**, **b**)

Volar Plate Injuries

Volar plate injuries occur when the thick ligament connecting two joints in the finger is injured. These types of injury result from either finger hyperextension (Fig. 20.31) or dislocated (Figs. 20.21 and 20.27). Most cases can be conservatively treated. This consists of a dorsal aluminum extension block splinting at a 15° flexion for 10 days, followed by a spontaneous mobilization and taping to adjacent fingers for sports only [25, 36, 37]. Plain radiography can be obtained if there is concern for avulsion or fracture [25, 36, 37]. Only unstable joints or large avulsion fragments need to be referred to hand surgery [25, 36, 37].

Central Slip Extensor Tendon Injury (CSETI)

CSETI is a relatively uncommon injury in sports that can happen when the PIP joint is forcibly flexed while actively extended [38]. This is usually accompanied by volar subluxation of the lateral bands [38, 39]. The injury is more common in basketball and can occur with volar PIP dislocation [25, 38, 39]. Patients are usually unable to actively extend the PIP joint [25, 38, 39]. Athletes typically have tenderness at the dorsal aspect of the affected PIP joint. Classic boutonniere deformity (flexion of the PIP joint and hyperextension of the DIP and MCP joints) usually develops over time if not treated appropriately. Plain radiographs may reveal dorsal avulsion fracture (central slip of extensor tendon) from the base of the middle phalanx [25, 38, 39]. The PIP joint should be placed in full extension using a finger splint for at least 6 weeks [25, 38, 39]. If there is any avulsion fracture or intra-articular fracture component of more than 25%, then a referral to a hand surgeon is recommended [25, 38, 39]. Athletes may return to sports with the finger splint as tolerated [25].

Soft Tissue Injuries

Fingertip injuries are relatively common particularly among men performing manual labors [40]. Its actual prevalence in sports is unknown, but it seems more common in high-speed and motorized sports. Most injuries are finger lacerations and crushes. Partial and complete amputations are rare [40, 41]. Appropriate management of these injuries is critical to minimalize the risk of the potential complications such as nail deformities, insensate or painful fingertips, loss of pinch strength, and neuromas [40].

Laceration

Finger lacerations are common injuries with either crush, blunt, or penetrating mechanisms (Fig. 20.18). Given the superficial location of tendons and ligaments, care must be taken when evaluating and repairing these injuries. Athletes with finger lacerations should be evaluated for injury to tendons or ligaments. Plain radiography can be useful if there is concern for foreign body retention. A tourniquet can be used for bleeding control (Fig. 20.32).

Irrigation of the wound with either saline or tap water should be performed to explore the wound. Local anesthetic should be used (regional or digital block) of the affected digit. Tetanus immunization status should be checked. A Tdap or DTaP booster should be given, if patient has not received a dose within 10 years for minor and clean wounds and 5 years for major and non-cleaned wounds [42]. Tetanus immune globulin (TIG) is only recommended in combination with tetanus toxoid (Tdap or DTaP) for people with unknown immunization status or < 3 tetanus toxoid immunizations and a non-cleaned and major wound [42]. Any concern for high-grade tendon or ligament injury, nerve damage, or amputation of the tip of the digit where the bone is exposed should be referred to a hand surgeon for evaluation. Antibiotics should be started for any injury with exposed bone [43].

Fig. 20.32 Using a finger tourniquet to reduce bleeding during finger procedures

Subungual Hematoma

Subungual hematomas occur with a crush or blunt impact to distal finger [4, 44]. Its actual prevalence is unknown, but it seems to be relatively common in sports [4, 44].

Patients generally present with acute injury to distal finger. There is common evidence of trapped blood beneath the fingernail with some discoloration. Symptoms are usually throbbing severe pain to the affected digit [4, 44]. Diagnosis is made clinically. Plain radiography can be used, if there is suspected distal phalanx or tuft fracture [4, 44].

Trephination (bore hole to relieve pressure) is recommended for all hematomas without disruption of the nail matrix [4, 44]. If there is nail matrix disruption, removal of the nail plate with repair of the nail matrix with 6-0 absorbable suture is recommended for satisfactory healing [4, 44]. Referral to a hand surgeon should be considered with significant nail matrix injury to evaluate healing and normal nail growth [4, 44]. Nail matrix laceration or disruption can result in permanent nail deformity and should be discussed with patient prior to treatment as this can still occur after acute care [4, 44].

Fingertip Amputations

Fingertip amputations are rare in sports, but when they do happen, appropriate management is critical to preserve the function and decrease the risk of complications [40, 41]. Most amputations require immediate referral to a hand surgeon. Management varies based on the extent of the injury to the bone, nail, nail bed, and soft tissue. Management of a partial and distal amputation may include only a primary closure, simple reattachment (Fig. 20.33), or in complicated cases may include complex reconstruction and replantation [40, 41].

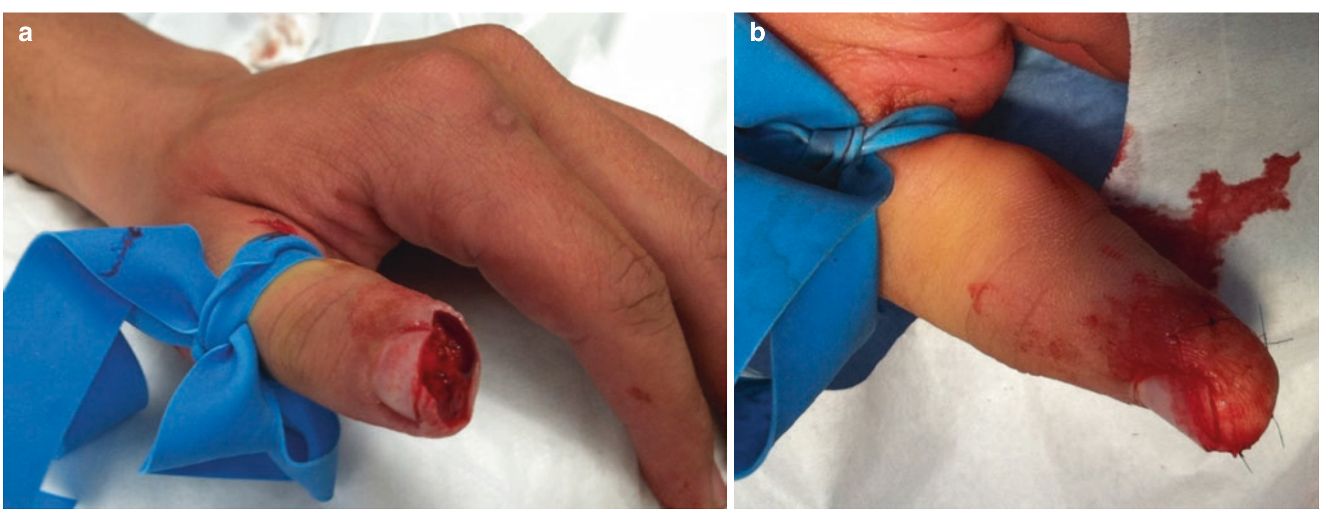

Fig. 20.33 Partial amputation of the distal left thumb in a 19-year-old male (**a**). The amputated part was reattached using superficial loose 6/0 sutures (**b**)

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Part IV

Acute Sports-Related Bones and Joints Trauma: Lower Extremity

Matthew Gammons

Pelvis

21

Yuka Kobayashi, Justin E. Hellwinkel,
and Morteza Khodaee

Key Points

- The most important aspect of treating pelvic injuries is the prevention of prehospital death mainly due to hemorrhage.
- Traumatic pelvic injuries will require a multidisciplinary approach to reduce mortality and morbidity.
- Following assessment of a high-energy trauma using ATLS protocol, proper pelvic binder placement can limit hemorrhage potential into the pelvic space.
- Pelvic avulsion fractures are painful and may limit activity but rarely require surgical intervention.

Introduction

Although pelvic ring fractures only account for 2–8% of all fractures, they are one of the most serious injuries in sports as the mortality rates are as high as 30% when multiple injuries are involved [1, 2]. Athletes who experience high-energy blunt force trauma to the pelvis may present with abdominal, low back, or diffuse pain, and it is imperative to recognize these signs because pelvic ring fractures are independent risk factors for mortality regardless of other injuries involved [3, 4]. In these situations, stabilization with a pelvic binder or sheet is integral to reducing hemorrhage [5]. Return to sport may be difficult following multi-injury falls or collisions, but simple fractures such as iliac crest and ischial tuberosity avulsion fractures have favorable outcomes [6].

Relevant Anatomy

The pelvis is a complex osseous ligamentous structure. The osseous structures that comprise the pelvis are the sacrum, coccyx, and two innominate bones (Fig. 21.1). The three segments of the innominate bones are the ilium, ischium, and pubis. All three parts converge to form the acetabulum, which articulates with the femoral head to form the hip joint. The two joints within the pelvic ring are the two sacroiliac (SI) joints at the junction of the sacrum and ileum posteriorly and the pubic symphysis at the anterior portion of the ring where the two pubic bones converge. The SI joint is the most stable joint in the body to distribute the weight of the upper skeleton through the pelvic ring to the lower extremities. This joint is augmented with a significant amount of ligamentous support by the iliolumbar, lumbosacral, anterior and posterior sacroiliac, sacrotuberous, and sacrospinous ligaments. The posterior sacroiliac ligaments are the strongest and most important for maintaining stability of the ring, as well as control internal rotation and vertical displacement. The pubic symphysis is comprised of a fibrocartilaginous disc between the two pubic bones and is significantly weaker than the other components of the pelvic ring. The distance between the two pubic bones is <2.5 cm in a healthy individual [7].

A significant amount of soft tissue lays on top of or traverses through the pelvic ring. Vascular structures include a venous plexus posteriorly resting on the internal surface of the iliac wings, the superior gluteal artery posterior to the iliac wings, and branches of the internal iliac vessels. Additionally, branches of the lumbar plexus, the sciatic nerve, and ventral rami, specifically the L5 nerve root, all lay near the SI joint and can be affected in cases of ring disruption. Organs of the genitourinary, gastrointestinal, and reproductive systems all lay in close proximity to the ring.

Y. Kobayashi
Department of Orthopedic Surgery, Medical College of Wisconsin, Milwaukee, WI, USA

J. E. Hellwinkel
Department of Orthopedic Surgery, New York Presbyterian/Columbia University Medical Center, New York, NY, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

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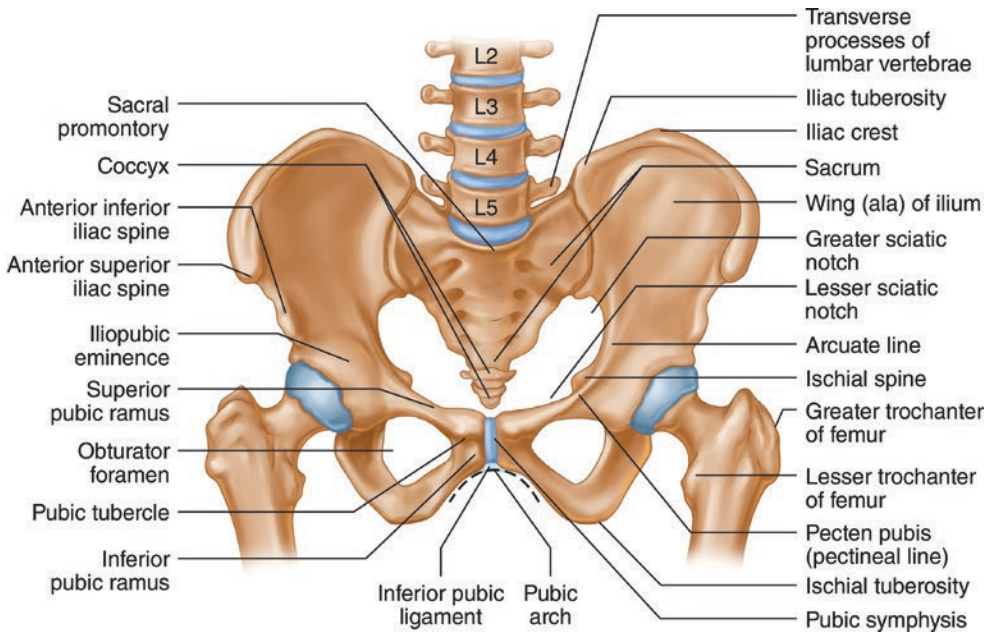

Fig. 21.1 Pelvis osseous anatomy

Pelvic Fractures

Pelvic Ring

Mechanism of Injury in Sport

Pelvic ring injuries result from a large kinetic force to the pelvic ring structures at great speeds or heights. Most pelvic ring injuries are due to motor vehicle accidents (60%), falls from a height (30%), and crush injuries (10%) [8]. As such, pelvic ring injuries are seen in high speed and impact sports such as two- and four-wheel motorsports, mountain biking, snowboarding, halfpipe, ski jump, and horseback riding. Low-energy pelvic ring fractures are becoming increasingly recognized as the geriatric population stays active longer [9]. Osteoporosis and osteopenia are predisposing factors in pelvic ring fractures from low-impact sports and falls of short distances.

Epidemiology

Pelvic ring fractures and injuries account for 2–8% of all fractures; however, in polytrauma patients, the occurrence jumps to 20–25% of cases [2]. When multiple injuries are involved, unstable pelvic fractures may lead to increased bleeding and the mortality rate can be up to 30% [1]. As many as 62% of high-energy pelvic fracture patients are admitted to the hospital in shock secondary to pelvic hemorrhage [3]. Moreover, pelvic ring fractures have been identified as an independent risk factor for mortality regardless of other injuries involved in blunt force trauma [4]. In children, pelvic fractures are the second leading cause of morbidity in trauma [10]. Three-fourths of all pelvic fractures in pediatrics are due to pedestrian versus motorized vehicle and sporting injuries only account for 3–5% [11].

Classification

The complex anatomy of the pelvic ring results in complex injury patterns in bony and ligamentous structures. As with any classification system, typical fracture patterns are categorized, but each injury can produce its own unique fracture fingerprint. The Young and Burgess classification system is commonly used to describe pelvic ring injuries because it helps describe the vector of force at the time of injury [12]. The three types of force vectors described are anterior to posterior compression (APC), lateral compression (LC), and vertical shear (VS) (Fig. 21.2). Within the APC and LC fracture patterns are three stages of severity, which are important to consider when determining pelvic stability. LC-I injuries are characterized as those with pubic ramus fractures and ipsilateral anterior SI buckle fracture. LC-II has the addition of an ipsilateral iliac wing fracture, and LC-III injuries classically have additional contralateral SI ligamentous disruption, which is described as a windswept pelvis. APC-I injuries are categorized as those with <2.5 cm of widening of the pubic symphysis, APC-II injuries have >2.5 cm of pubic symphysis diastasis and disruption of the anterior sacroiliac ligaments, and APC-III injuries contain all the elements of APC-II with addition of posterior sacroiliac ligament damage. LC-I and APC-I are typically the only two stable pelvic ring fractures. VS injuries are classified based on superior displacement of any part of the hemipelvis with concurrent fracture or rupture of ligamentous attachments.

Due to the complex injury dynamics and fracture locations, multiple classification schemes are needed to describe pelvic ring fractures. The Torode and Zieg classification has been preferred in the pediatric setting [13]. This slightly simplified system classifies type-I as avulsion fracture, type-II as iliac wing fracture, type-III as a simple and stable pelvic ring fracture, and type-IV as unstable ring disruption fractures (Fig. 21.3).

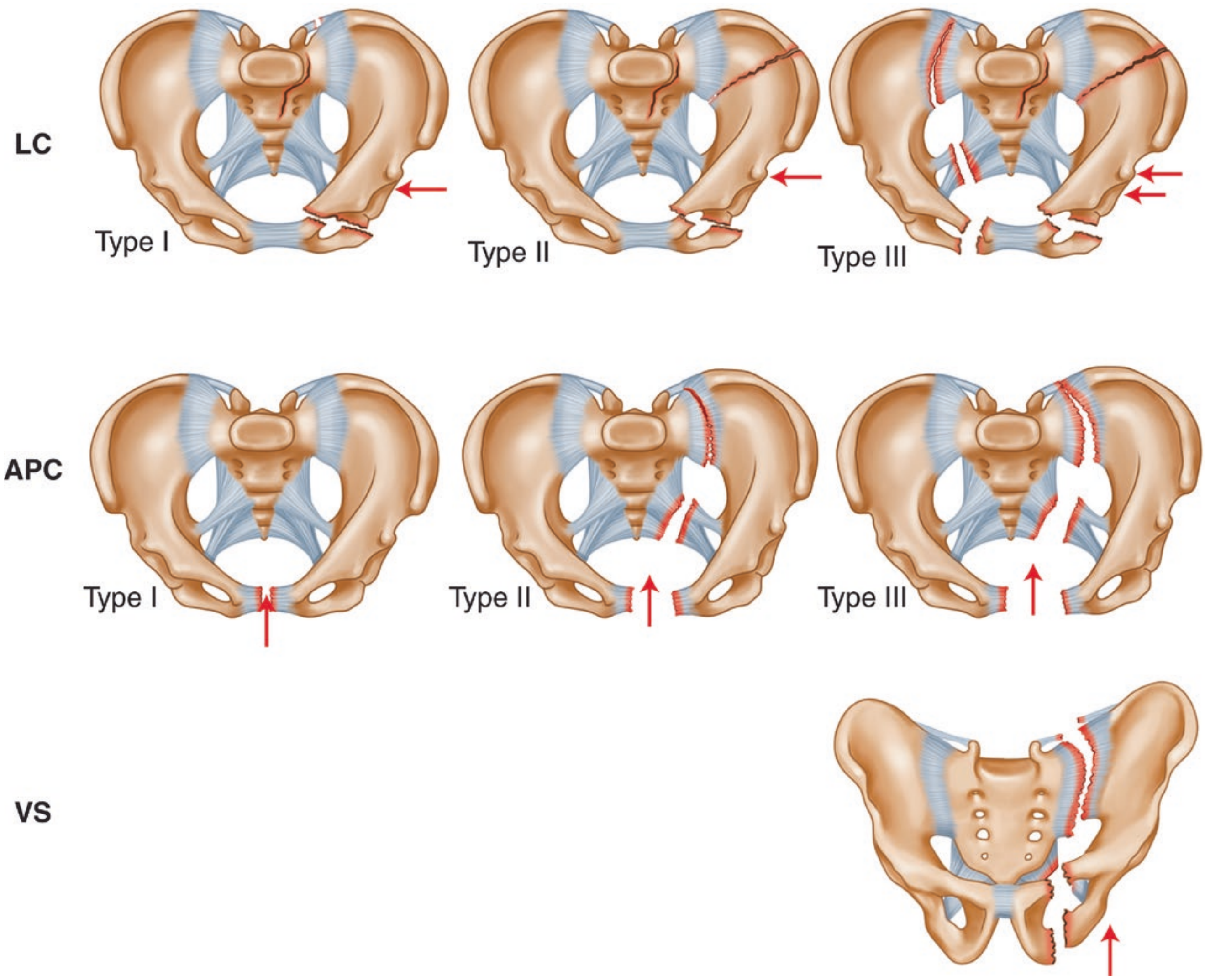

Fig. 21.2 Young and Burgess pelvic fracture classification system [12]

Clinical Presentation

Patients will present with significant pain localized to the hip or groin region, but they may also have abdominal, low back, or diffuse pain [3]. Depending on the severity of injury, weight bearing can range from uncomfortable to intolerable. Lower extremity neurologic symptoms may also be present if nerve roots passing near the injured part of the ring are affected. There are no physical examination findings sensitive or specific enough to rule out a pelvic ring fracture or dislocation. Less commonly, a patient may present unconscious secondary to head injury or severe hemodynamic instability. In an obtunded patient, a pelvic fracture should always be considered.

Diagnosis

After a general assessment, the physical examination should first begin with assessment of lower leg length discrepancy and rotational deformity. This should be followed by removal of sports gear and skin examination for ecchymosis or frank blood near the urethral meatus, vagina, and perineum, which can indicate presence of an open fracture and rectal, vaginal, or scrotal trauma. Light palpation of the iliac wings, anterior superior iliac spine (ASIS), pubic symphysis, sacrum, and coccyx will elicit pain at the site of fracture and is the most sensitive test for pelvic injury. Rotational stress testing is a gross method for diagnosing a pelvic ring injury, though this test has poor sensitivity [14]. External stress on the pelvis is performed by administering an outward and posteriorly directed force on the ASIS, and internal stress is tested by a direct inward force on the iliac wings. This test should only be performed once, due to risk of fracture displacement or dislodging a clot that is controlling internal hemostasis [15]. Plain radiography (Figs. 21.4, 21.5, 21.6, and 21.7) should be performed as soon as possible to evaluate the extent of the injury. Often times, a scout (portable) view is recommended. Due to the possibility of internal organ injuries, a FAST exam should be performed followed by a CT scan.

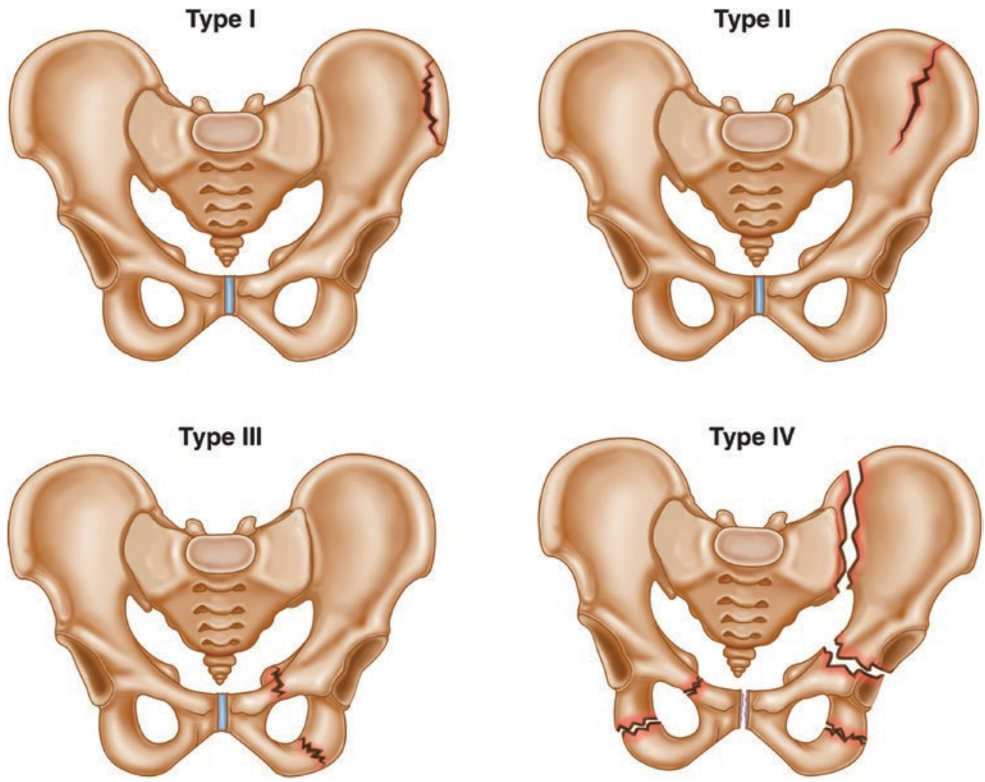

Fig. 21.3 Torode and Zieg pelvic fracture classification [13]

Fig. 21.4 LC-I fracture with mild diastasis of the right SI joint (arrows), right type-II sacral fracture (arrowheads), and right inferior pubic ramus fracture (open arrows) in a 16-year-old female as a result of a fall while mountain biking

Fig. 21.5 APC-III fracture in a 34-year-old male after falling from 6 m high while motorcycle racing. He was hypotensive in the field, fluids received en route to the hospital. Symphysis pubis widening and severe diastases of the right sacroiliac joint with zone III fracture of the right sacral ala (arrow) are present

Concurrent musculoskeletal injury occurs in many cases of pelvic ring injuries, especially long bone, acetabular, and vertebral fractures. A thorough assessment for other bony injuries should be performed and stabilized as appropriate. Given the nature of these high-energy injuries, painful distracting injuries may contribute to missed pelvic fractures. Soft tissue injury is common as well and may be more difficult to assess on initial exam. APC-type patterns tend to be associated with injury to vasculature, bowel, or genitourinary tract, whereas LC patterns tend to have concurrent brain, lung, or abdominal injury.

Initial Management

Pelvic ring injuries are usually caused by high-energy trauma and patients should be first stabilized per standard ATLS protocols. Attention should then be turned to the pelvic injury, as it is usually the most significant cause of patient decompensation. The only patients who do not require pelvic stabilization are those who are awake and oriented with no signs or symptoms of a pelvic fracture [16]. It is debated whether stable pelvic injuries should be treated with provisional stabilization on the field; however, the extent of injury is often

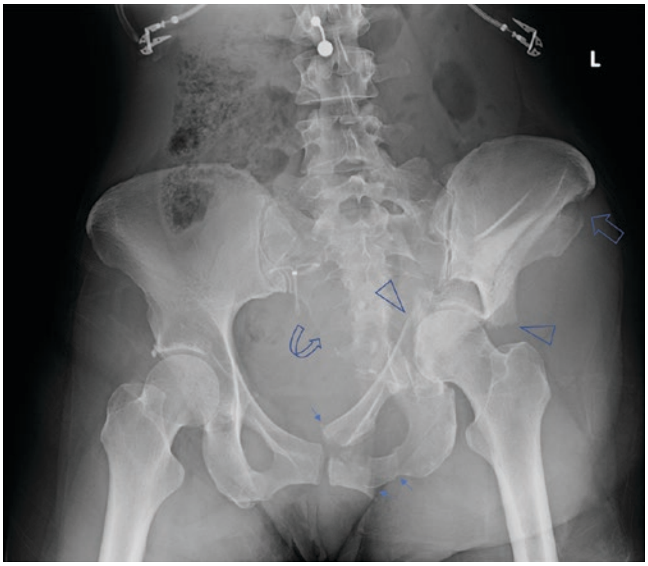

Fig. 21.6 Pelvic ring fracture in a 40-year-old female restrained driver in four-wheel motor vehicle accident against tree. LC-III fracture with inferior and superior pubic ramus (arrows), iliac crest (open arrow), acetabular (arrowheads), and zone II sacral (curved arrow) fractures

not completely clear. When in doubt, it is best to err on the side of pelvic stabilization because of the significant morbidity associated with missing an unstable pelvic fracture.

Given the risk of significant blood loss with shear injury to the pelvic venous plexus, the primary management to stabilize the patient is to reduce pelvic volume as quickly as possible. Normally the pelvis can accommodate 4 liters of blood and just 5 cm diastasis of the pubic symphysis will increase that volume an additional 20% [17]. APC-III injuries with complete SI joint disruption (Fig. 21.5) can increase this volume to up to 15 liters and require, on average, 12.6 units of transfused blood [18]. Regardless of fracture pattern, the best method to stabilize the pelvis and reduce pelvic volume is application of a pelvic binder or bedsheet circumferentially, both of which are equally effective [5]. The binder or sheet should be placed at the level of the greater trochanters directly over the skin and tensioned with enough force to restore anatomy, which can be gauged by monitoring landmarks on the patient's legs. Overtightening can cause additional injury in fracture patterns that are unstable with internal rotation.

Improper placement of the binder superior to the greater trochanter occurs almost 40% of the time and fails to reduce the diastasis of the pubic symphysis [19]. Binder placement at the level of the greater trochanters is critical, especially in hemodynamically unstable patients. If a pelvic binder or sheet is not available, and the patient is not suspected to have a lower extremity fracture, the patient's legs and ankles can be taped together in internal rotation, which will help reduce the pelvic volume to near anatomic in up to 20% of cases [20]. If using this technique, tape should be placed at multiple levels down the legs to avoid pressure necrosis of the skin.

After a compression device is placed, the patient should continue to be monitored for signs of hemodynamic instability or shock. Fluid resuscitation should begin concurrently with primary assessment in 250 cc bolus increments to restore peripheral pulse. Rapid large volume resuscitation raises concern for increasing hemorrhage into the pelvis if a vascular injury is present, however may be appropriate in cases of concurrent brain injury or shock [21]. The patient's pain should be appropriately managed and other injuries should be addressed at this time. Transfer to a stretcher should be performed with an 8-person scooping method rather than a log roll method, which carries a risk of fracture displacement and increased hemorrhage (Chap. 4; Fig. 4.1).

It is critical that the patient be evaluated in a setting where appropriate interventions can be performed. The patient will require a FAST exam to look for hemoperitoneum and chest and pelvic radiographs immediately upon arrival in the ED. A hemodynamically unstable patient with a positive FAST exam will need to go directly to the operating room (OR).

Indications for Orthopedic Referral

The approach toward a pelvic ring injury is a multidisciplinary one. Teams from trauma surgery, plastic surgery, neurosurgery, urologic surgery, and others should be made aware of the patient on route to the hospital. Orthopedic surgery consultation is necessary for all pelvic ring fractures, and the pelvic stabilizer should remain in place until orthopedic assessment. In severe cases, these fractures can be orthopedic emergencies and require immediate surgical stabilization to save a patient's life.

Follow-Up Care

Follow-up care will be determined greatly by the extent of the initial injury and the organs involved. The multidisciplinary team should continue to follow the recovery of the patient and communicate with each other for optimal recovery. Rehabilitation is dependent on the extent of the initial injury. If there was no extensive soft tissue or dermatologic injury, aquatic therapy may be the optimal first step. It will improve range of motion, coordinated motions, and endurance [22]. Unloaded treadmills are a modality to transition from the post-injury or postoperative phase to return to sport.

Return to Sports

Return to sport will be based on the stability of the injury and patient level of comfort. Patients with stable pelvic fractures should be treated with an initial period of protected weight bearing, then graduated return to sport with appropriate physical therapy [23]. After the patient sustains a high-energy complex pelvic fracture, return to sports may be difficult. Initially, the athlete may lack awareness of their physical tolerance following a hospitalization. In severe trauma cases, psychological intervention during recovery may be warranted.

Complications

As described above, pelvic ring injuries are associated with many severe complications due to the high-energy mechanism of trauma. The most immediate complications are exsanguination, urethral injury, bladder rupture, blunt trauma

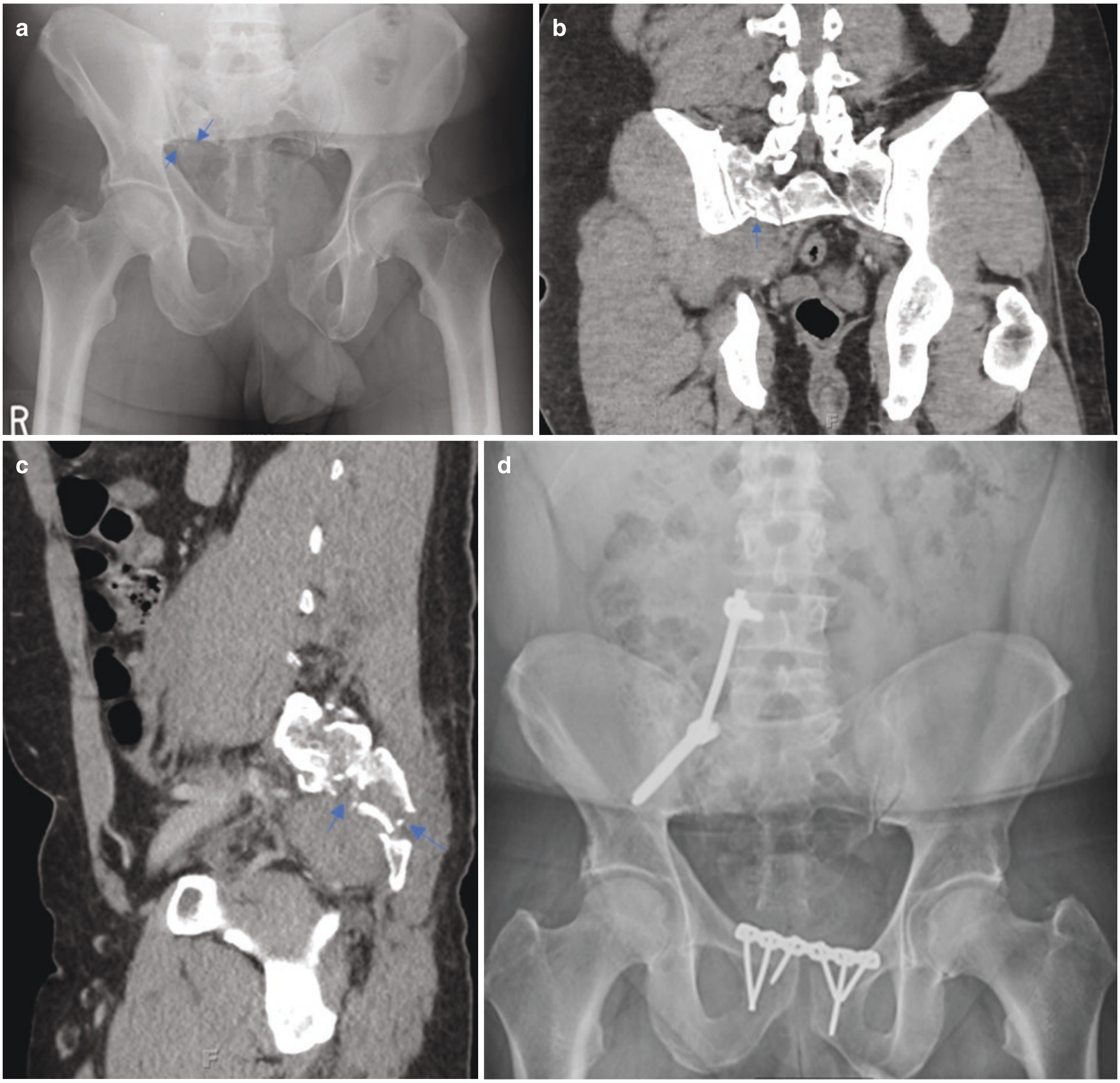

Fig. 21.7 Pelvic ring fracture in a 43 year-old male as a result of a fall. Significant symphysis pubis widening and zone II sacral (arrows) fractures (**a**) are present. CT images (**b**, **c**) demonstrate the sacrum fracture better. Postoperative plain radiography reveals near anatomical alignment (**d**)

to abdomen, intraperitoneal laceration from fracture fragments, and lung, brain, and additional musculoskeletal injuries. Long-term complications result secondary to any of these injuries and chronic instability of the pelvis.

Pediatric Considerations

Although the incidence of pelvic fractures is twice as high in adults compared to children, the severity of these fractures (abbreviated injury score ≥ 4) is similar [24]. Children have greater cartilaginous volume and bone plasticity to allow for improved absorption of stress prior to fracture [25]. Given the anatomical differences and the need for potentially higher energy to cause a pelvic fracture in children, any pelvic injury should be a red flag [26]. Neurologic complications occur in half of pediatric athletes with pelvic fractures and concussions are the most common [13].

On the field, assessment of polytrauma and hemodynamic status is performed similar to that of an adult. Notably, pediatric patients tend to compensate much better with blood loss and vital signs should be monitored closely, as a rapid change is gravely concerning. The mortality for this population is closer to 5% and massive blood loss is more often secondary to intra-abdominal injury rather than pelvic vasculature disruption [10]. Regardless, they should be placed in a pelvic compression device for stabilization and transported to a medical facility. With increasing involvement of high-energy sports in this population, pelvic fractures are increasingly gaining focus in the pediatric world.

Sacrum

Mechanism of Injury in Sport

Injuries to the sacrum have been described in vertical shear injuries such as falls from heights, or compression injuries in MVA and crush injuries. Vertical forces lead to more superior displacement of the ala. Lateral compression injuries lead to anterior forces on the ala and distraction forces posteriorly. As described above, sacral fractures also result from high-energy trauma for young athletes and low-energy falls in older athletes. Case reports of sacral fatigue stress fractures have also been reported in long-distance runners and soccer players but are very rare [27, 28].

Epidemiology

The prevalence of non-osteoporotic sacral fractures has increased from 0.67 per 100,000 persons to 2.09 between 2002 and 2011 [29]. A retrospective study also showed the rate of surgical treatment for sacral fractures increased [30]. Female long-distance runners are the most prone to sustaining sacral stress fractures [31]. Sacral stress fractures are also associated with the female athlete triad.

Classification

Zone I, alar zone, is a fracture through the ala without involvement of the foramina or central sacral canal (Figs. 21.8, 21.9, and 21.10). In lateral compression injuries,

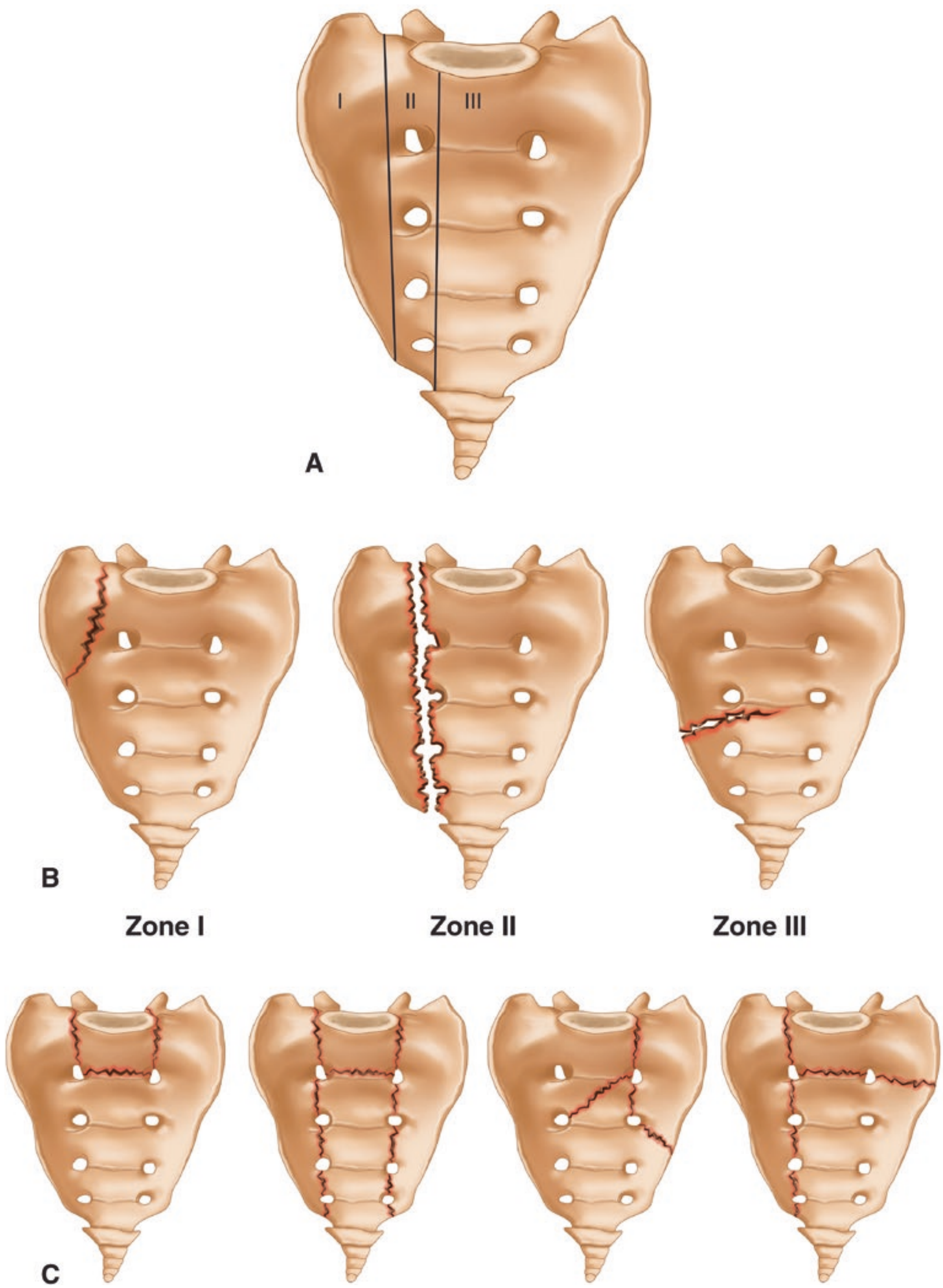

Fig. 21.8 Denis classification of sacral fractures (**a**, **b**). Zone III fractures (**c**) can have multiple patterns [32]

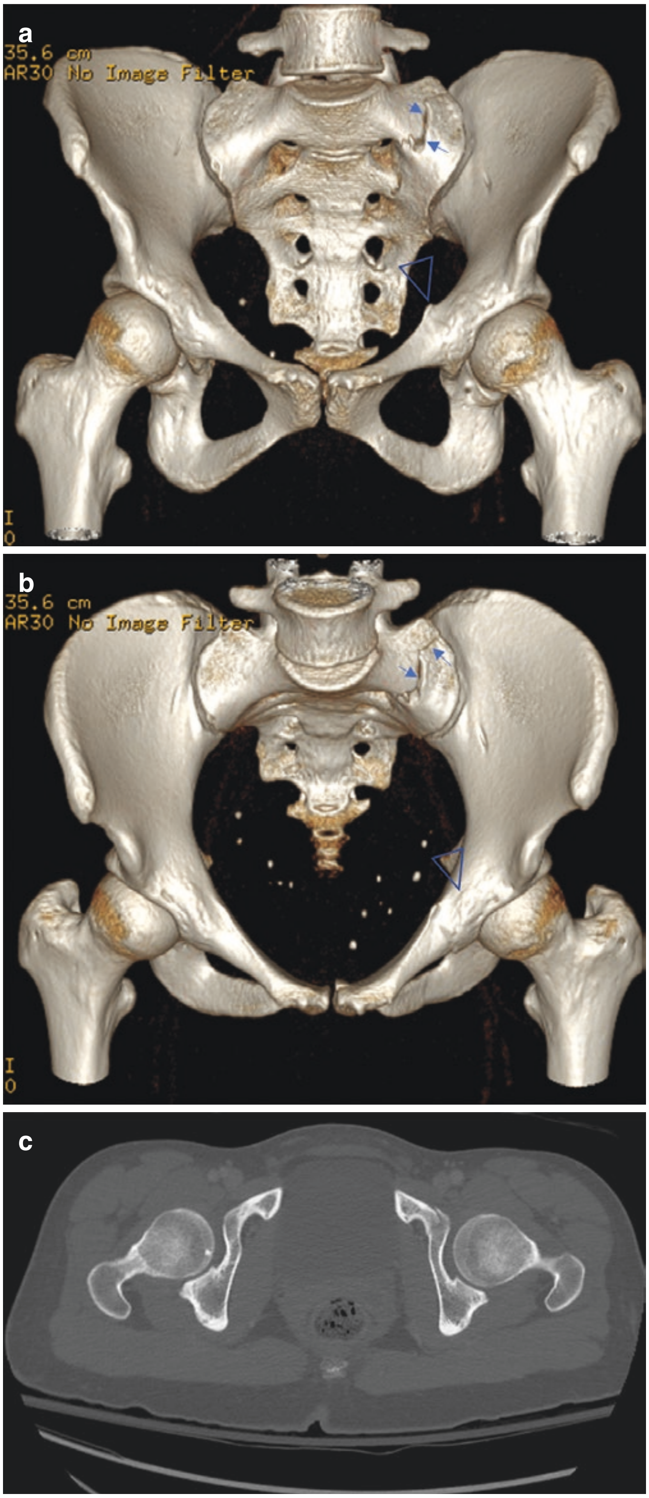

Fig. 21.9 Denis zone I left sacral ala (arrows) and superior pubic ramus (arrowheads) fractures in a 22-year-old female as a result of a ski injury visible on 3D CT images (**a**, **b**). CT (**c**) also reveals the extent of the left superior ramus fracture into the anterior and superior acetabulum (open arrow)

there is less displacement when compared to vertical shear injuries. Severe pelvic instability may result from sacrotuberous ligament avulsions involved in this type of fracture. Also, open-book deformities can be involved in even minor alar fractures. Zone II, foraminal zone, is when ≥1 foramina are involved without impingement on the central sacral canal (Figs. 21.4, 21.8, and 21.11). Neurologic concerns in this zone warrant more timely involvement of surgical referral. Zone III, central zone, involves the central sacral canal (Figs. 21.8 and 21.12). Neurologic damage occurs in over half of these patients [32]. U-type sacral fractures (Fig. 21.8) result from axial loading and can lead to spinopelvic dissociation, resulting in much higher incidence of neurologic complications [33]. It has been suggested more recently that the Denis classification overestimates the prevalence of nerve injuries in zone I and II injuries. This group suggested that neurological deficits were seen primarily with displaced, comminuted, or zone III fractures or with U-type spinopelvic dissociation [34]. Lastly, transverse fractures often involve three zones and have higher nerve dysfunction risk (Fig. 21.8).

In sacral stress fractures, high-risk stress fractures can be classified as a visible fracture line or bone marrow edema on MRI, whereas a low-grade fracture has no fracture line and bone marrow edema is only evident in STIR and/or T2-weighted sequences on MRI [35].

Clinical Presentation

The most common symptom of sacral fracture is peripelvic pain. Athletes may present with soft tissue trauma around the pelvis and this should always raise suspicion for pelvic or sacral fractures. Zone I fracture without significant superior migration does not typically present with neurological deficits and can be stable fractures. Zone II fractures present with neurologic symptoms attributed in up to 28% of cases [30]. Symptoms also include bowel and urinary dysfunction, foot drop, and mixed radicular involvement. The main concern of neurologic involvement is with zone III and U-type fractures, which may include nerve root avulsions with higher likelihood for chronic deficits. In the case of sacral stress fractures, the athlete may present with chronic lumbar spine pain.

Diagnosis

Ecchymosis, deformity. and swelling during inspection following a blunt trauma should raise concerns of a sacral fracture. Careful palpation of the iliac wings and subcutaneous fluctuance will help determine pelvic instability or lumbosacral fascial degloving, also known as a Morel-Lavallee lesion [36]. Neurological exam includes assessment for light touch,

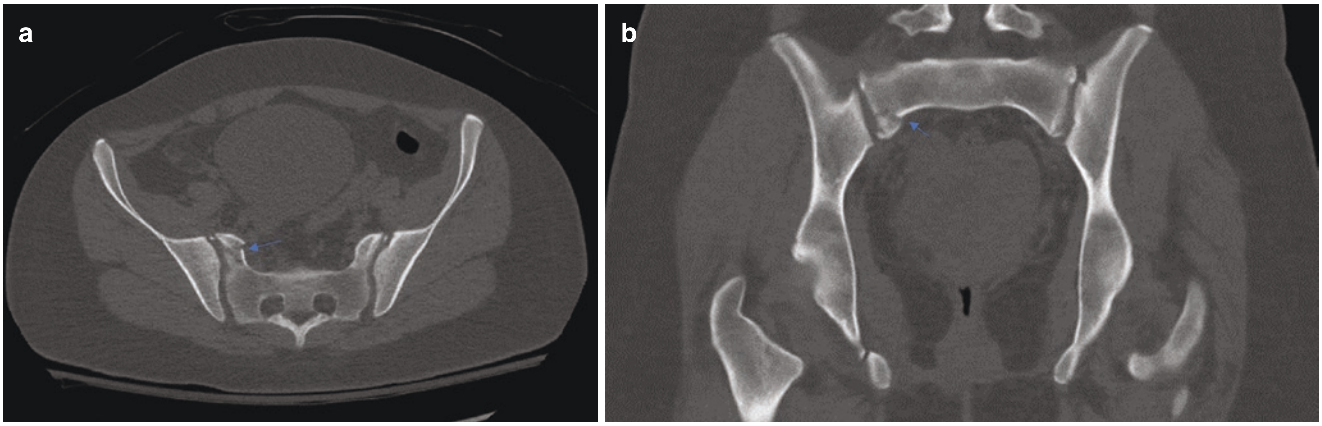

Fig. 21.10 Right Denis zone I sacral alar fracture (arrows) in a 16-year-old female as a result of a fall visible only on CT (**a**, **b**)

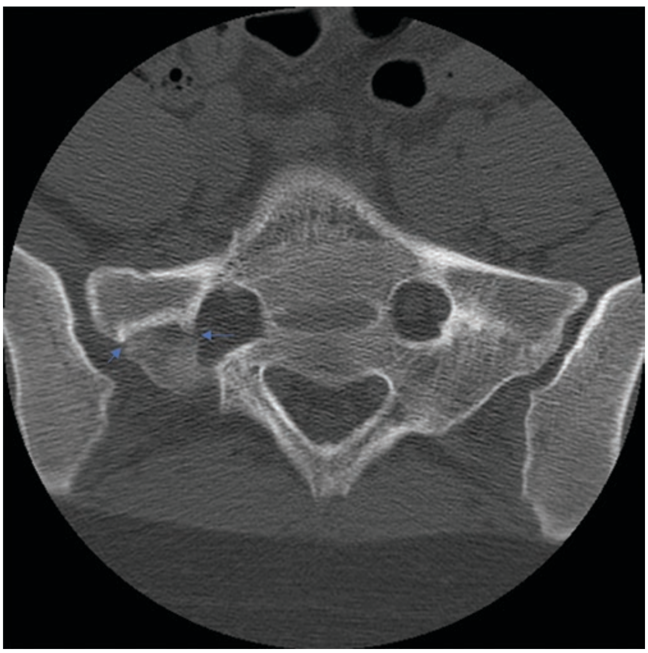

Fig. 21.11 Denis zone II sacral fracture in a 16-year-old female only visible on CT scan (arrows)

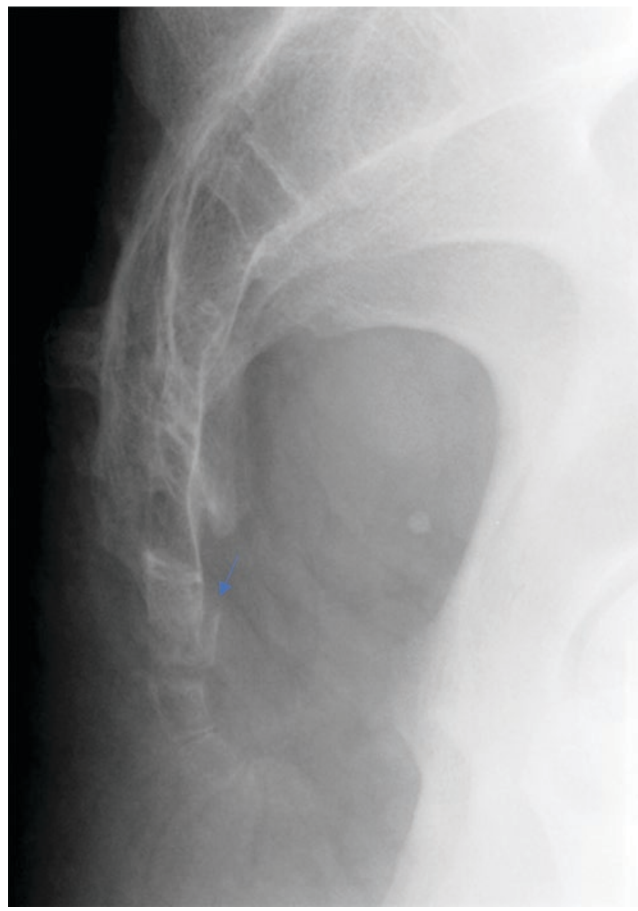

Fig. 21.12 A mildly displaced Denis zone III sacral (S5) fracture (arrow) in a 44-year-old male cyclist as a result of a backward fall

pinprick sensation, perianal wink, and bulbocavernosus and cremasteric reflexes. As with any trauma examination, the vascular integrity needs to be assessed. In the emergency department, plain radiographs are first line for any pelvic concerns. However, up to 80% of sacral fractures are missed on X-ray; thus, a CT scan is almost always warranted [37] (Figs. 21.7, 21.9, 21.10, and 21.11). Further assessment of neurologic involvement may require an MRI. As much as 75% of sacral fractures are missed in neurologically intact patients and that number is still 50% in those with neurological deficit [33].

Initial Management

It is extremely difficult to distinguish an isolated sacral fracture from a complex pelvic ring fracture given the great force it takes to cause both these injuries. Please see the section above for details on immobilization, which will be the appropriate initial step for high-energy blunt trauma cases. If there are indications of L5 root involvement with a superior displacement of the alar fragment, early traction may be warranted under fluoroscopy [38]. Transcondylar traction as a closed reduction technique in vertically unstable pelvic ring disruption is often used.

Indications for Orthopedic Referral

Unstable sacral fractures associated with pelvic instability always warrants an orthopedic referral. Please see "pelvic ring" fracture section for more details. Orthopedic surgery should be consulted for stable zone I fractures with superior displacement and/or neurologic involvement. Treatment of zone II fractures with sciatic involvement and severe foraminal obstruction is surgically addressed with early foraminotomy to prevent prolonged or permanent symptoms. If the pain interferes with life-saving cares, such as respiratory care, an external fixator may be indicated. All vertical shear fractures involving the lumbar plexus require decompression with early traction [38]. Zone III fractures should be assessed for acute phase decompression as it is less difficult due to extreme tightness found when decompression is attempted later.

Follow-Up Care

Stable fractures are treated with early ambulation, non-weight bearing, or partial weight bearing with crutches as symptoms permit and if there is no vertical shear displacement of the alar component. Progressive weight bearing with or without orthosis is indicated in cases with displacement less than 1 cm and no neurologic deficit [39]. Entrapment of the L5 root must be evaluated on follow-up as the alar fragment may migrate caudally. If the patient has sciatica, initial bed rest is the treatment of choice. If sciatica persists, surgical intervention such as sacral laminectomy may be warranted.

When the displaced fracture is greater than 1 cm or there is concern for a Morel-Lavallee lesion, surgical fixation with debridement is indicated [39]. Also, if the patient fails non-operative treatment with worsening neurological symptoms, surgical fixation is needed. Lastly, any patient with acute neurologic injury should be surgically decompressed.

Return to Sports

Athletes will be toe-touch weight bearing for up to 8 weeks [40]. This is followed by 6 weeks with no running, then a 6–8-week period of a return to running progression. Most published works indicate athletes may have a full return to activity by 4–6 months with rare cases taking up to 14 months [33]. However, these are estimations and each plan should be individualized. Given the high-risk region of sacral stress fractures, a high-grade lesion takes an average of 4–5 months with a median of 3 months for return to play. The low-grade lesions take a median of 2.3 months for return to play [41].

Complications

Complications are reported in 26% of patients and that number has remained steady over time [30]. Injuries involving the sacrum lead to more neurological deficits when compared to pelvic injuries without the involvement of the sacrum [42]. Complications may include bladder and bowel dysfunction, sexual dysfunction, sciatica, and foot drop with involvement of the L5 root. The nerve roots are larger in relation to its exit foramen at the higher sacral levels, making intraforaminal damage more likely at S1 and S2 compared to S3 and S4 [43]. There may be chronic radiculopathies from foraminal entrapment of the S1 root. Malreduction is also of concern, especially in cases with vertical displacement. The rate of neural involvement correlates with the degree of fracture instability [44]. Venous thromboembolism is always a postoperative concern, but especially with pelvic fractures because of prolonged immobility.

Pediatric Considerations

Pediatric sacral fractures make up 0.2% of pediatric blunt traumas [45]. As with adults, advanced imaging is often needed to diagnose sacral fractures in children (Fig. 22.10 and Chap. 22; Fig. 21.12). Neurologic involvement or sequela is rare in these fractures. Due to the extreme forces needed to cause fractures in the pediatric population, recovery is typically complicated by multi-organ involvement.

Pubic Rami

Mechanism of Injury in Sports

Pubic ramus fractures can be a component of a pelvic ring fracture from high-energy blunt force trauma (Figs. 21.4, 21.6, and 21.9). Lateral forces primarily concentrate at the sacrum and pubic rami and thus are the more commonly fractured segments of the pelvic ring. When in isolation, pubic ramus fractures are stable fractures (Figs. 21.13, 21.14, and 21.15). Soccer, ballet, and distance running are sports associated with low-energy stress fractures in the pubic rami [23]. Acute increase in training or repetitive motions will often accompany violent muscle contractions to cause stress fractures. The displacement of the fracture is a result of stress from the inguinal ligament and insertion of the pectineus muscle and adductors.

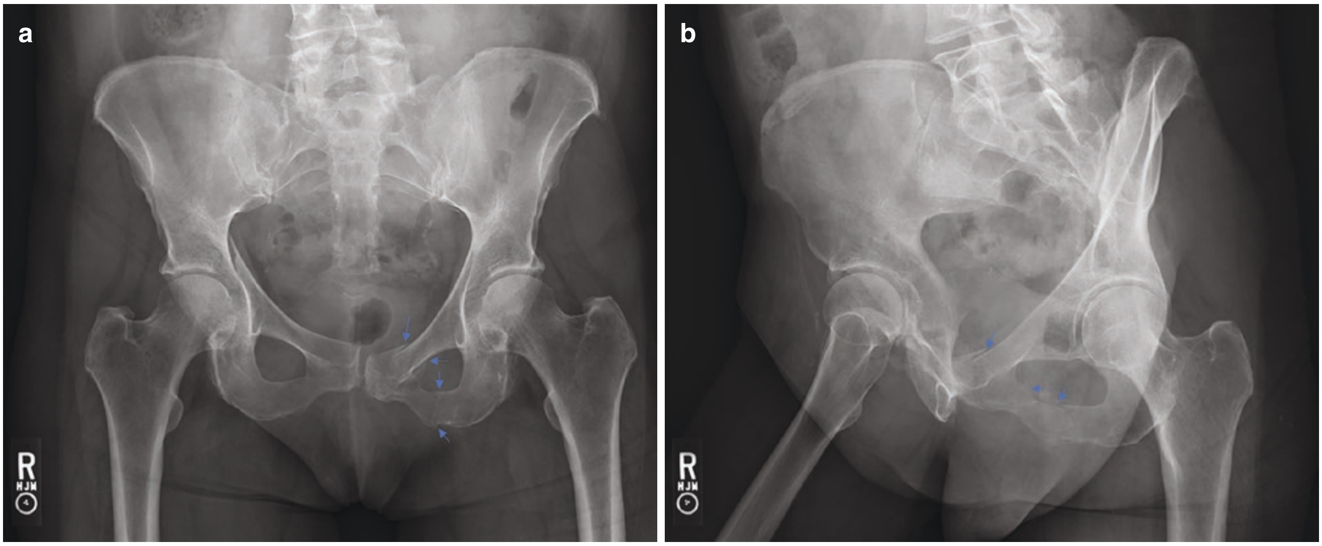

Fig. 21.13 A 50-year-old female presents after jumping off from 3 meters height. Plain radiography images (**a**, **b**) show mildly displaced fracture of the left superior and inferior pubic ramus fractures (arrows) seen in both views

Fig. 21.14 Comminuted left superior and inferior pubic ramus fractures (arrows) in a 75-year-old female as a result of a ski injury

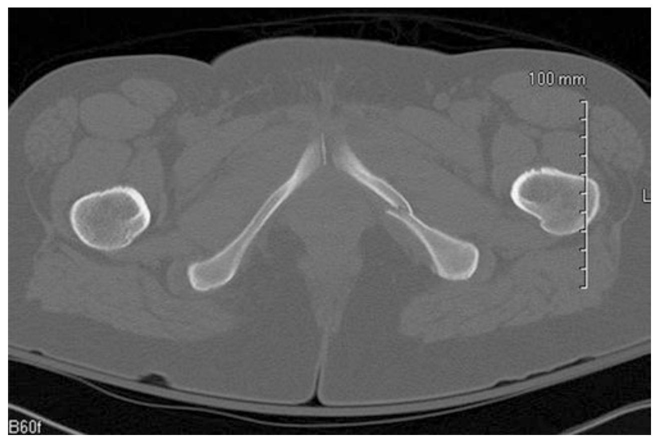

Fig. 21.15 A mildly displaced left inferior pubic ramus fracture (arrow) seen on CT. The fracture was not visible on plain radiography

Epidemiology

As with all high-energy pelvic ring fractures, the presence of one area of fracture is usually associated with a second site of fracture. Although high-energy pelvic fractures are more common in men, stress fractures of the pelvis are more common in women. Less than 2% of all stress fractures are in the pubic rami. Studies show that isolated traumatic pubic ramus fractures are stable with an average displacement of 3–4 mm [46]. The presence of a pubic ramus fracture acts as an indication of posterior pelvic injury, but these can be difficult to detect [47].

Classification

The pubic ramus fracture site is separated into a parasymphyseal fracture, midramus fracture, or pubic root fractures. Superior pubic rami act as the stabilizing agent in the pelvic ring; therefore in the presence of a high-energy trauma, complex fractures must be considered. For further details on classification, please see the "pelvic ring" fracture section.

Clinical Presentation

When the mechanism of action is high energy, the athlete will have sustained complex injuries, resulting in immobility. In low-energy stress fractures, there will be tenderness to

palpation, inguinal pain exacerbated by flexion and adduction, bruising, and stiffness. Inguinal swelling and mass are very rare in pubic ramus fractures. Patients may be able to weight bear following this type of injury, but an antalgic gait may be seen coming off the field.

Diagnosis

Pubic ramus fractures are one of the two most common fractures in a high-energy pelvic ring fracture. Please see "pelvic ring" fracture section for diagnosis during high-energy impact or fall-type injuries. Mildly displaced and non-displaced fractures may not be visible on plain radiography, so CT scan is a preferred diagnostic modality in cases with suspected fractures (Fig. 21.15).

Initial Management

Although these isolated fractures are stable, the athlete should be restricted from activity and evaluated with imaging. In the event of a high-energy blunt trauma, the initial management should not deviate from that of an assumed pelvic ring fracture, described above. If the patient is hemodynamically stable and there is no concern for complex injury, the patient can be carefully transported without immobilization. However, pain may be the limiting factor and athletes may need to be non-weight bearing until pain is controlled while awaiting further assessment.

Indications for Orthopedic Referral

Most cases of isolated pubic ramus fractures without >2 cm of displacement are treated conservatively and show excellent healing. Need for surgical intervention in a stable fracture is rare. Fixation is typically reserved for continued pain and malunion after trial of conservative management [48].

Follow-Up Care

More than 50% of pubic ramus stress fractures will be missed on X-rays [49]. A useful view may be the cephalic angulation view. If there is clinical suspicion for a stress fracture, the patient should be treated as such. If the athlete is in season, MRI may be used to rule out fracture. The concern for high-grade stress fractures is complete fracture and malunion. The athlete will be weight bearing to pain as with other stress fractures. When the female athlete triad is in question, a DEXA scan and labs are appropriate [50]. Repeat imaging on follow-up can be a helpful marker to determine appropriate healing versus need for further imaging when symptoms persist.

Return to Sports

Most stable pubic ramus fractures are treated conservatively. However, the athlete may need to be on weight-bearing restrictions as tolerated by pain. This period could take as long as 1–6 weeks before the pain improves. A return-to-sport protocol for gradual strengthening and increase in sport-specific activities can take another 6–8 weeks.

Complications

The superior gluteal and obturator arteries, as well as the venous plexuses, sit adjacent to the SI joint and pubic rami, which are the common sites of injury in pelvic fractures. Hemorrhage is a great concern in high-energy traumas. In low-energy traumas leading to stress fracture, the most common complication is persistent pain or weakness with or without nonunion.

Pediatric Considerations

Isolated pubic ramus fractures are rare in pediatrics (Fig. 21.16). Plain radiograph is not the modality of choice for these fractures in both adults and children (Fig. 21.15). To avoid repeated radiation exposure, MRI should be considered when the index of suspicion remains high following a negative X-ray. Management of these fracture is similar to adults.

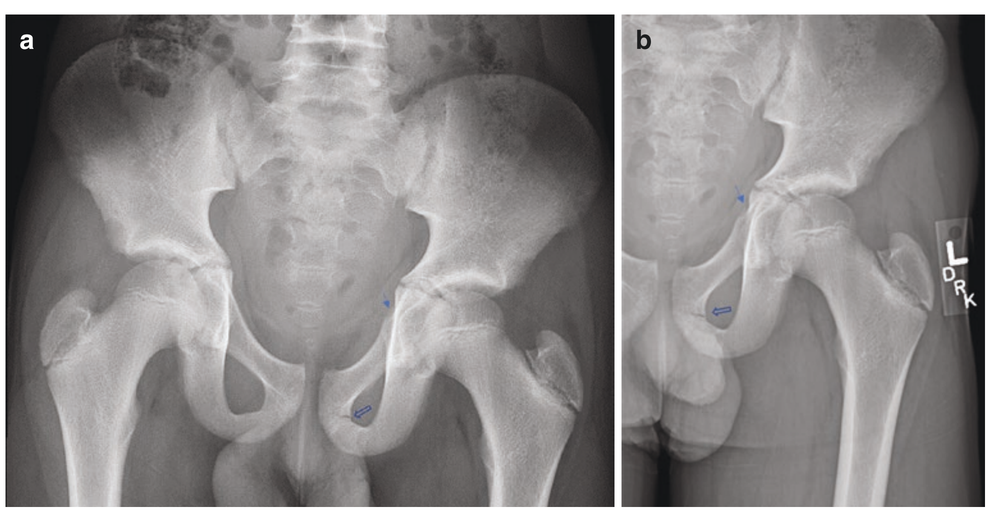

Fig. 21.16 Left inferior (open arrows) and superior (arrows) pubic ramus fracture in a 13-year-old male snowboarder as a result of a fall (**a**, **b**)

Iliac Crest

Mechanism of Injury

High-energy falls and collisions can lead to severe iliac crest fractures. High-speed skiing, snowboarding, and waterskiing can lead to iliac crest fractures without pelvic ring involvement (Fig. 21.17). Iliac crest avulsions occur during acute forceful contraction of the transversus abdominis muscle, external or internal oblique muscles, all which attach to the iliac apophysis (Figs. 21.18 and 21.19). Common sports that lead to such injuries include running, tennis, and soccer. Patients often feel a crack or a pop at the time of injury. The most common mechanisms of apophyseal separation are explosive running and kicking exercise [51]. Iliac crest fractures in sports have been described in low-velocity twisting injuries and high-velocity blunt trauma, which causes a pelvic ring-type injury containing an iliac wing component, as described above.

Epidemiology

Iliac crest apophysis avulsions are the least common of the four types of pelvic apophysis avulsions in athletes, accounting for 2% of all apophyseal injuries [52]. The iliac apophysis does not typically begin fusing until 13–15 years of age; thus, this injury is more often seen in adolescent males. Stress fractures of the iliac wing in sports injuries are exceedingly rare, with few cases described, mostly in runners and military recruits. Traumatic fractures of the iliac wing are almost always associated with pelvic ring injuries.

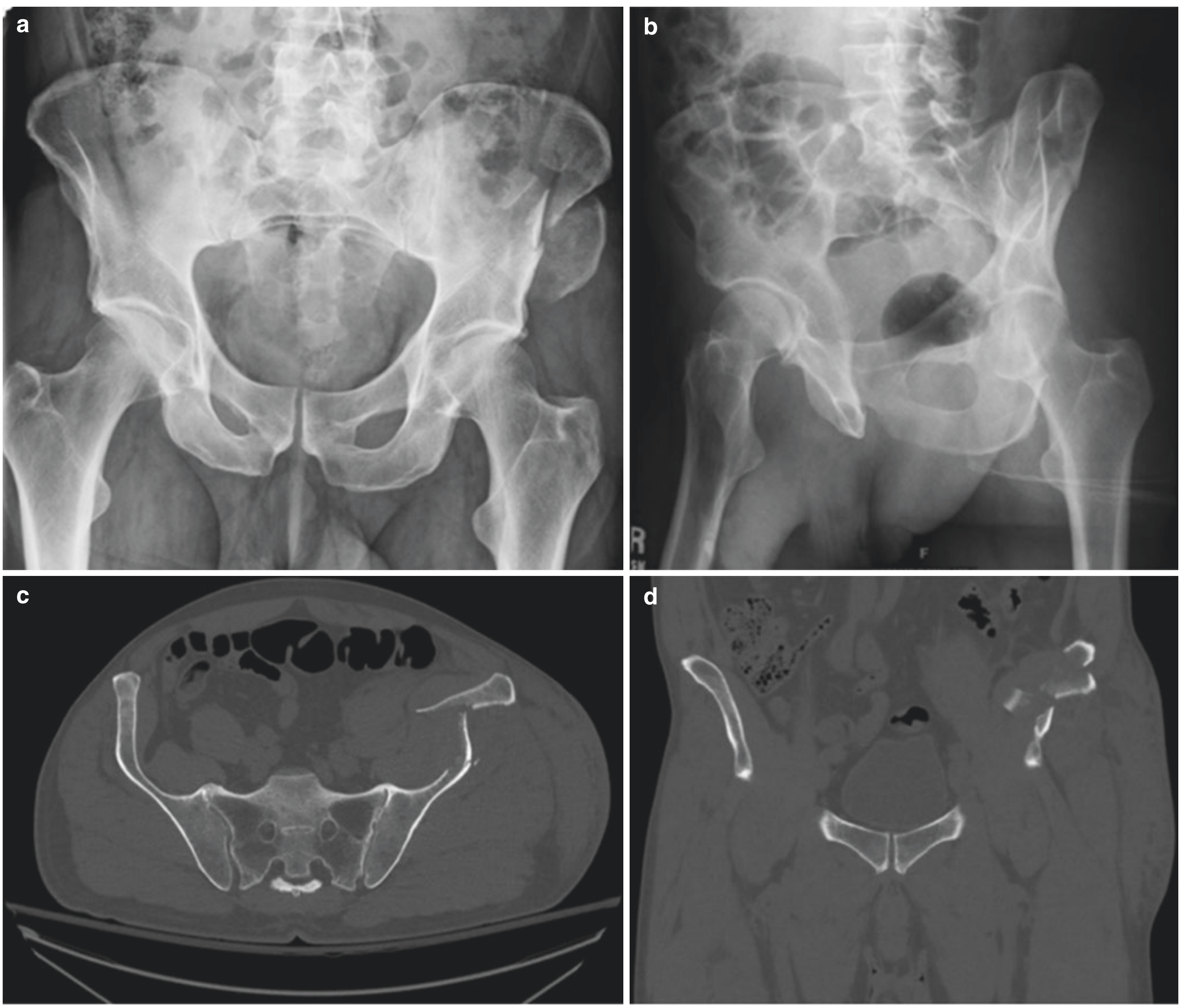

Fig. 21.17 Isolated left ilium fracture in a 57-year-old male as a results of a tree strike while skiing (**a**, **b**). CT scan images reveal the extent of the fracture (**c**, **d**)

Fig. 21.18 A 16-year-old male cross-country runner felt a pop in his left hip during a race. He was unable to continue the race due to left hip and low back pain. Plain radiography reveals avulsion of the left iliac crest apophysis (arrow)

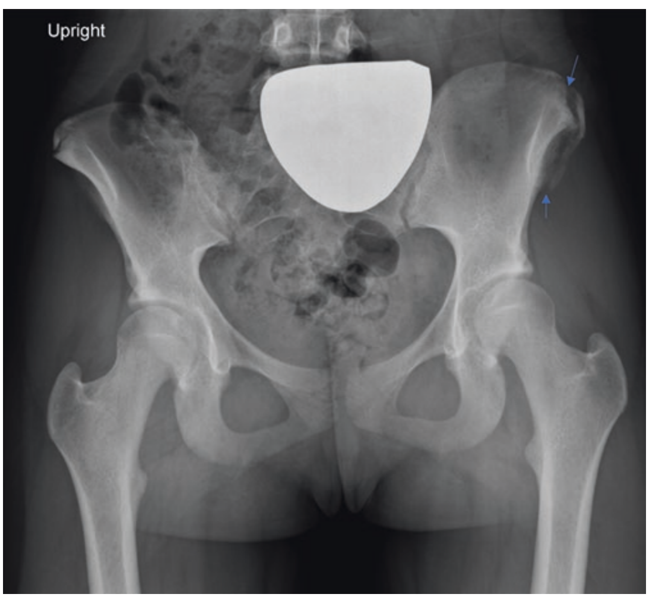

Fig. 21.19 A 16-year-old female track athlete runner felt left hip pain during first half of 3200 m race, then a pop towards the end of race. Plain radiography reveals avulsion fracture of the left iliac crest apophysis (arrows)

Classification

Avulsion fractures (Figs. 21.18 and 21.19) are classified based on the degree of displacement, with >3 cm considered an indication for surgical fixation for faster return to play [6]. Iliac crest avulsions are associated with extension to ASIS avulsion fractures as well. Stress fractures may be classified by systems used to describe pelvic insufficiency fractures in the elderly; however, these are a separate entity in younger patients and do not fracture in predictable patterns. Extension of fracture line into the SI joint is seen in half of stress fractures [53]. Low-energy traumatic fractures can extend into the acetabulum [54]. High-velocity traumatic fracture is much more likely to result in injury to other parts of the pelvic ring (Figs. 21.6, 21.20, 21.21, and 21.22) and can be classified by the Young and Burgess system (Fig. 21.2).

Clinical Presentation

Iliac apophysis avulsions present nonspecifically with difficulty with ambulation and pain with abdominal contraction or lateral flexion. The patient will point directly to their iliac crest as the source of pain. Stress fractures typically present as atraumatic, activity-related lateral hip pain with varying degrees of discomfort. Patients will have insidious onset of pain for weeks or months that does not resolve with activity modification.

Diagnosis

Iliac crest avulsion injuries can be identified through history of a forceful twisting mechanism and tenderness to palpation of the iliac crest. AP and oblique plain radiographic views help the clinician determine the degree of separation [6]. Plain radiography will demonstrate separation of the apophysis; however, contralateral X-rays may be needed to differentiate from a normal apophysis. CT scan is the best imaging modality to determine bony fracture; however, since this injury occurs in young patients with unfused physes, MRI is preferred when available to reduce radiation exposure. Pelvic inlet and outlet views can reveal subtle fractures on plain radiography (Fig. 21.20).

Diagnosis of a stress fracture of the iliac wing requires a thorough history of predisposition for stress fractures, including previous stress fractures, recent changes in exercise regimen, medical conditions that predispose to osteopenia, and screening for female athlete triad. On examination, patients may or may not have an abnormal gait and tenderness to palpation on the iliac wing depending on the site of fracture. Plain radiography can be unremarkable and MRI is the best imaging study to evaluate periosteal change and marrow edema [55]. CT imaging can be helpful to characterize fracture pattern or rule out other bony abnormalities (Figs. 21.17, 21.20, and 21.22).

Initial Management

Iliac crest avulsions can be treated initially with analgesics, restricted weight bearing, and rest. Stress fractures should also initially be treated with pain medications, restricted weight bearing, and rest from physical activity for a period of weeks [53]. There is no consensus; however, NSAIDs can potentially slow bone healing in some cases [56].

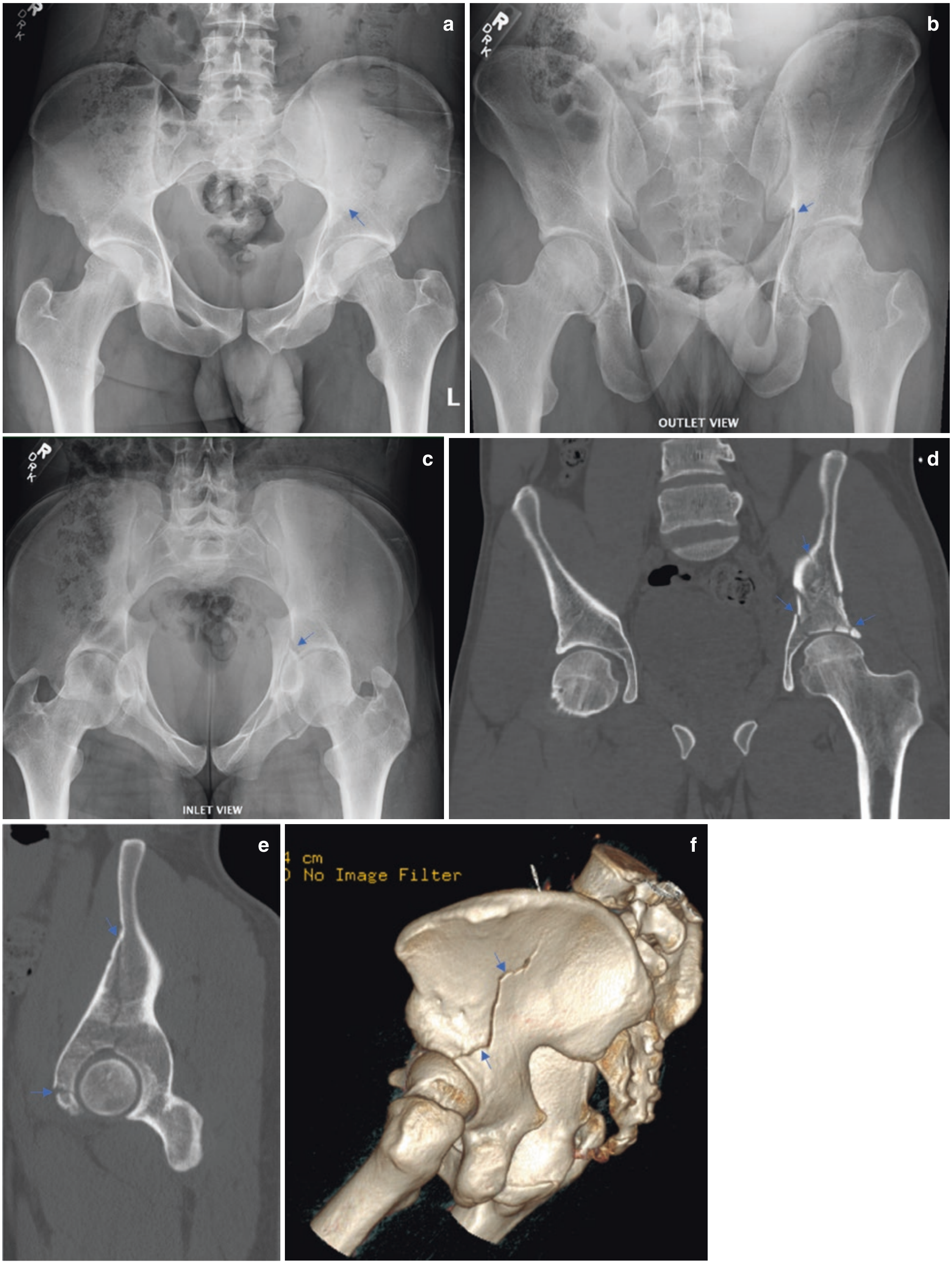

Fig. 21.20 Left superior acetabular fracture extending into the ilium (arrows) in a 24-year-old male snowboarder as a result of a collision with another snowboarder (**a**–**c**). Fracture line is more visible in outlet (**b**) and inlet (**c**) views. CT (**d**, **e**) and 3D CT (**f**) images demonstrate the fracture better

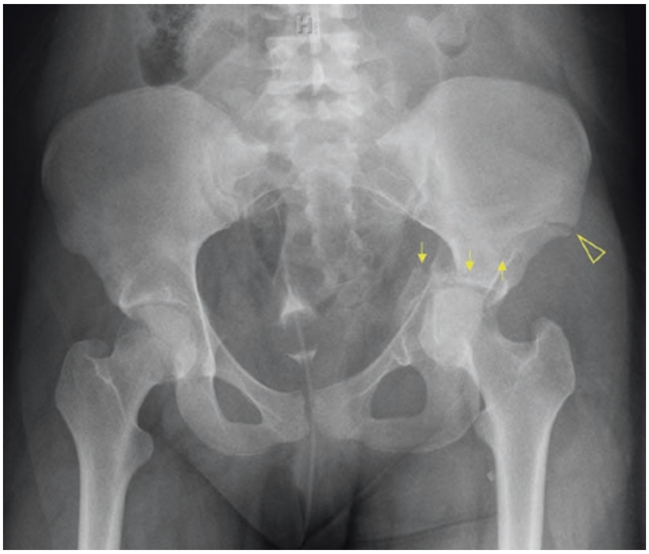

Fig. 21.21 Left ilium fracture in an 18-year-old female as a result of a mountain bike injury. The fracture lines extend from ilium ala (arrowhead) into the body of ilium and acetabulum (arrows)

Indications for Orthopedic Referral

Apophyseal avulsion fractures displaced >3 cm are surgical candidates and should be referred to an orthopedic surgeon. Other smaller avulsions and stress fractures do not require orthopedic consultation.

Follow-Up Care

Patients with iliac crest avulsion fractures should be restricted with weight bearing for approximately 1 week; then the patient can advance to weight bearing as tolerated. Resolution of symptoms typically takes 4–6 weeks. Stress fractures in young athlete should raise concern for underlying metabolic abnormalities that predispose them to insufficiency fractures. Patients should be monitored with serial X-rays for evidence of fracture healing and MRI can be repeated as needed if no improvement is seen.

Return to Sports

Iliac crest avulsion fractures typically heal well and patients can return to play within a few weeks of injury once pain has resolved [6]. Patients with stress fractures can advance weight bearing as tolerated after a period of rest and are able to return to sport when the fracture is fully healed on X-ray [53].

Complications

Avulsion fractures may have residual pain months after the injury, and stress fractures of the iliac wing in marathon runners have been associated with gluteal tendon tear, but few other complications occur with proper rest [57].

Pediatric Considerations

These injuries occur prior to physeal ossification in pediatric athletes (Figs. 21.18 and 21.19). The Tanner and Risser stage of patients should be noted at the time of injury to predict time to ossification of physes. The iliac crest secondary ossification centers appear between 13 and 15 years of age and fuse between 15 and 17 years. Isolated acute iliac crest fractures other than avulsion fractures are rare in the pediatric population.

Ischial Tuberosity

Mechanism of Injury in Sports

Hamstring injuries are common at all levels of athletics, but an avulsion of the ischial tuberosity is rare. They are seen more in the pediatric population as fusion of the apophysis occurs in late adolescence (Figs. 21.23 and 21.24). Often, apophyseal fractures are categorized as muscular injuries. These fractures occur in sports such as sprinting, hurdling, soccer, and waterskiing as they are a result of forceful hip flexion with knee extension. All hamstring muscles except the short head of the bicep femoris attach at the ischial tuberosity.

Epidemiology

In the adolescent population, ischial tuberosity is the second most common site of pelvic avulsions after the AIIS [58]. Athletes age 17 to 18 are at increased risk of these avulsion fractures [59]. The sports most associated with these avulsion sites are lower extremity ball sports.

Classification

There is no official classification system of the ischial tuberosity. Avulsion fractures are classified by displacement and an ischial tuberosity avulsion <2 cm can be treated conservatively [60]. Because the obturator foramen is a fixed ring structure, one fracture can lead to fracture of the ischiopubic ramus. It is important to note that fractures in the body of the ischium near the acetabulum have more potential to cause serious injury because an acetabular fracture (discussed in Chap. 22) can lead to inability to weight bear.

Clinical Presentation

Patients present with proximal hamstring pain that suddenly worsens during competition or practice. They may hear or feel a pop and examination may reveal ecchymosis and swelling of the posterior thigh and palpable mass or gap in the proximal posterior thigh. There is limitation in motion with stiff leg to avoid hip and knee flexion [61].

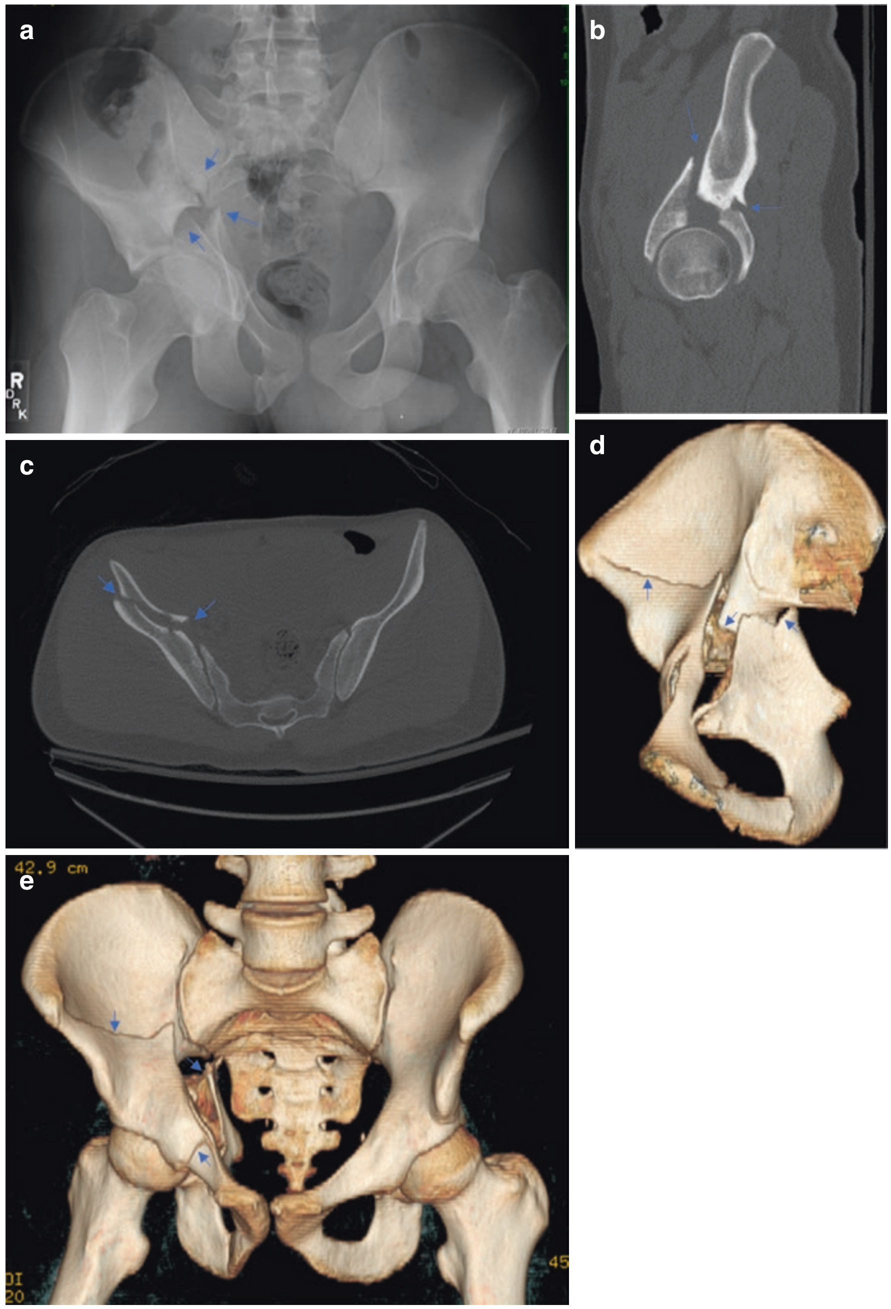

Fig. 21.22 Right acetabular and ilium fractures (arrows) in a 19-year-old male as a result of a mountain bike injury (**a**). CT (**b**, **c**) and 3D CT (**d**, **e**) reveal the extent of the fracture

Diagnosis

Passive stretch of the hamstring will elicit pain on examination. X-rays (Figs. 21.23 and 21.24) may show avulsions off the ischial tuberosity; however, if there is high suspicion for avulsion due to continued pain or activity limitations following a hamstring injury, MRI or ultrasound may be necessary to see the fracture.

Initial Management

Athletes should be evaluated with radiography to differentiate a muscular injury versus fracture. The area should be iced with pain control on the field and the patient taken out of play. The athlete should remain non-weight bearing until imaging is completed.

Indications for Orthopedic Referral

Most avulsion fractures can be managed conservatively, but the degree of disruption and the activity level of the athlete dictates need for surgical intervention. Proximal avulsion ruptures, partial avulsions that lead to failed nonoperative treatment with persistent symptoms for 6 months, and ≥ 15 mm of retraction in a young active patient require orthopedic referrals [58].

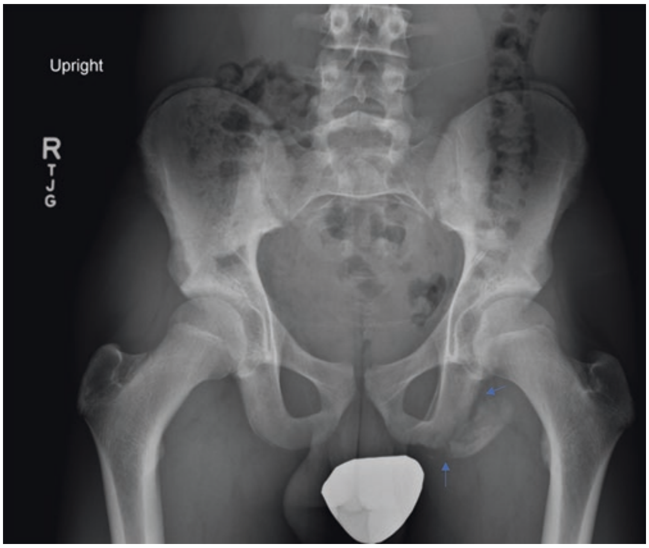

Fig. 21.23 A left ischial tuberosity avulsion fracture (arrows) in a 16-year-old male as a result of a noncontact injury during a soccer practice

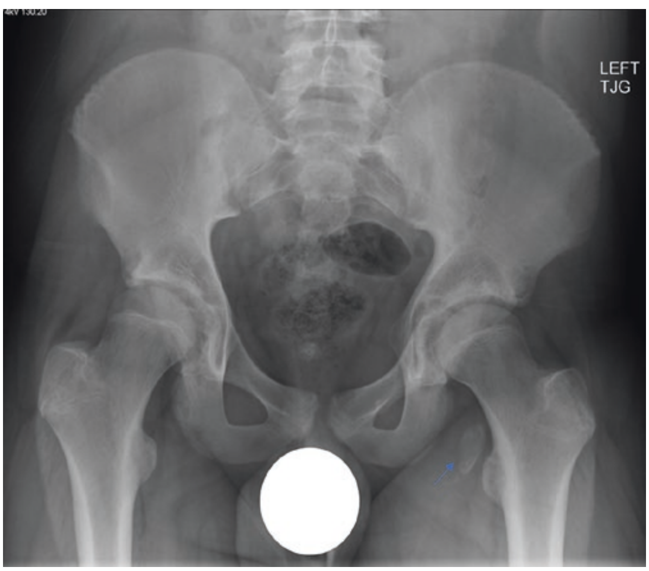

Fig. 21.24 A displaced left ischial tuberosity avulsion fracture (arrow) in a 14-year-old male as a result of a noncontact injury (sprinting toward the first base) during a baseball game

Repair of the ischial tuberosity with suture anchors or screws may be necessary in those cases as it is easier to mobilize acute ruptures and there is less potential sciatic nerve scarring.

Follow-Up Care

Nonoperative treatment includes pain control, limited activities, and partial weight bearing for 3–6 weeks [62]. There are some rare cases where the patient will need to remain non-weight bearing to ensure optimal healing [63]. Rehabilitation is key to return to sport. Pain control will need to be tailored to the athlete's specific condition, as some experience little pain and some have discomfort with activities such as defecation. In females, pelvic swelling can become an uncomfortable issue [64].

Return to Sports

Most athletes return to sport after suffering an ischial tuberosity fracture. With conservative management, partial weight bearing is typically started immediately with a range lasting 0–3 weeks. Full weight bearing begins on average 5 weeks out in nonsurgical cases. The average return to sport is 2–3 months postoperatively with a range of 2–6 months [58]. In a subgroup of athletes who underwent surgical intervention, their percentage of return to sport was higher (92%) than those who were conservatively managed (80%) [65].

Complications

There may be continued weakness in hip extension and knee flexion. If there is continued buttock and ischial tuberosity pain, the patient may be experiencing hamstring syndrome which requires surgical release and sciatic nerve decompression. Other neurological sequelae include sciatic nerve scarring and neuralgia. Nonunion is more common in conservatively treated patients and heterotrophic ossifications are seen in postoperative groups [58]. These patients may experience pain with sitting with tumoral growth appearance on follow-up imaging. Osteonecrosis and exostosis can also result as a complication from these fractures [66]. Ischiofemoral impingement is a rare complication where the soft tissue structures between the os ischium and lesser trochanter of the femur are constricted [67].

Pediatric Considerations

Fractures are most common between puberty and late adolescence when the apophysis is the weakest link. The ischial apophysis appears later in adolescence between 15 and 17 years of age and fuses between 17 and 19. As adolescents ramp up sport-specific training in this age group, the incidence of hamstring injuries and avulsion fractures increases.

Coccyx

Mechanism of Injury in Sports

This could be a part of a pelvic ring fracture as the coccyx is attached to the sacrum by a fibrocartilaginous joint. But coccyx fractures commonly occur with direct falls onto the coccyx. Most coccyx injuries are contusions but can result in fracture dependent on the surface and height of the fall. Fractures may also occur during straining or friction, such as with rowing or cycling.

Clinical Presentation

The coccyx is three to five bones fused at the end of the sacrum. It serves as the attachment to the levator ani muscles

which control defecation function. It also plays a small part as the attachment of the gluteus maximus to aide in thigh extension. Athletes will present with tenderness over the coccyx that increases with sitting or getting up from a chair. That pain may also increase with bowel movements. Bruising and swelling may be evident on physical examination.

Diagnosis
Plain radiographs can show the fracture but are not necessary (Figs. 21.25 and 21.26). This can be diagnosed with physical examination and treated conservatively. Anal sphincter function should be evaluated. On rectal examination, the fracture may be palpated along the sacrococcygeal curvature.

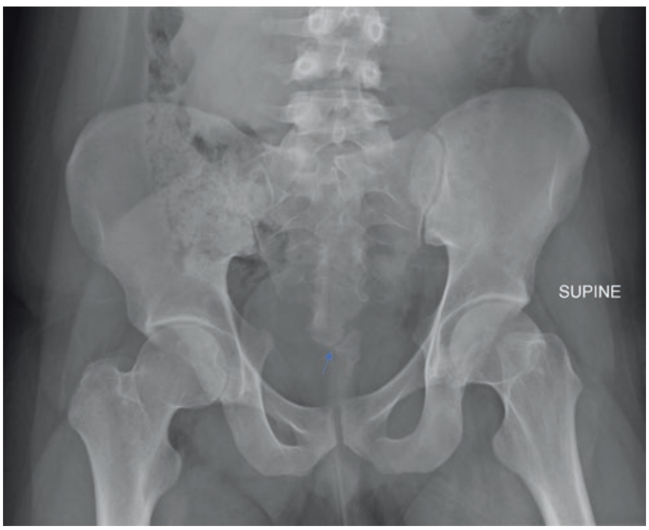

Fig. 21.25 A displaced coccyx fracture (arrow) in a 17-year-old male skateboarder as a result of a fall off a ramp during practice

Initial Management
The goal is to manage pain until the bone heals. The location of the coccyx and the number of muscles attached to it make it difficult to prevent it from moving while it is healing. Generally, pain will resolve on its own. A donut cushion can provide pain relief while sitting. The area may remain painful for a long period of time, even after the fracture has healed. Bed rest may be needed for a day or two or moving only as comfort allows. It is important to note that if the athlete has no neurological deficits or bowel incontinence, they can continue to play.

Indications for Orthopedic Referral
Surgery for a painful coccyx fracture is rare and does not often result in outcomes much better than conservative management. If pain continues and causes disability, a coccygectomy might be recommended.

Follow-Up Care
Continue to monitor for neurologic deficits. NSAIDs are appropriate for pain control and stool softeners may be considered to decrease pain with defecation.

Return to Sports
Patient can return to sports with a coccyx fracture. Limitation for return to play is pain.

Complications
Complications are rare in coccyx fractures. Athletes may experience prolonged pain especially if they continue to stress the region with continued participation.

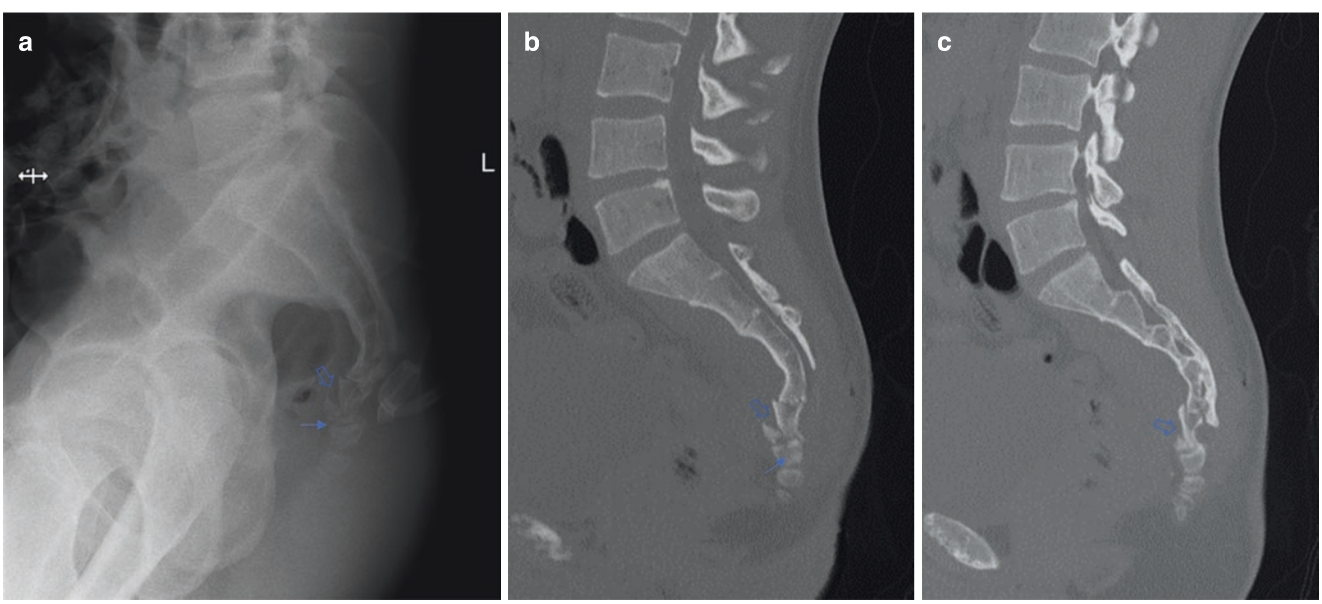

Fig. 21.26 Mildly displaced zone III sacral (S5) fracture (open arrows) and coccyx fracture (arrows) in a 26-year-old male as a result of a fall on his buttocks (**a**). CT images demonstrate the extent of the fractures (**b**, **c**)

Pediatric Considerations

Coccyx fractures in children are rare. In a sacrococcygeal joint fracture and dislocation, the Salter-Harris classification will be used [68]. As with adults, conservative management is standard of care with pain control, icing, and reducing pressure off the region while healing. Closed reduction has been proven to be ineffective in these cases [68].

Soft Tissue Injuries

Pelvic soft tissue injuries are relatively common in sports. Most injuries are mild and often the athlete does not seek medical care. Therefore, the incidence in sports is unknown. General superficial soft tissue injuries in sports are covered elsewhere in the book (Chap. 40 – Skin and Subcutaneous).

Fig. 21.27 Iliac crest contusion in a 60-year-old male as a result of a fall while mountain biking

Iliac Crest Contusion (Hip Pointer)

Mechanism of Injury in Sports

Blunt trauma to the iliac crest is a common injury to the hip during athletic activities and is referred to as a hip pointer. Anteriorly there are multiple muscular insertions and origins on the iliac crest including internal and external oblique muscles, transversus abdominis, sartorius, iliacus, rectus femoris, latissimus dorsi, tensor fasciae latae, and gluteus medius which can also be injured in the blow. This injury occurs most often in contact sports including rugby, football, and ice hockey.

Epidemiology

Hip pointers make up approximately 11% of hip injuries [69].

Clinical Presentation

Athletes presenting with a hip pointer will note acute pain, swelling, and hematoma over the iliac crest that occurred at a specific point in time coinciding with impact with another player or a hard surface (Fig. 21.27). They may have an antalgic gait and difficulty with trunk rotation and be limited in their ability to return immediately to sport due to pain.

Diagnosis

Examination will reveal ecchymosis or hematoma over the iliac crest with point tenderness. Often there is pain with activation of abdominal muscles or hip range of motion. There should be intact motor and sensory function about the hip, and deficits should prompt further evaluation of other injuries [70]. Generally, no imaging is needed for this diagnosis; however, if symptoms do not improve with conservative measures in the first several days, it is prudent to send the patient for radiographs to rule out an associated fracture or avulsion fracture. Ultrasound can be helpful in detecting intramuscular hematoma. MRI is indicated if there are concerns for injuries such as occult fractures.

Management

Immediate removal from play is recommended. Hip pointers are generally treated with conservative measures including ice, compression, rest, and anti-inflammatories. Careful monitoring of the athlete's pain and strength over a few weeks should show improvement and return to play is indicated when pain-free.

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22 Hip Joint

Stephanie W. Mayer, Kimberly M. Spahn, and Rebecca Griffith

Key Points

- Athletic hip injuries are becoming more prevalent in the sports community.
- Most injuries are the result of high impact, quick change of direction, or repetitive use.
- Hip dislocations and fractures are devastating injuries if missed in the acute setting.
- Adolescent and adult athletes are at risk for different types of hip injuries.

Introduction

Hip injuries in adolescent and adult athletes are a common occurrence and can range from muscle strains to hip fractures requiring emergent treatment. It is important to take into consideration the mechanism, which caused the injury as more severe injuries are associated with higher-energy collision sports. As with any high-energy injury, it is important to first perform basic Advanced Trauma Life Support (ATLS) protocols. Head and neck injuries should be evaluated for and properly stabilized prior to moving the athlete.

Anatomy

The hip is a ball and socket joint that allows for multidirectional movement. The range of motion of the joint is stabilized by a thickened capsule comprised of an extension of the three main ligaments about the hip, the iliofemoral, ischiofemoral, and pubofemoral ligaments (Fig. 22.1). The labrum is a fibrocartilaginous ring around the edge of the acetabulum which acts to maintain a suction seal around the femoral head, affecting the fluid pressures and congruity of the joint (Fig. 22.1). Multiple muscles cross the hip joint and are responsible for the various positions of the joint in space and provide the strength to change positions rapidly. Injuries can occur at any of these anatomical layers including fractures, dislocations, labral tears, and ligament and tendon sprains and tears.

Adult Athlete Hip Injuries

Tendonitis, Strains, and Tears

Acute or overuse injuries resulting in tendon pathology or bursitis about the hip joint are the most common causes of hip-related pain in an athlete. There are multiple proximal muscle origins near the hip joint that can be chronically injured or acutely strained or torn during athletic activities.

The major hip flexors include the iliopsoas, rectus femoris, sartorius, and tensor fasciae latae. Because the iliopsoas tendon passes anterior to the hip joint, it can become irritated with repeated flexion/extension of the hip or with rotation and can "snap" or catch over the femoral head or iliopectineal eminence and acutely can cause a sensation of a hip subluxation. This is referred to as internal snapping hip syndrome. Physical examination with repeated flexion/extension of the hip such as a bicycle motion or rising from a chair may be able to cause the snapping sensation or even an audible sound. Acute hip flexor strains or tears are reported most commonly in athletes with repeated forceful hip flexion such as jumping or kicking sports. Rectus femoris tendon strains or tears are more common with a kicking or sprinting mechanism while iliopsoas injuries occur more often with a quick change of direction [1]. Signs of these injuries during examination on the field will include focal pain and swelling in the anterior hip, pain with passive range of motion of the

S. W. Mayer (✉) · R. Griffith
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: Stephanie.Mayer@cuanschutz.edu

K. M. Spahn
Department of Orthopaedics, Naval Medical Center San Diego, San Diego, CA, USA

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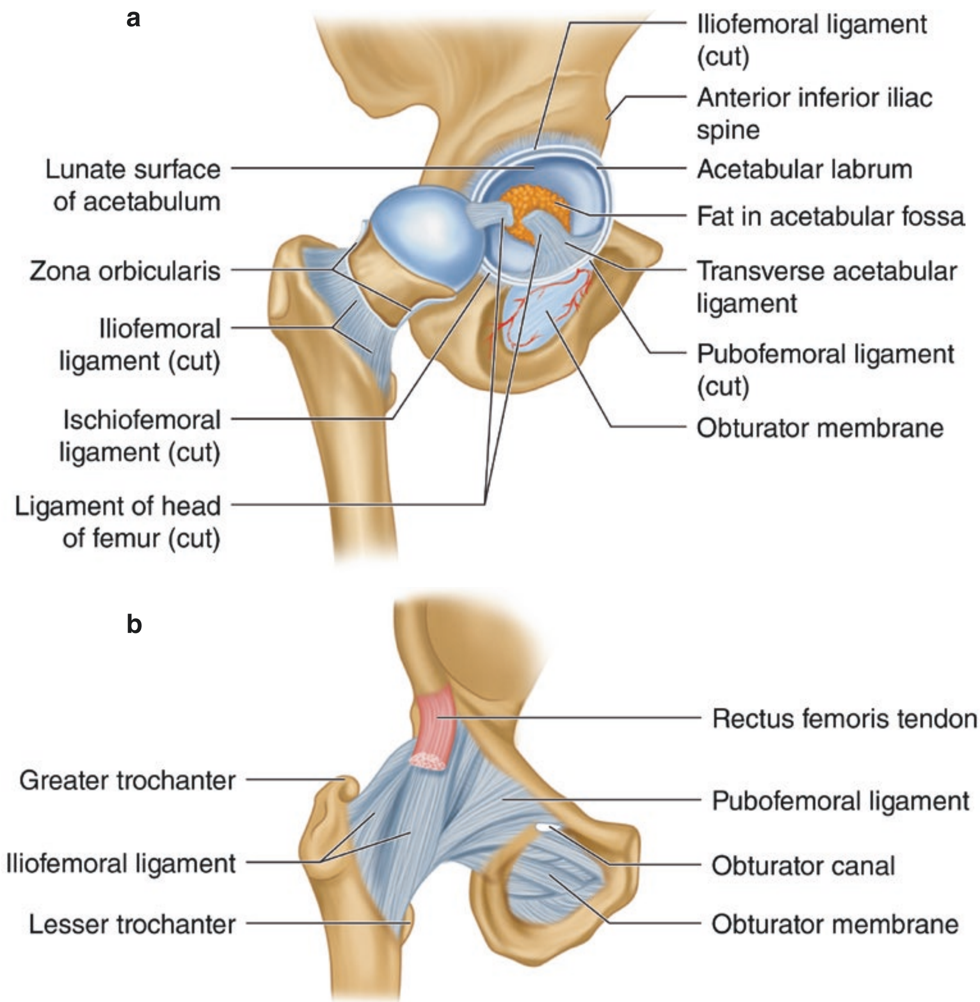

Fig. 22.1 Hip joint anatomy. Acetabulum and femoral head anatomy (**a**). Capsular anatomy and ligaments of hip joint (**b**)

hip or inability to perform active hip flexion, and, in cases of rectus femoris injuries, knee extension [2].

The proximal hamstrings originate from the ischial tuberosity and insert about the knee. Proximal hamstring tendinopathy is commonly seen in distance runners, sprinters, and long-distance athletes. It is generally thought to be due to chronic mechanical overload combined with placing the hamstrings on repeated stretch [2]. Acute proximal hamstring ruptures are common in waterskiing, soccer, American football, and rugby and usually occur with forced hip flexion with knee extension as would be seen with a high kick, slide tackle, or in dance routines [3]. Up to 12% of all hamstring injuries involve the proximal tendon and 9% result in a proximal tendon avulsion [4]. Examination of suspected proximal hamstring injury should begin with inspection to identify any posterior ecchymosis or swelling that can be associated with a rupture. Palpation of the tendinous origin of the hamstrings can isolate areas of pain or defect in the tendon, which signifies a tendon rupture or partial tear [3]. The athlete will often be unable to bear weight [5]. Strength should be tested in the prone position with resisted knee flexion and hip extension [6].

The adductor muscles of the hip include adductor longus, magnus, and brevis as well as pectineus, obturator externus, and gracilis. The adductor longus is most frequently the culprit for medial groin pain due to strain placed on the tendinous origin at the pubis. These injuries are caused when quickly decelerating with the lower extremity abducted and externally rotated [7]. With a great force, there is a risk of proximal adductor tear from the pubic origin. Adductor strains are commonly seen in soccer players, American football, ice hockey, swimming, and other high-energy sports [7, 8]. Athletes will present with medial groin pain. There can be tenderness to palpation over the pubic bone or along the tendon. Pain is exacerbated with passive abduction or with resisted adduction. Decreased strength with resisted adduction due to pain or rupture can be noted along with decreased hip range of motion [8].

The iliotibial (IT) band can cause external snapping about the hip or trochanteric bursitis as the IT band passes over the greater trochanter of the femur with extension/flexion of the hip. An acute snap can cause the sensation of hip subluxation.

Fig. 22.2 Right anterior inferior iliac spine (AIIS) avulsion fracture (arrow) in a 34-year-old female soccer player as a result of a forceful hip flexion during a kick

Initial diagnosis is generally made on physical examination for most chronic tendonitis and acute strains; however, MRI can be obtained to evaluate the extent of tendon tears if suspected. In the case of an acute avulsion injury, bony fragments attached to the avulsed tendon can be identified on plain radiography (Fig. 22.2). Ultrasound has also proven to be a valuable modality for identifying tendon rupture [9].

Initial management of chronic tendonitis should include rest, ice, anti-inflammatory medications, and physical therapy. Return to play can be successful when symptoms have resolved and the athlete is pain-free [10, 11]. If an acute simple tendon strain is suspected, the athlete should be removed from play; however, conservative treatment with rest, ice, and anti-inflammatory medications and partial weight bearing with crutches until pain-free is acceptable. Return to sports can commence when the athlete is pain-free and strength is regained. If symptoms do not improve with conservative management of tendonitis or a simple strain, referral to a sports medicine physician for further imaging is necessary. In cases of an acute tendon tear or avulsion, the athlete should not attempt return to play and prompt referral to a sports medicine physician or orthopedic surgeon should be placed.

Hip Dislocation

The native hip joint is stabilized not only by the bony anatomy of a ball and socket joint but also by a network of very strong capsular and ligamentous tissues. As such, a dislocation of the hip requires a considerable amount of force, which is more commonly encountered in high-energy motor vehicle accidents [12]. Hip dislocations in the athlete have been reported in higher-energy sports including American football, rugby, skiing, bicycling, gymnastics, and basketball, but make up only 1–2% of all hip injuries [13, 14]. Ninety percent of acute dislocations occur posteriorly as a fall with direct driving force to the knee with the hip internally rotated and adducted [14, 15]. Anterior and inferior dislocations are rare but occur in the setting of forced abduction and extension of the hip such as the splits in a gymnast or track and field athletes [12, 14].

While a hip dislocation in the sports setting is rare, prompt recognition and management are important to decrease morbidity. On physical examination, the athlete with an acute hip dislocation will experience severe pain and likely be unable to ambulate. With a posteriorly dislocated hip, the leg is held internally rotated and shortened (Fig. 22.3a). In the setting of an anterior dislocation, the leg is held in an externally rotated position with flexion and abduction [12]. A simple check of leg lengths and leg position can be a good indicator of hip dislocation if suspected. The neurovascular status of the leg should be evaluated acutely including distal pulses and sciatic nerve function. This can be tested by evaluating the sensation of the dorsal and plantar surface of the foot as well as the ability of the patient to dorsi- and plantar flex at the ankle or great toe. The athlete should be transported off the field on a gurney and should not attempt to weight bear. Prompt reduction should be attempted preferably in an emergency room if that is possible in the acute setting. There are on-field reduction maneuvers that can be attempted by an experienced individual if the diagnosis is extremely clear and should only be attempted in the setting of a posterior dislocation. Patient should be placed in the supine position. Gentle, in-line traction of the femur should be pulled with the hip flexed to 90°. Rotational manipulation is highly discouraged as this could worsen a fracture if present or cause one if not. If the athlete is able to relax the musculature surrounding the hip, a "clunk" may be felt as the hip reduces. Failure of reduction after several minutes should prompt the immediate transfer of the athlete to an emergency room for imaging and sedation prior to any additional reduction attempts [14]. Imaging is of the utmost importance and an X-ray should be obtained if at all possible prior to any attempts at reduction to preclude the presence of a femoral neck fracture.

Radiographs are needed prior to reduction attempts to identify any associated fractures of the acetabulum or femur and to confirm the direction of dislocation (Figs. 22.3b, 22.4a, and 22.5a, b). Following reduction (Fig. 22.3c), plain radiography should be performed (Figs. 22.3d, 22.4b, and 22.5c, d). In addition, CT can be utilized to identify any intra-articular fragments or fractures of the pelvis and hip [2]. MRI is also a valuable modality post reduction to review any soft tissue injuries including labral tears, chondral injuries, or the presence of loose bodies, which can be present in up to 80% [16, 17].

Fig. 22.3 A 19-year-old female with left hip dislocation as a result of falling while skiing. Note the internally rotated and shortened appearance of the leg (**a**). Plain radiographs confirmed the diagnosis of posterior dislocation without any obvious fracture (**b**). Clinical appearance post-reduction (**c**) radiograph shows reduced femoral head in acetabulum with symmetric joint space to uninvolved hip (**d**)

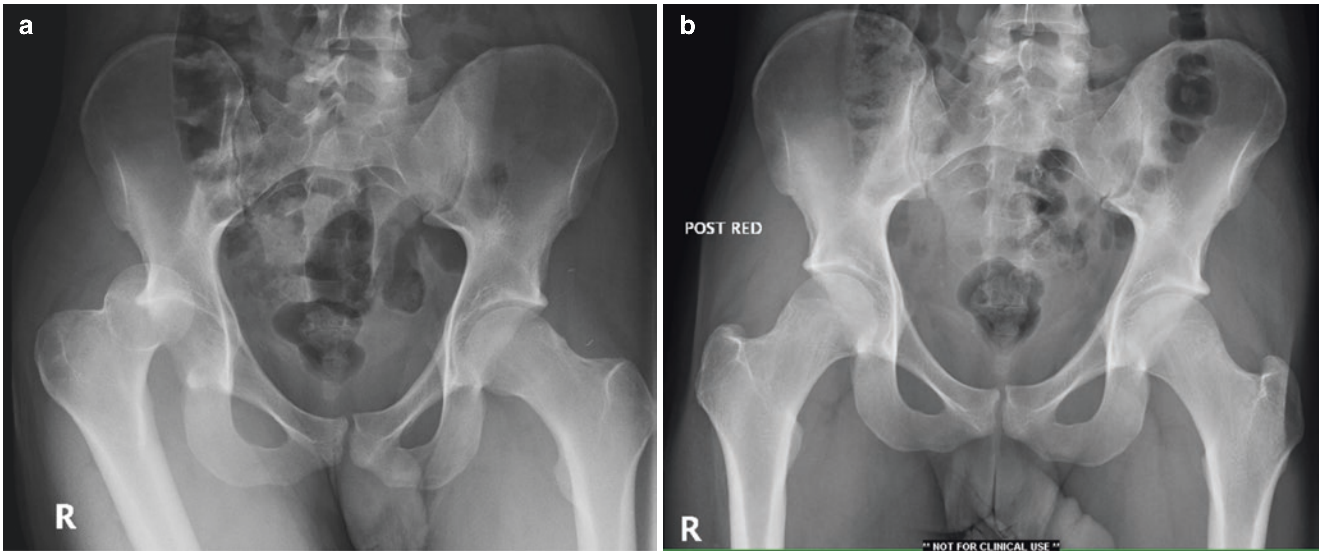

Fig. 22.4 Right posterior hip dislocation in a 23-year-old male as a result of a ski injury (**a**). Post-reduction radiograph reveals normal alignment without any fracture (**b**)

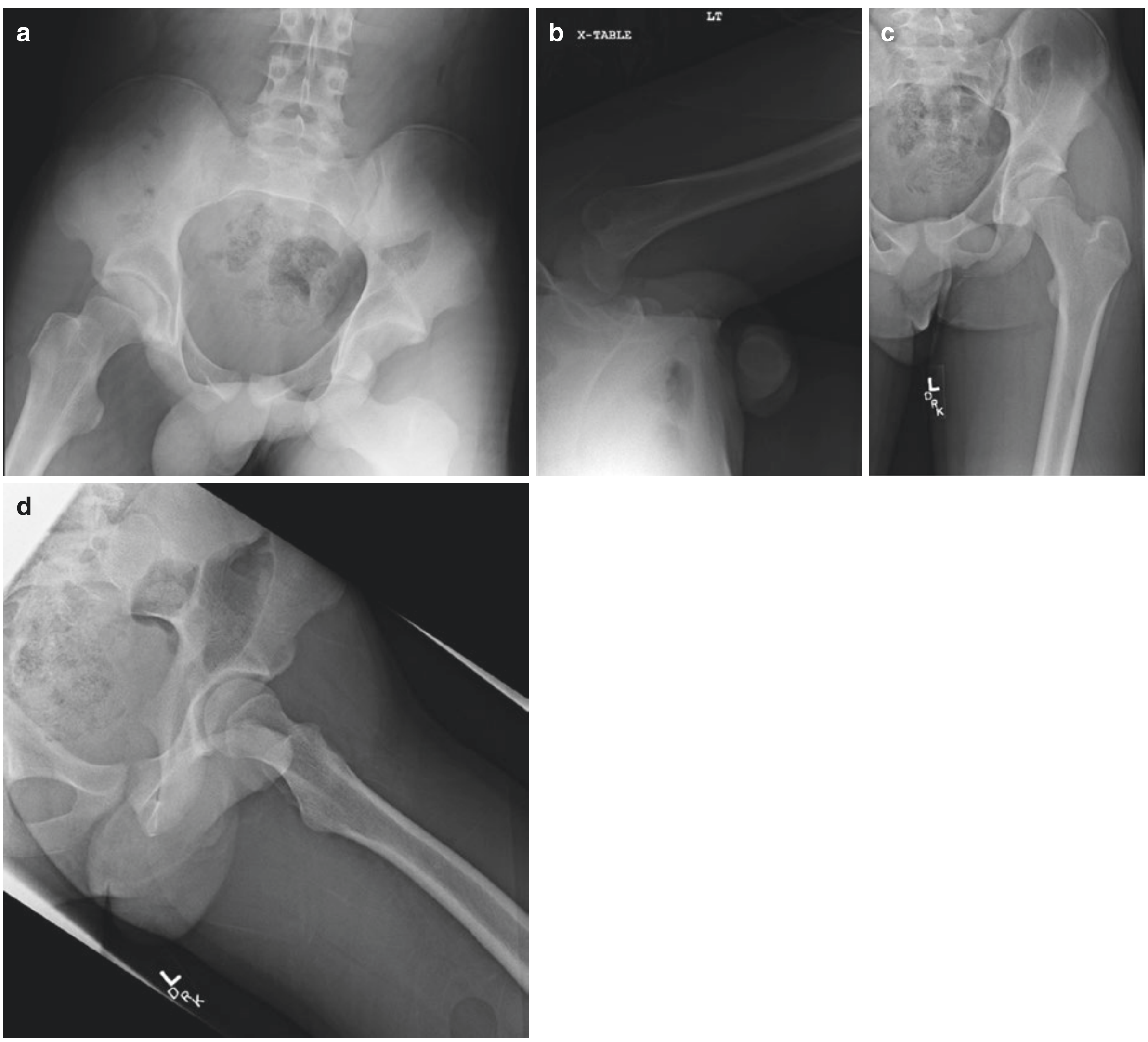

Fig. 22.5 Left inferior-medial hip dislocation in a 13-year-old male as a result of a ski injury (**a**, **b**). Post-reduction radiographs reveal congruent reduction (**c**, **d**)

Following reduction, range of motion can be safely started within the first week after dislocation, but posterior hip precautions should be followed for 6 weeks [14]. Physical therapy for strength and range of motion is important and return to sports generally will take 3–6 months. These athletes should be followed closely by an orthopedic surgeon for repeat exams and imaging as sequelae of AVN (avascular necrosis) and soft tissue injuries can occur [16–18]. Any onset of new pain should prompt removal from sports and evaluation.

Femoral Neck Fractures

Femoral neck fractures can be the result of chronic overuse causing stress fractures or caused by acute trauma. Femoral neck stress fractures in the young adult population most commonly occur in endurance athletes and are more prevalent in female athletes, military recruits, and runners [19]. They can be a complication in those with female athlete triad including eating disorder, irregular menses, and decreased bone density. Providers should maintain a high index of suspicion when athletes present with insidious onset of groin and vague thigh and hip pain with axial loading especially in the setting of recent increase in activity level as 5–10% of hip pain in this population is due to a stress fracture [19, 20]. Acute femoral neck fractures are less common in athletes and generally occur as the result of high-energy trauma (Fig. 22.6). Pre-existing stress fractures can complete and become acute displaced fractures.

On physical examination, patients with an acute traumatic femoral neck fracture are unable to bear weight. Often times there is an obvious deformity. Any ROM makes the pain worse. Patients with femoral neck stress fracture will gener-

Fig. 22.6 Right femoral neck fracture as a result of a ski injury in a 65-year-old female (**a**, **b**). The fracture was treated surgically with three screws for fixation (**c**)

ally have pain with axial loading and extremes of range of motion with decreased strength. Athletes with acute femoral neck fractures will be unable to ambulate and often hold the leg in an externally rotated position. Athletes with a suspected stress fracture of the femoral neck should be removed from sports participation and referred to a sports medicine physician. An athlete with a history of chronic hip pain who suddenly experiences acute sharp pain in the hip and inability to ambulate should be removed from play and made non-weight bearing with crutches and undergo urgent X-ray imaging of the pelvis and hip to diagnose a possible completed fracture. Athletes with an acute femoral neck fracture should be removed from the field on a gurney and transported to an emergency room for radiographic evaluation.

If a stress fracture is located on the compression side of the neck (inferior/medial), they are generally treated with protected weight bearing and allowed gradual return to activities after 6 weeks if pain-free [21]. Full return to sports participation may take between 3 and 12 months [22]. If the fracture occurs on the tension side of the neck (superior/lateral), there is a very high rate of completion of the fracture or subsequent displacement and surgical intervention is warranted [23]. Surgical fixation is always indicated for an acutely displaced femoral neck fracture or a stress fracture that goes on to displace (Fig. 22.7) [24].

These fractures generally heal in 6–12 weeks and physical therapy is necessary once healed to increase strength and hip mobility prior to return to sport. These athletes should be followed closely by an orthopedic surgeon for repeat exams and imaging to watch for the occurrence of the sequelae of AVN. Any onset of new pain should prompt removal from sports and evaluation [25].

Fig. 22.7 Femoral neck stress fracture prophylactic fixation with three screws

Acetabular Fractures

Acetabular fractures occurring during sports participation are rare [26–28]. An acetabular wall fracture associated with a hip subluxation or dislocation when the femoral head strikes the acetabular rim can occur (Fig. 22.8). Sports most commonly associated with this injury are those in which the potential for a high-energy injury or fall is more likely such as football, motor sports, and downhill skiing or snowboarding. The suspicion for an acetabular fracture should be higher when there has been a high-energy blow to the limb or a fall from height; however, there are reports of acetabular wall fractures with lower-energy injuries as well. Patients will present with significant pain and inability to bear weight, with pain generally in the groin, but possibly in the posterior hip as well. Physical examination can be difficult due to very limited range of motion due to pain. The fracture will not be palpable nor cause a definite external deformity unless there has been some subluxation of the hip joint, in which case the limb will appear similar to a femoral neck fracture and be shortened and possibly rotated. Swelling and bruising may not be visible immediately due to the depth of the joint, although scrotal or labial swelling or hematoma can occur [28]. Important to the initial evaluation is a neurovascular evaluation, as up to 20% of acetabular fractures are associated with a sciatic nerve injury [29]. Immediate non-weight bearing on the field and limited range of motion should be initiated, and the athlete should be taken to the emergency department at that time for imaging. Diagnosis generally includes plain radiographs and a CT scan (Figs. 22.8, 22.9, and 22.10). Non-displaced fractures can be missed on plain radiographs, and so CT is commonly ordered if there is suspicion for a fracture. If this injury is diagnosed, an inpatient orthopedic consultation is generally obtained for surgical versus nonsurgical decision making. If nonoperative treatment is indicated, a period of 6–8 weeks of non-weight bearing ambulation will occur for fracture healing, followed by physical therapy for gait training and strength. Return to sports will depend on the length of time to full strength, but generally will be around 6 months. Complications of acetabular fractures include post-traumatic arthritis, stiffness, hip instability, and heterotopic ossification formation [30].

Femoroacetabular Impingement and Labral Tears

The hip is a ball and socket joint that allows for multidirectional range of motion. As such, extremes of motion can result in dynamic femoroacetabular impingement (FAI) of the femur and acetabulum leading to tearing of the labrum [31, 32]. Morphologic deformities of the femur or acetabulum can cause FAI and are commonly noted in athletes. Labral tears and FAI can occur in many different sports and

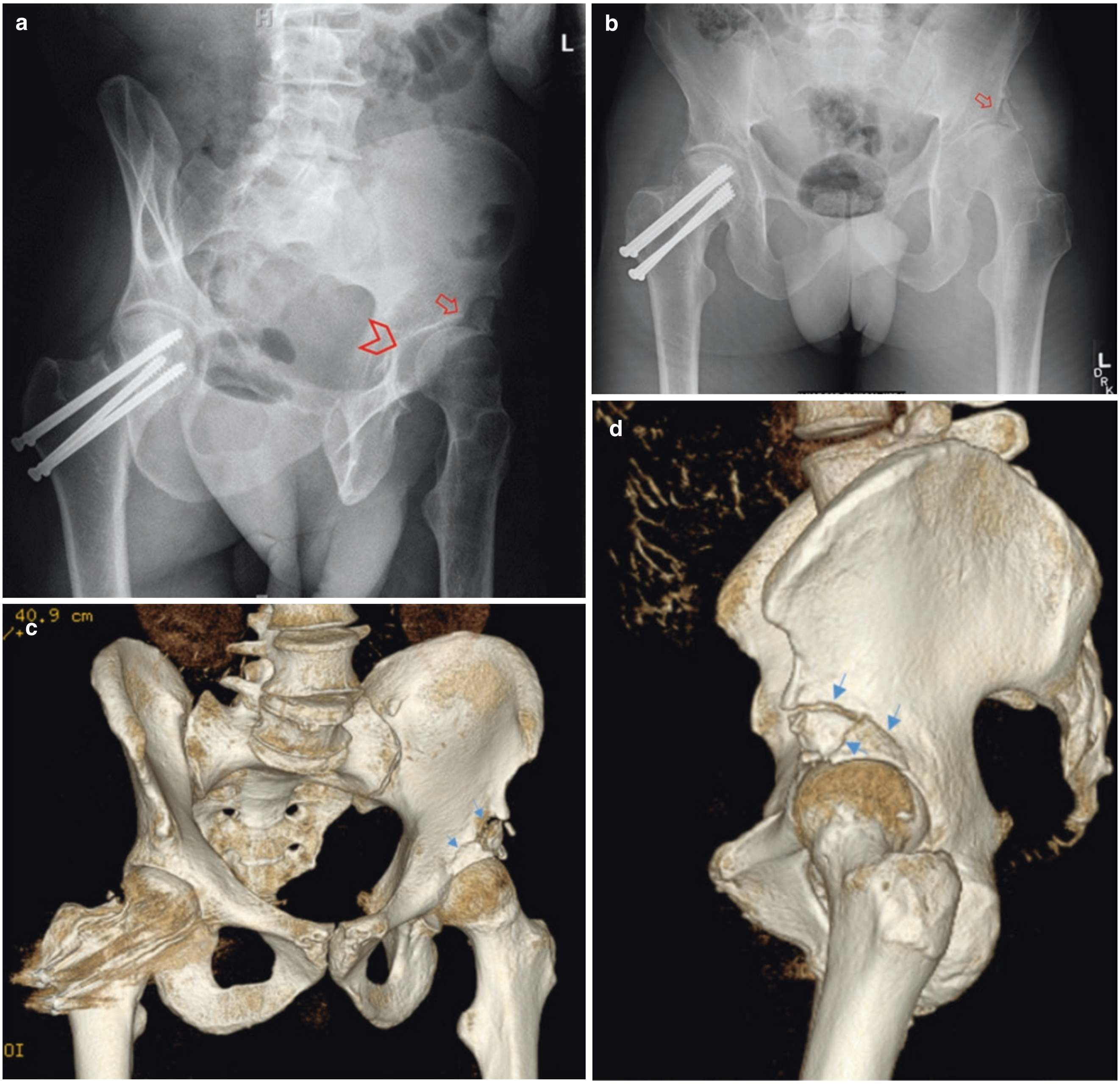

Fig. 22.8 Plain radiographs show a left acetabular fracture (open arrows) and hip joint subluxation (arrowhead) in a 44-year-old male as a result of a mountain bike injury (**a**, **b**). The patient already had a right femoral neck fracture with fixation from previous injury few months earlier. The fracture is not visible on AP view (**b**). Three-dimensional CT demonstrates reduced subluxation and the fracture (arrows) (**c**, **d**)

are being recognized most commonly in soccer, lacrosse, American football, ice hockey, dance, and gymnastics.

Typically, FAI is not found as an acute injury, but is described as insidious development of worsening groin pain. History may include mechanical symptoms including popping or catching of the hip during activities involving rotation and flexion. Extremes of motion, especially hyperflexion, cause pain. Athletes may describe pain with common sporting activities such as running and squats and even sitting for long periods.

Physical examination should begin with hip maneuvers including range of motion, strength, and stability. Careful attention should be paid to pain and limited internal rotation of the hip when flexed to 90°. Since there is variability in the hip range of motion, the affected side should be compared to the unaffected side [33]. Pain due to a labral tear can often be elicited with the hip in a flexed, adducted, and internally rotated (FADIR) position [2]. On-field examination consistent with FAI or a labral tear does not preclude the athlete from participation; however if pain is severe, removal from

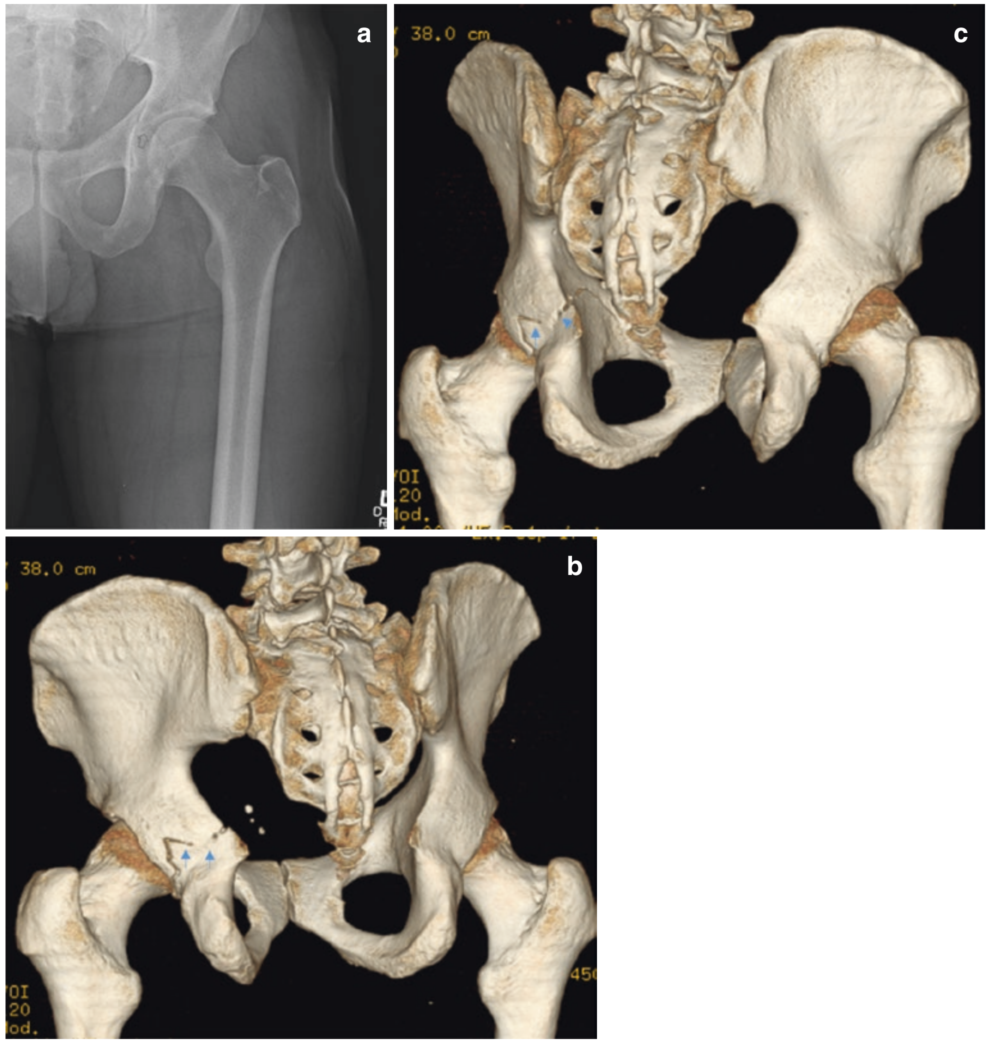

Fig. 22.9 Plain radiographs show a left acetabular fracture (open arrow) in a 35-year-old male as a result of a mountain bike injury (**a**). Three-dimensional CT confirmed the diagnosis (arrows) (**b**, **c**)

sport is indicated and further examination to rule out other conditions should be performed.

Initial treatment for FAI can be conservative to include NSAIDs, rest, and physical therapy. Referral to an orthopedic surgeon is advised for co-management and confirmation of the diagnosis after X-rays and possibly an MRI. Often non-surgical management is successful, with return to sports when pain-free and strength is regained. Often this will require a period of 2–6 weeks of treatment and therapy. However, some cases are refractory to conservative treatment and require surgical intervention. These athletes have an 80–95% return to sports rate after 4–8 months of postoperative recovery.

Adolescent Athlete Hip Injuries

Pelvic Apophyseal Avulsion Fractures

Avulsion fractures usually occur in athletes from 13 to 17 years of age, as the pelvic apophyses are not fully fused. Incomplete closure of the growth plates of the pelvis combined with a sudden and forceful contracture of the muscles, which originate from these areas, causes the apophysis to avulse from the pelvis (Table 22.1). Apophyseal avulsion fractures can occur with falls, kicking, sprinting, or jumping and accordingly are seen in sports such as soccer, football, and track due to the explosive nature of these sports [34].

Often the athlete will report a "pop" or "snap" especially after a kick or jump. The athlete often reports pain and weakness of hip or knee flexion. The athlete may report inability to bear weight or may walk with an antalgic gait. There can be a history of pain in these areas (apophysitis) prior to the injury in both avulsion fractures. The presence of an avulsion fracture can be confirmed with radiographic evaluation (Figs. 22.11, 22.12, and 22.13) [35, 36].

It is important to assess both the hip and the knee as a "pop" is also commonly associated with knee ligament ruptures. The knee exam should include assessment of effusion, tenderness to palpation, and ligamentous exam if tolerated. Assessment of strength and gait is important to help deter-

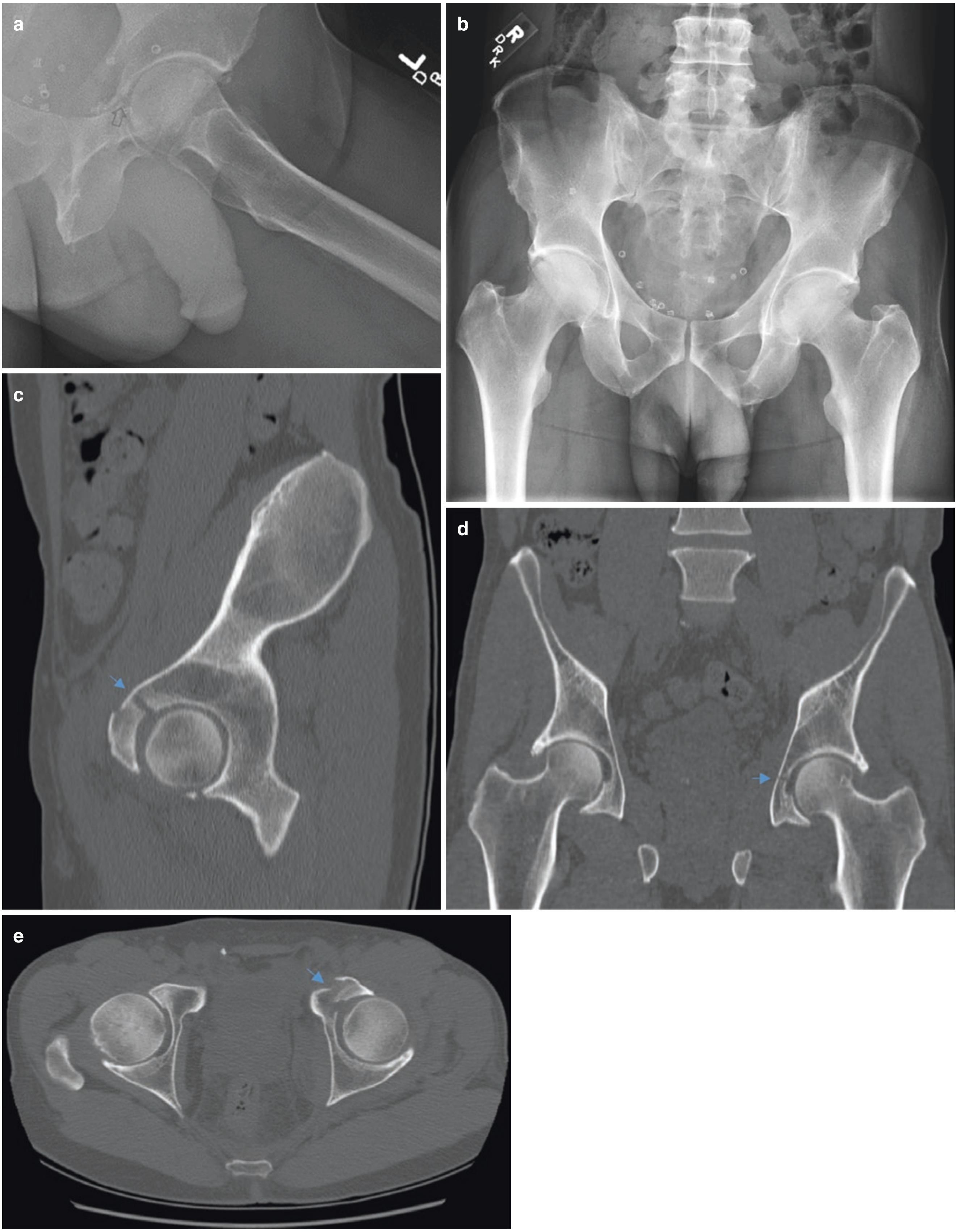

Fig. 22.10 Plain radiographs show a left acetabular fracture (open arrow) in a 58-year-old male as a result of a snowboarding injury (**a**). The fracture is not visible on AP view (**b**). CT confirmed the diagnosis (arrows) (**c**–**e**)

mine where the injury has occurred. Often the area of the avulsion is focally tender to palpation, and pain is reproduced with passive stretching and strength testing of the suspected muscle involved. For example, an avulsion of the rectus femoris muscle would be tender to palpation about the AIIS and pain would be reproduced with resisted extension of the knee. Once this injury is suspected or has been identified, the athlete should be removed from play. Initial treatment is usually supportive to include off-loading of the injured extremity with crutches, use of ice to the area, and anti-inflammatories until the athlete can be formally evaluated with an X-ray and exam. A referral to a sports physician or orthopedic surgeon is recommended for radiographic examination and treatment. An apophyseal avulsion fracture is evident on most AP pelvis films. Occasionally, they require additional views of the pelvis [34, 35, 37].

The mainstay of treatment for these avulsion injuries is nonoperative with a period protected weight bearing and rest from sport. Bracing can also be used for comfort with the hip flexed to 20°. Physical therapy is helpful to improve strength, range of motion, and flexibility to prevent further injury upon return to play. Return to sports is often restricted for a period of 6 weeks to allow sufficient healing of the fracture and to regain strength. Surgical treatment is reserved for extremely displaced fractures or symptomatic nonunions or malunions. Conservative treatment with protected weight bearing, ice, and NSAIDs is generally successful; however, when these injuries are missed and continued participation in sports is allowed, a delay in healing may occur [34–36, 38].

Table 22.1 Avulsion fracture location and muscle insertion associated with avulsion

Location	Muscle
Iliac crest	Tensor fasciae latae
Anterior superior iliac spine	Sartorius
Anterior inferior iliac spine	Rectus femoris
Ischial tuberosity	Hamstrings
Pubic rami	Adductor/gracilis
Greater trochanter	Gluteus medius/minimus
Lesser trochanter	Psoas

Fig. 22.12 Left minimally displaced AIIS avulsion fracture (arrow) in a 13-year-old male

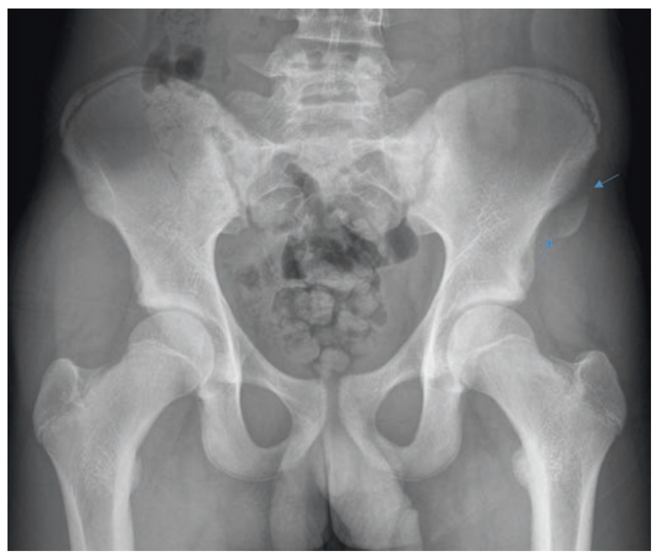

Fig. 22.13 Left anterior superior iliac spine (ASIS) avulsion fracture (arrows) in a 14-year-old male

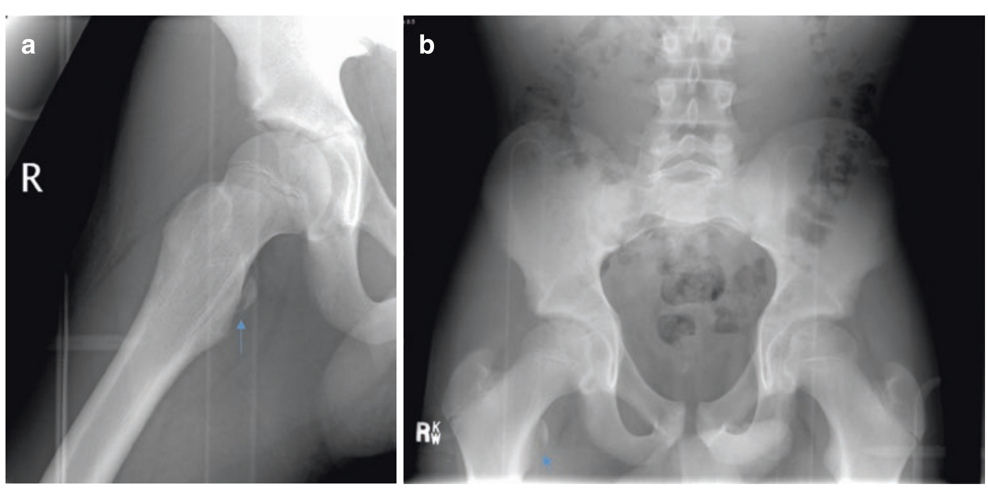

Fig. 22.11 A 14-year-old boy with an avulsion of the right lesser trochanter (arrows) as a result of a kick during a soccer game (**a**, **b**)

Traumatic Adolescent Hip Dislocations

Traumatic hip dislocations in adolescent athletes are very rare and are associated with high-energy mechanisms such as impact sports, motor vehicle accidents, or falls. The most common sports to have occurrences of hip dislocations are basketball, American football, rugby, snowboarding, and skiing. A posterior dislocation most commonly occurs with a posteriorly directed force directed along the femur with the hip in a flexed and adducted position (Fig. 22.5). The knee is also commonly in a flexed position, which can also result in injuries about the knee. In anterior dislocations the leg is usually in a flexed, abducted, and externally rotated position [39, 40]. Posterior dislocations account for 80–90% of sports-related dislocations. Anterior dislocations occurring during sport are more likely associated with skiing injuries. Given the high energy associated with these injuries, 13% have an associated fracture.

In very young athletes the dislocation event may not be the result of a high-energy collision. Less energy is required for dislocation of the hip due to the cartilaginous acetabulum and overall laxity of surrounding structures in young children. In either case, these injuries are considered an orthopedic emergency requiring immediate medical attention for reduction [14, 40].

The athletes usually present with severe pain, inability to bear weight, and the leg held in shortened, abducted, and internally rotated position. The dislocated leg may be resting on the uninjured leg, and the greater trochanter may appear prominent. Once the basic ATLS protocols have been performed, a focused exam on the injured extremity can begin. It is imperative to perform a neurovascular examination on the athlete. The examiner must assess that the femoral and distal pulses are present and that the foot is warm and well perfused. Motor examination can be compromised due to injury or stretching of the nerves in the leg. If the sciatic nerve has been injured, this should be documented prior to reduction of the hip. The knee also should be examined as posterior cruciate ligament injury can coexist with dislocations due to the flexed position of the knee with a posteriorly directed force causing this injury. Assessment of swelling, ecchymosis, and tenderness to palpation about the joint as well as a ligamentous exam can help to diagnose an associated knee injury.

It is important to prevent further attempts at weight bearing and remove the athlete from the field on a gurney. The athlete should be emergently transferred to a hospital for immediate reduction of the dislocation. This is not a reduction that should occur on the field. Fractures such as ipsilateral femoral shaft fractures, acetabular fractures, or femoral neck fractures are commonly associated with posterior hip dislocations and should be evaluated with X-rays prior to reduction attempts. Given the high likelihood of surgical treatment under anesthesia, there should be no further food or drink provided to the athlete on the field. After the dislocation has been reduced, follow-up X-rays, CT scan, or MRI is obtained to evaluate for loose bodies or intra-articular pathology or to further characterize associated fractures.

Hip subluxation events are more common than full dislocations. Subluxation events are more difficult to diagnose at the time of initial injury, but the athlete may have recurrent subluxation events or present with chronic hip pain. Initially the athlete may report severe groin pain, a popping or slipping sensation of the hip, and inability to bear weight; however, the hip is reduced and no deformity is present. The examination may demonstrate pain with passive and active range of motion of the hip. The knee and neurovascular status should also be evaluated in these cases. If a subluxation is suspected, these athletes should be made non-weight bearing until evaluated by a physician. In cases of subluxation, the X-rays are often normal, but there can be associated fractures of the acetabulum or soft tissue injuries to the labrum cartilage or capsule of the hip joint. MRI may be necessary to identify a subluxation [14, 17, 40].

Timing of return to sport depends on the severity of the injury and treatment needed and the presence or absence of associated fractures, but may require 3 months for return. Recurrent dislocations and AVN can also occur after the initial injury, and the onset of pain following return to sport should prompt removal from sport and evaluation by a physician [19].

Femoral Neck Fractures

In adolescent athletes, femoral neck fractures are usually the result of a high-energy mechanism. These injuries are considered an orthopedic emergency due to the risk of disruption to the blood supply of the femoral head resulting in avascular necrosis (AVN). While these fractures are rare in the young athlete, early recognition is imperative to expedite treatment and prevent AVN [41].

Femoral neck fractures are usually the result of a high-energy fall or impact as the result of a direct blow to the hip in an abducted position. When the mechanism of injury is not the result of a high-energy incident, suspicion for a pathologic or insufficiency fracture, such as a neoplastic process or stress fracture, should be raised. A history of groin pain prior the injury, amenorrhea, fevers, chills, weight loss, or rapid increase in training could be signs of a possible pathologic fracture [42].

The severe sequelae of avascular necrosis in femoral neck fractures occur as a result of disruption to the blood supply of the femoral head. The medial and lateral circumflex arteries provide the majority of the blood supply to the femoral head. The terminal branches of the arteries are intracapsular, and when a displaced fracture occurs, the blood supply can be

compromised. Prompt recognition and reduction with fixation of these fractures to restore the blood flow to the femoral head can improve outcomes [43].

Athletes usually report a severe onset of pain and inability to bear weight. Once the basic ATLS protocols have been performed as necessary, the assessment of the injured extremity can begin. The injured extremity may show no sign of obvious deformity or can be held in an abducted, shortened, and externally rotated position depending on the amount of displacement. Pain is reproduced with passive motion of the hip to include log roll, flexion, and extension as well as attempts at active motion. Ecchymosis and swelling may or may not occur with these fractures. Part of the on-field assessment should include a neurovascular examination and prevention of any further attempts at weight bearing [43, 44].

The athlete should be removed from the field on a gurney with immediate transfer to a hospital with the capabilities of treating adolescent hip fractures. Given the high likelihood of surgical treatment with these injuries, no further food or drink should be given to the athlete until evaluated by the treating physician.

Diagnosis of the femoral neck fracture is confirmed with pelvis and hip X-rays (Figs. 22.14 and 22.15). If the fracture is non-displaced and not visible on plain X-ray but suspicion for this fracture remains, other modalities such as CT or MRI have proven to be useful in diagnosing an occult hip fracture [44, 45].

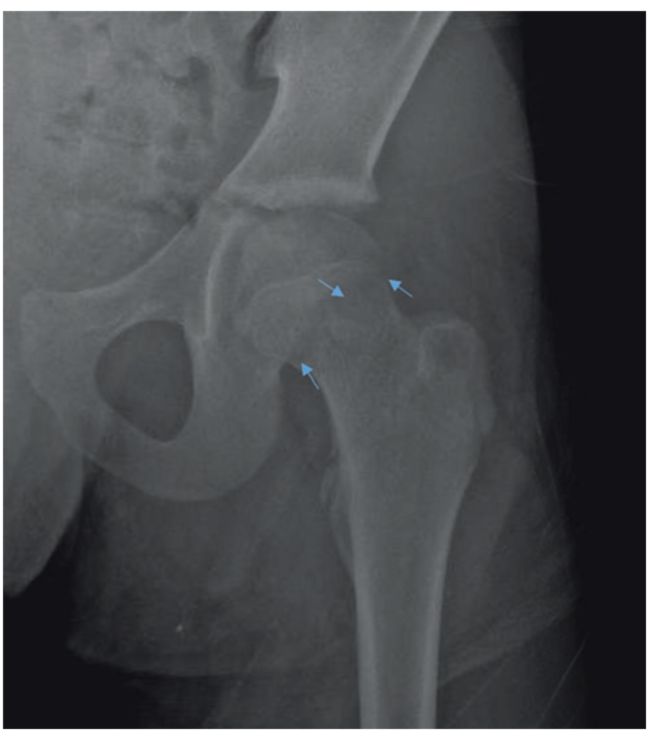

Fig. 22.14 An 11-year-old male with minimally displaced left femoral neck fracture (arrows)

Femoral neck fractures require urgent anatomic reduction and stable fixation for treatment. The risk of nonunion, malunion, and avascular necrosis is increased with delayed treatment or nonanatomic reductions of the femoral neck [45].

After surgical treatment, the athlete will be kept non-weight bearing for 6–12 weeks while the bone is healing. Physical therapy is recommended for improvement in range of motion, strength, and eventual gait training or return to sport programs. The diagnosis of avascular necrosis can occur even up to a year after the injury. If the athlete has a recurrence of hip pain following return to sports, they should abstain from sport until evaluated by their physician [43–45].

Acetabular Fracture

Acetabular fractures occurring during sports participation in children and adolescents are rare. Most will occur in an adolescent population. A high-energy injury will generally be needed to fracture the relatively elastic pelvis in this age group, although low-energy injuries have also been reported [46]. As in the adult population, an acetabular wall fracture may be associated with a hip subluxation or dislocation.

Fig. 22.15 An 8-year-old male with a displaced femoral neck fracture. Note the varus deformity and shortening of the limb due to muscular forces

Similar to adult athletes, sports most commonly associated with this injury would be those in which the potential for a high-energy injury or fall is more likely. Patients will present with inability to weight bear and painful range of motion. Depending on the amount of displacement or associated hip dislocation, there may be shortening and a rotational deformity of the limb. Examination on the field can be difficult due to pain and the lack of external visible signs of the injury. Important to the initial evaluation is a neurovascular evaluation, as up to 20% of acetabular fractures are associated with a sciatic nerve injury [29]. Immediate non-weight bearing on the field and limited range of motion should be initiated, and the athlete should be taken to the emergency department at that time for imaging. As in adults, diagnosis generally includes plain radiographs and a CT scan (Figs. 22.8, 22.9, 22.10, and 22.16). Non-displaced and even displaced frac-

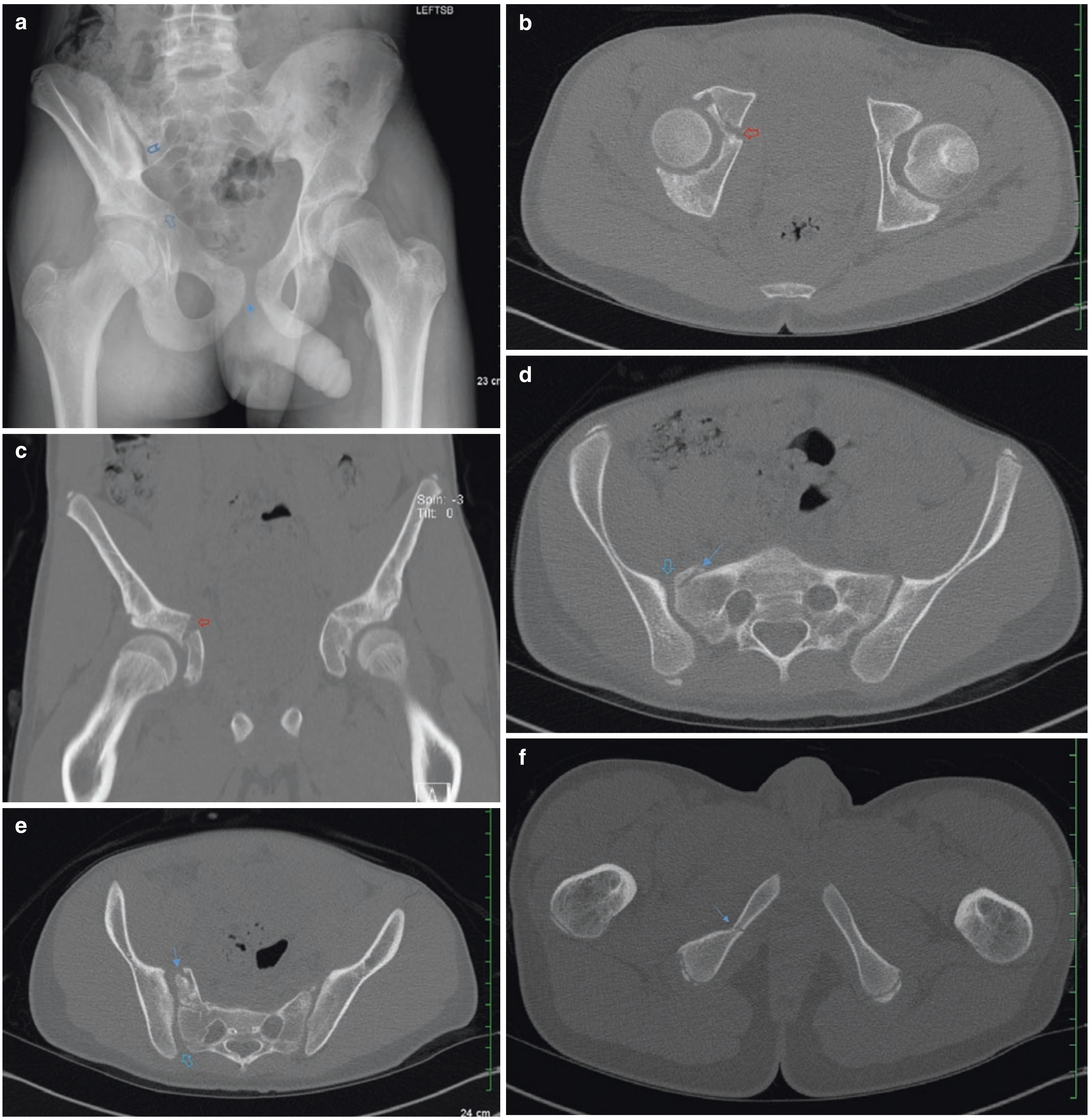

Fig. 22.16 A 15-year-old boy with a dirt bike injury. Plain radiographs show widening of the pubic symphysis (arrow) and SI joints (arrowhead) suspicious for pelvic fracture as well as acetabular fracture (open arrow) (**a**). CT confirmed the displaced anterior column fracture of the right acetabulum (red open arrows) (**b**, **c**). A comminuted right sacral ala fracture (arrows) and diastases of the right SI joint are noted (blue open arrows) (**d**, **e**) as well as a non-displaced right inferior pubic ramus fracture (arrow) (**f**)

tures can be missed on plain radiographs due to the potential for a fracture propagating through the uncalcified cartilage in the wall of the developing acetabulum. CT can be helpful for diagnosis, but often MRI is utilized due to its ability to evaluate the cartilaginous walls of the acetabulum before skeletal maturity [47]. An inpatient orthopedic consultation is generally obtained for surgical versus nonsurgical decision making. If nonoperative treatment is indicated, a period of 6–8 weeks of non-weight bearing ambulation will occur for fracture healing, followed by physical therapy for gait training and strength. Return to sports will depend on the length of time to full strength, but generally will be around 6 months. Complications of acetabular fractures include post-traumatic arthritis, physeal injury with a growth disturbance, neurovascular injury (particularly the sciatic nerve), hip instability, stiffness, and heterotopic ossification [48].

Acute Slipped Capital Femoral Epiphysis

Acute slipped capital femoral epiphysis (SCFE) is the most common acute adolescent hip disorder accounting for 10.8 cases per 100,000 adolescents [49–51]. This condition affects athletes between 9 and 13 years of age with a peak onset of 11 years of age. It is more prevalent in males and has a higher incidence in Hispanic and African American children than Caucasians [49–51]. SCFE occurs when the epiphysis of the femur remains in the acetabulum and the metaphysis of the proximal femur dissociates at the physis and slips proximally and laterally. The devastating results of this condition can be avascular necrosis, femoral acetabular impingement, or chondrolysis [50].

The cause of SCFE is likely multifactorial to include abnormal forces seen across the physis from obesity or the orientation of the physis in relation to the metaphysis. Endocrine abnormalities such as hypogonadism, hypothyroidism, or renal osteodystrophy can weaken the physis causing the metaphysis to slip from the epiphysis [50, 51].

There can be a prodromal history of hip or knee pain in the affected limb. When there is a history of pain over 3 weeks in the hip and history, imaging, and examination support the diagnosis of SCFE, this is considered chronic SCFE. Cases of a rapid onset of hip pain for only a few days to less than 3 weeks are classified as an acute SCFE versus a chronic SCFE in which pain has been ongoing for more than 3 weeks. Ninety percent of athletes will still be able to bear weight despite the pain, and this is termed a stable SCFE rather than an unstable SCFE in which the athlete cannot bear weight. Therefore, the ability to bear weight does not rule out this condition. All athletes with symptoms concerning for SCFE should be evaluated by a physician. Any complaints of knee pain should prompt examination of the hips during evaluation as some athletes may present with knee rather than hip pain [51, 52]. In chronic, stable SCFE, the athlete may present with relatively mild pain. Chronic, stable SCFE can progress to an acute stage and may become unstable. For example, a player may play through several weeks of lower-level hip pain and then have a sudden and sharp increase in the pain with inability to bear weight.

In acute unstable SCFE, the athlete may report groin, thigh, or knee pain. The leg is held in external rotation and there is very limited internal rotation on examination. It is important to inquire about symptoms on the contralateral hip as SCFE often occurs bilaterally. The patient should be made non-weight bearing, and in cases of acute, unstable slips, it may be too painful to even use crutches. It is important to off-load the hip to prevent further progression of the slip since more severe slips are associated with poorer outcomes and higher incidence of AVN [49, 53, 54].

The diagnosis of SCFE is generally made on plain radiographs (Fig. 22.17) and classified as stable or unstable based on physical exam. An unstable SCFE will generally be more

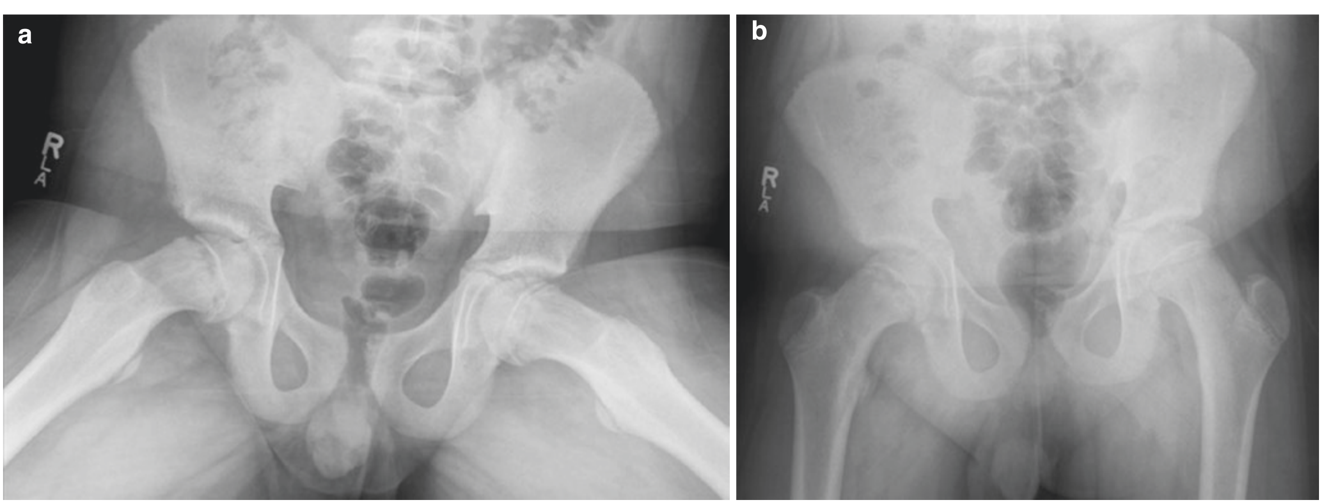

Fig. 22.17 A 13-year-old male with bilateral (right worse than left) SCFE (**a**, **b**)

displaced on plain radiographs. If plain radiographs appear normal, but the suspicion for SCFE remains, CT or MRI can be used to identify subtle deformity or edema at the physis that would be more consistent with a stable SCFE as it is not displaced. SCFE requires prompt surgical intervention at the time of diagnosis. The treatment varies from pinning the affected hip (and possibly the contralateral hip at risk) with a screw to more invasive realignment procedures. In stable slips the risk of osteonecrosis is very low when recognized and treated. In the more acute, unstable slips, the risk of AVN approaches 50%, thus the importance of prompt recognition and treatment [54, 55].

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Femur

23

Lauren Oberle and Morteza Khodaee

Key Points

- Femur fractures are often present in the setting of polytrauma as both trochanteric and diaphyseal fractures require high-velocity forces in individuals with normal bony anatomy or lower-energy forces in individuals with abnormal bone structure.
- Trochanteric fractures present with internal rotation, hip flexion, and limb length shortening, while diaphyseal fractures can present with severe pain, limb deformity, limb shortening, or decreased range of motion of the ipsilateral hip and knee.
- Diaphyseal fractures are a medical emergency and can be complicated by fat emboli or hemorrhage leading to hemodynamic instability or shock.
- All athletes with trochanteric and diaphyseal fractures should have orthopedic referral for surgical management unless surgery is contraindicated due to medical comorbidities or the injury is an isolated avulsion fracture of the greater or lesser trochanter.
- Athletes should be able to return to sport after gaining complete range of motion, strength, and the ability to perform sport-specific tasks and skills.

Introduction

The femur is the strongest bone in the body, and, due to its size and structure, it requires large forces or structural abnormalities to fracture the upper leg [1]. There are several different types of injuries seen in the femur including soft tissue injuries, fractures, stress fractures, and avulsion fractures. This chapter primarily focuses on the femur and bony injuries including fractures of the diaphysis and trochanteric regions.

L. Oberle
Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Anatomy

As the longest bone in the human body, the femur is the strongest and heaviest bone, measuring at approximately one fourth of the height of an athlete [1]. The important bony landmarks of the femur begin proximally at the femoral head which sits against the articular cartilage of the acetabulum and affixes to the remainder of the bone by the neck of the femur. The two trochanters of the femur protrude laterally from the junction between the neck and diaphysis (shaft). The extracapsular zone between the trochanters is known as the intertrochanteric area and is comprised of cancellous bone which is able to endure twisting forces [2–4]. The greater trochanter occupies the lateral edge of the proximal diaphysis, while the lesser trochanter lies along the posteromedial surface. The diaphysis (shaft) of the femur is divided into three parts that are considered proximal, medial, and distal based on their respective anatomic locations. The diaphysis is made of cortical bone which is especially strong and difficult to fracture [2]. The medial and lateral condyles arise from the distal diaphysis and project medially and laterally to make up the end of the femur. The muscles of the upper leg are divided into three compartments with associated nerves and vasculature [2]. The anterior muscles flex the hip and extend the knee, the medial muscles primarily adduct the leg, and the posterior muscles function to extend the hip and flex the knee [4].

Soft Tissue Injuries

Injuries to the soft tissues surrounding the femur are more common than bony injuries of the femur, and many are discussed elsewhere in this book. These injuries include, but are not limited to, muscle, tendon, and ligament injuries. Muscular injuries include contusion, strain, and rupture. Following this

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type of injury, the athlete should practice weight-bearing as tolerated unless the injury is a complete tear or rupture; in this case, an orthopedic consult is recommended [2]. There are no set guidelines regarding return to play; however, based on the clinical situation, athletes should be safe to continue play when the clinician finds an absence of obvious bony injuries on examination, the player is able to bear weight on the injured leg, and the athlete can appropriately complete the roles of his/her position in the sport [5]. Athletes who have quadricep contusions are at risk of developing hematomas. Therefore, icing can decrease further development of the hematoma by vasoconstriction. Myositis ossificans, another common sports injury, is the ossification that may develop secondary to a prior skeletal muscle injury. Athletes present with localized pain and swelling with calcification seen on plain radiography. Healing is generally self-limited with progression from light activity at 2–3 months to full activity by 6 months [6]. A Morel-Levallee lesion is a degloving injury between the hypodermis and fascia that is caused by shearing forces [7]. Morel-Levallee lesions are discussed elsewhere in the book. One of the most severe soft tissue injuries that could present in the thigh is compartment syndrome. Due to the large volume the upper leg can hold, compartment syndrome of the upper leg is less common than that of the lower leg. However, this diagnosis would be an emergency and needs to be on the differential diagnosis in the setting of trauma leading to upper leg pain and swelling [2].

Fractures

This chapter will focus on trochanteric region and diaphyseal fractures. Proximal and distal intra-articular fractures are discussed elsewhere in the book.

Trochanteric Region

Mechanism of Injury in Sports

Trochanteric femur fractures are uncommon in young, healthy athletes as they require high-energy mechanisms to fracture the intertrochanteric region. The mechanism usually includes a fall directly onto the greater trochanter [1]. Therefore, they are most likely seen in sports with falls from heights or high-velocity collisions such as winter sports, climbing, and motor sports [3].

Epidemiology

Just under half of hip fractures occur in the trochanteric region [8]. The incidence of intertrochanteric fractures increases with age, and women over the age of 60 years are the most prone to these injuries due to the increased risk of osteoporosis [1, 3, 9]. The decrease in trabeculae predisposes individuals with osteoporosis to hip fracture by weakening the inherent bone structure [1, 2]. Athletes can also develop osteoporosis secondary to other medical conditions such as celiac disease and relative energy deficiency in sports, formerly known as female athlete triad, leading to decreased bone strength in a younger population [10]. Trochanteric and femur neck fractures are otherwise less common in young, healthy athletes [3, 10].

Classification

The OA classification categorizes trochanteric femur fractures by location and pattern of the fracture [11]. The trochanteric region lies distal to the level of the superior edges of the trochanters and proximal to the distal border of the lesser trochanter (Fig. 23.1) [12]. The fractures are further characterized by simple and fragmented fractures (Fig. 23.1). The classification further subcategorizes by the type of fracture and which trochanters and walls are involved [3]. Fractures can also be classified as stable versus unstable. Unstable fractures are comminuted, reverse oblique, involving the lateral wall, or extending into the subtrochanteric region [8, 12, 13].

Clinical Presentation

Athletes with femur fractures will likely be unable to bear weight, with the exception of an avulsion fracture [1, 3, 10]. There is usually a history of a high-velocity injury. The injured leg will be flexed proximally, externally rotated, and abducted [3]. Due to proximal flexion and distal extension, the leg may appear shortened and the athlete's leg may become so externally rotated that the lateral foot will be in contact with the exam table [3]. On physical examination, bony tenderness and occasionally crepitus are present [3]. Internal and external range of motion will be painful [3, 10]. Other possible findings that might indicate fracture include pain with a heel strike and inability to raise the leg at the hip [3, 10]. Trochanteric region fractures due to high-impact trauma are rarely isolated injuries (Fig. 23.2). The athlete should be evaluated for other bony and soft tissue injuries as up to 75% of young, healthy athletes will have other associated injuries [2, 3]. Avulsion fractures are seen most commonly in teenaged athletes after a rapid and strong change in direction or as a result of a forceful kick (Chap. 22; Fig. 22.11). Avulsion can happen at the apophyses of the pelvis and presents as instantaneous pain with a "pop" sound [2].

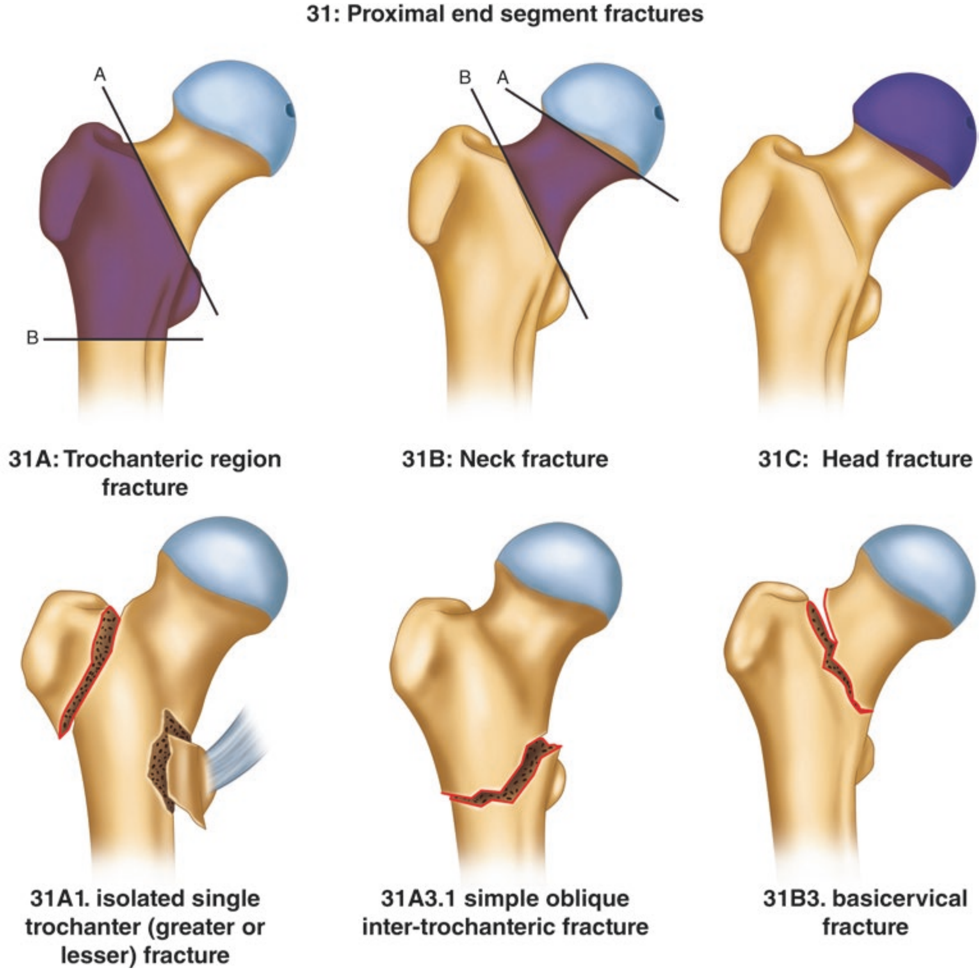


Fig. 23.1 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for proximal femur fractures

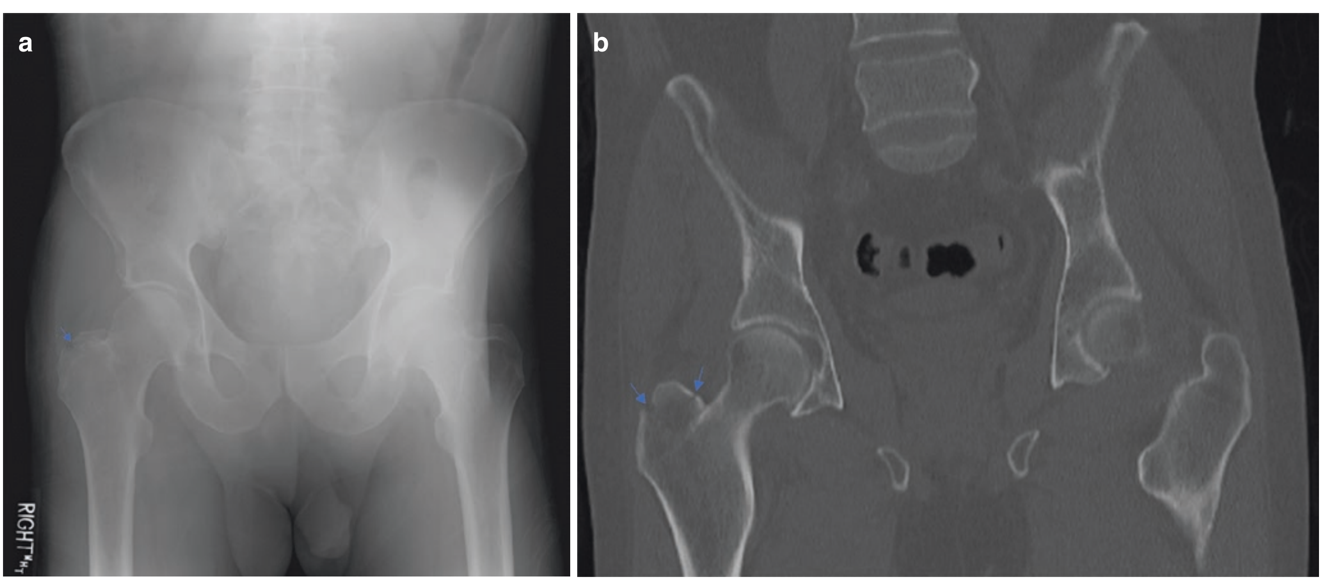


Fig. 23.2 Right greater trochanter fracture (arrows) in a 52-year-old male as the result of a fall (**a**). CT image confirms the diagnosis (**b**)

Diagnosis

If fracture is suspected, the clinician should obtain plain radiographs of the femur in at least two views (Figs. 23.2, 23.3, 23.4, and 23.5). Often, additional views such as oblique are required to evaluate the extent of the fracture. If the plain radiograph is unrevealing, a CT scan and MRI are other reasonable options (Fig. 23.2) [1, 3, 14].

Initial Management

The injured athlete should also be evaluated for serious and life-threatening injuries such as abdominal and pelvic injuries. Early stabilization is important for morbidity and mortality, and early reduction and compression can help prevent short- and long-term complications [3]. As significant hemorrhage may occur, the patient should be monitored for hemodynamic status. Unless there is an isolated avulsion fracture, the athlete should be stabilized and transported to a higher-care facility.

Indications for Orthopedic Referral

In the absence of an isolated avulsion fracture, all individuals with a proximal femur fracture should be evaluated by an orthopedic surgeon as soon as possible [1, 3, 10]. Almost all proximal

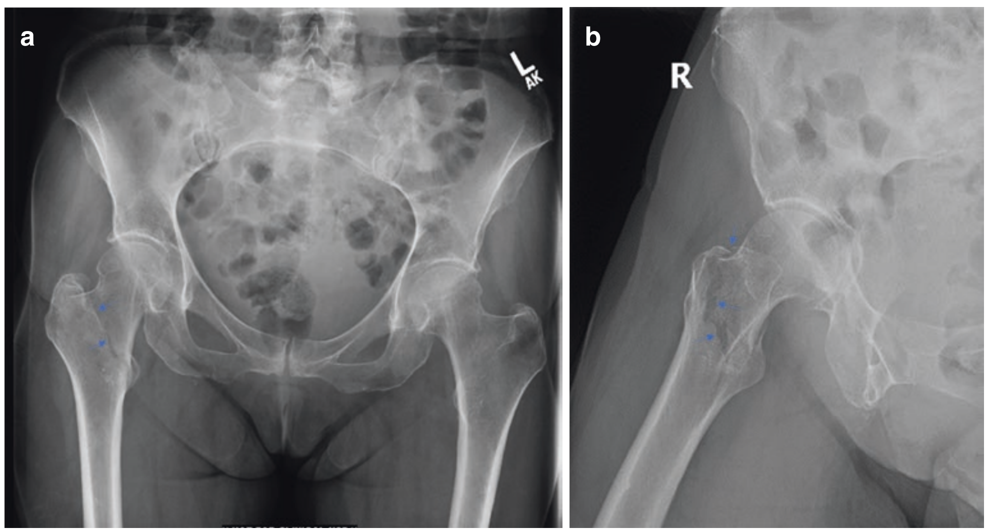


Fig. 23.3 Right intertrochanteric fracture (arrows) in a 72-year-old female as the result of a fall (**a**, **b**)

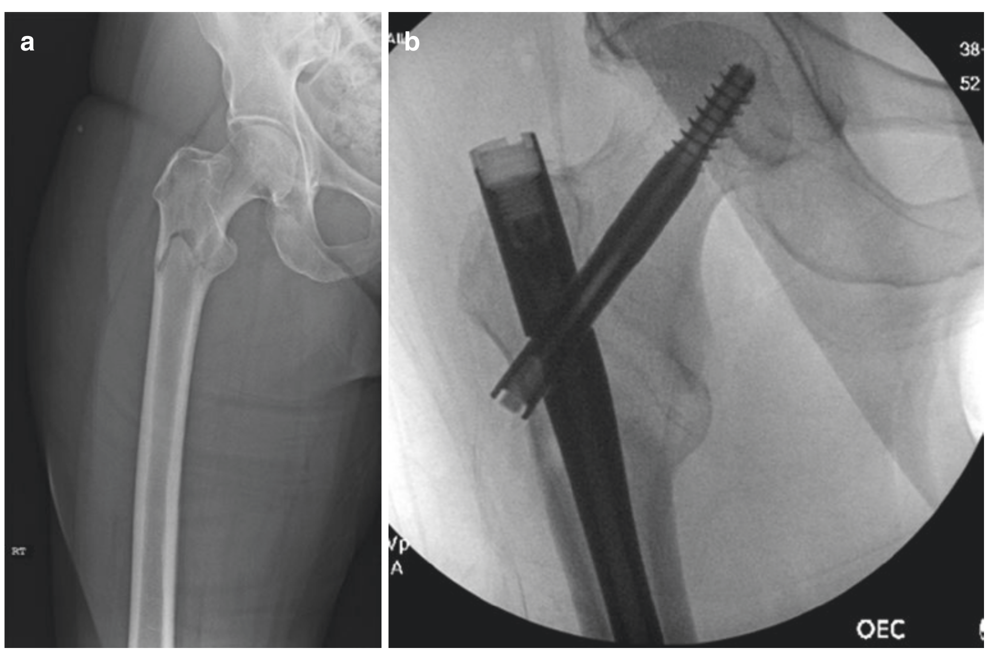


Fig. 23.4 Right intertrochanteric fractures in a 67-year-old female as the result of a ski injury (**a**). Intraoperative fluoroscopy reveals near-anatomic alignment (**b**)

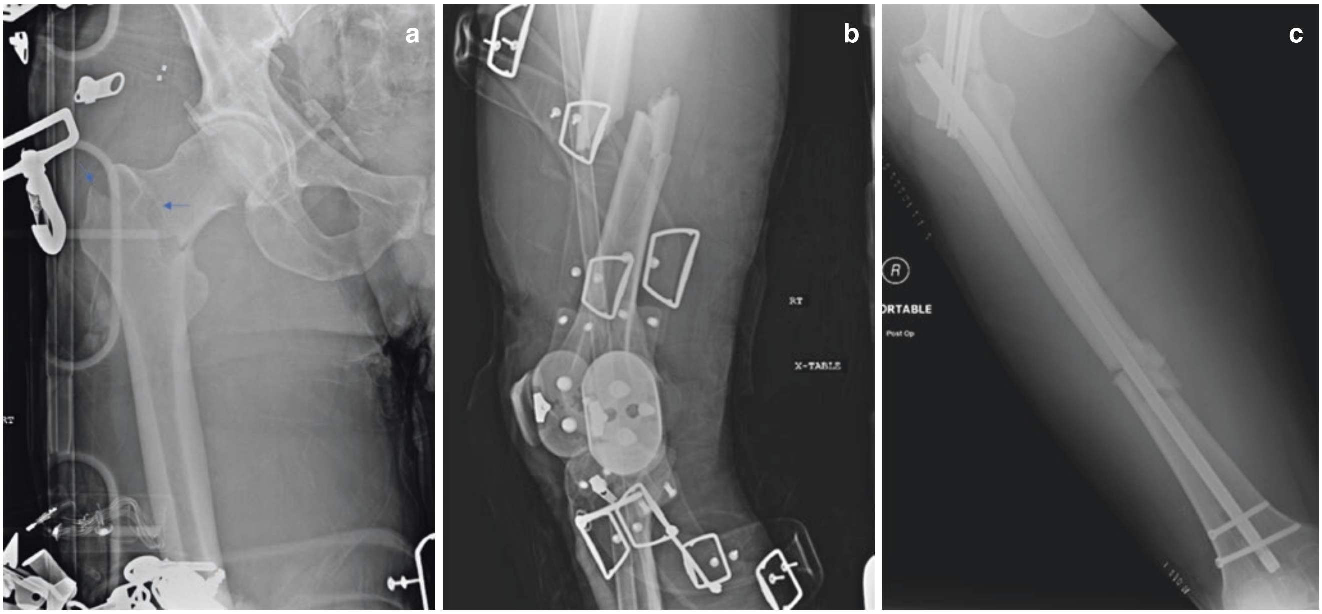


Fig. 23.5 Right ipsilateral intertrochanteric (arrows) and diaphyseal femur fractures in a 50-year-old male as the result of a ski injury (**a**, **b**). Postoperative image shows near-anatomic alignment (**c**)

femur fractures require surgery to ensure stability and healing [1, 3, 10, 13]. Surgical management is typically accomplished through internal fixation (Figs. 23.4, and 23.5), while, depending on the stability of the fracture, some athletes are treated with a sliding hip screw [12]. Low-trauma fractures such as those in the elderly are time-sensitive, while high-energy fractures are more important to stabilize prior to surgery [1, 3, 10, 13]. Surgery within 24 hours has been shown to improve mortality [9, 10, 15]. Avulsion fractures can often be treated conservatively, but surgical consultation is recommended for displaced fractures that have shifted more than 2 cm [2].

Follow-Up Care

The athlete should weight-bear as tolerated and begin to ambulate with a walker or crutches as soon as other comorbidities are stable [3]. The athlete should continue to progressively bear weight as tolerated with patient-specific restrictions. The injury should be reevaluated at 2 weeks postoperatively, and each athlete should have follow-up evaluation and radiographs at 6 and 12 weeks to ensure appropriate union and healing [3]. Physical therapy and rehabilitation programs with early mobilization are essential elements of follow-up care [16].

Return to Sports

There is little data regarding return to sport after a trochanteric fracture in athletes. There is more information in the literature referencing elderly patients and trochanteric fractures, and about one third of these individuals are able to function at the level prior to their injury [3]. Non-geriatric patients have been found to be at their pre-injury activity level approximately 1 year after injury, but there is no athlete-specific data [17].

Complications

Complications of a trochanteric fracture include avascular necrosis of the head of the femur, joint instability, nonunion, malunion, fixation failure of a hip screw, and need for repeat surgery with potential long-term side effects from limb shortening or decreased range of motion after surgical fixation [1, 3, 17]. Fortunately, due to the location of the trochanteric region in relation to the vasculature of the hip, these types of fractures cause less avascular necrosis in comparison to other more proximal femur factures [1].

Pediatric Considerations

The long bones of pediatric athletes continue to ossify into adolescence, and femurs can have open metaphases up until age 14 years in females and age 16 years in males [18]. As in non-elderly adults, neck and trochanteric region fractures are not common and often the result of high-velocity injuries (Figs. 23.6, and 23.7). For this reason, most pediatric trochanteric region fractures are not isolated injuries. Children with nondisplaced fractures may have a limp with decreased range of motion at the hip, and those with dis-

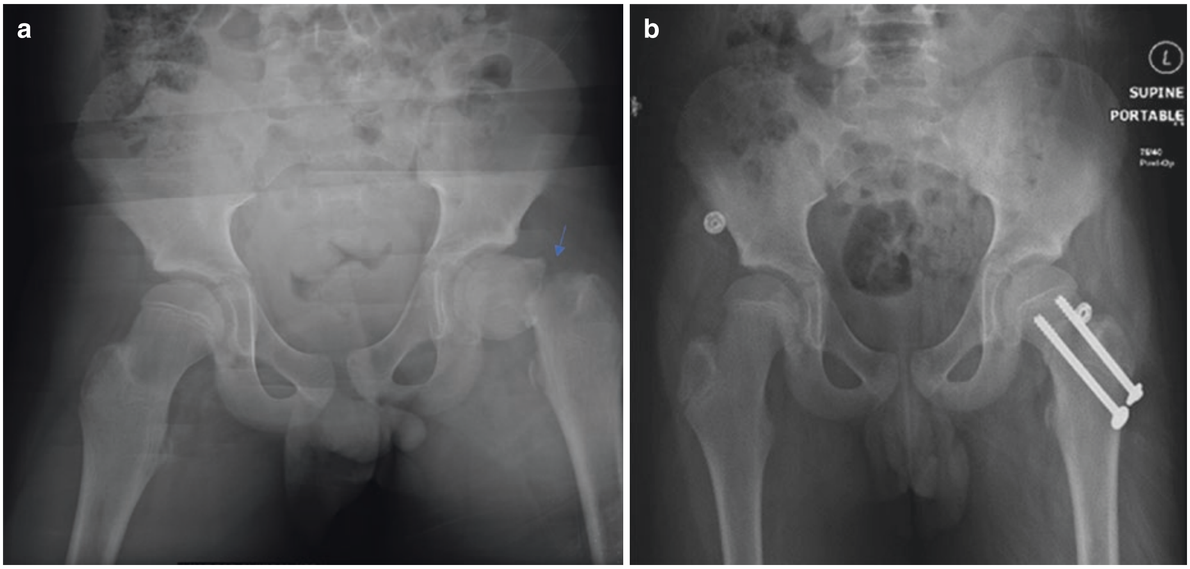
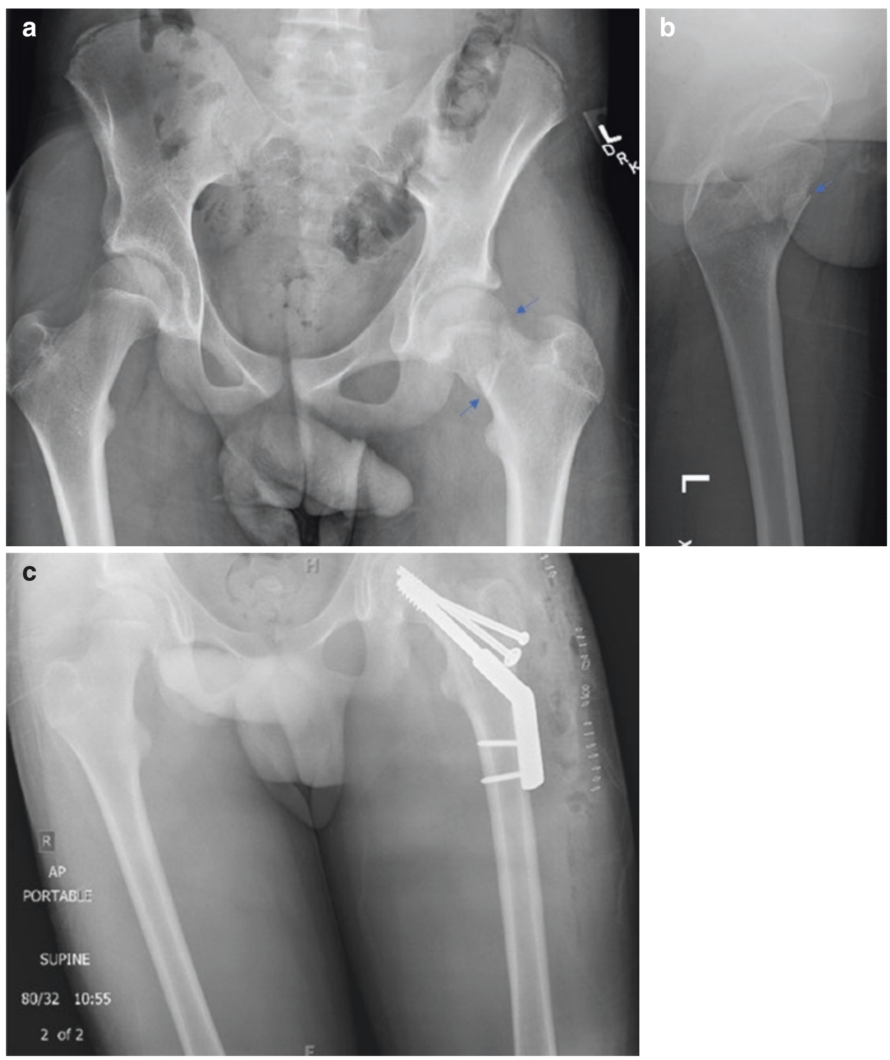


Fig. 23.6 Left displaced and angulated femoral neck fracture (arrow) in a 13-year-old boy as the result of a snowboard injury (**a**). Postoperative image shows near-anatomic alignment (**b**)

Fig. 23.7 Left femoral neck fracture (arrows) in a 16-year-old male as the result of a mountain bike injury (**a**, **b**). Postoperative image shows near-anatomic alignment (**c**)



placed fractures often will not tolerate weight-bearing with the affected leg appearing shortened in external rotation [18]. Treatment of displaced fractures and fractures in individuals older than 8 years old is surgical in nature (Figs. 23.6, and 23.7), while nondisplaced fractures in athletes less than 8 years old can be managed with cast immobilization [18, 19]. Reduction and stabilization with immobilization within 24 hours are recommended. Possible complications of pediatric intertrochanteric fractures include osteonecrosis; similar to adults, this is less common in the intertrochanteric region in comparison to the other areas of the hip due to vascularity [18, 19]. Other complications include nonunion, chondrolysis, infection, as well as extension into the metaphases which can lead to growth discrepancies [18, 19]. Due to the possible long-term effects of the potential complications, proximal femur fractures in pediatric athletes, although rare, can have significant long-term effects [19].

Diaphysis

Mechanism of Injury in Sports

High-velocity trauma causes this type of fracture in younger populations [4, 20–23]. These types of injuries are usually associated with motor vehicle accidents or injuries from high-impact sports such as skiing, climbing, football, and ice hockey [2, 4, 23, 24]. The risk of fracture increases with age and osteoporosis [4]. Due to the mechanism of femur shaft fractures, the fracture is likely to be transverse and may be associated with concurrent injuries [2, 22, 23].

Epidemiology

Diaphyseal fractures are uncommon injuries with an incidence of 13/100,000 people and remain rare in sports [23, 24]. Males are slightly more prone to femur fractures than females with an average age of 25 years [22]. Femur fractures after a low-impact injury are typically seen in older adults over the age of 60 or among athletes with relative energy deficiency in sports [22].

Classification

The OA classification for diaphyseal fractures includes three sections between the horizontal transverse line at the inferior border of the lesser trochanter and above the line at the superior border of the condyles [11]. The classification is further subdivided into the type of fracture including simple, wedge, and multifragmentary (Fig. 23.8). Those subdivisions are further classified by the orientation of the fracture [11]. Subtrochanteric fractures (Figs. 23.9, and 23.10) are just distal to the intertrochanteric region [3].

Clinical Presentation

Athletes with a diaphyseal fracture usually present with extreme upper leg pain and possible deformity, leg length discrepancy, decreased range of motion, and ecchymosis. Although rare, patients may also present with open femur shaft fractures (Fig. 23.11). As femur fractures are often associated with other injuries, athletes may present with limb shortening and decreased range of motion of both the hip and the knee [22]. Due to the anatomic makeup of the thigh, an average of greater than 1 liter and up to 3 liters of blood can be lost into the thigh after a femur fracture [2, 24–26].

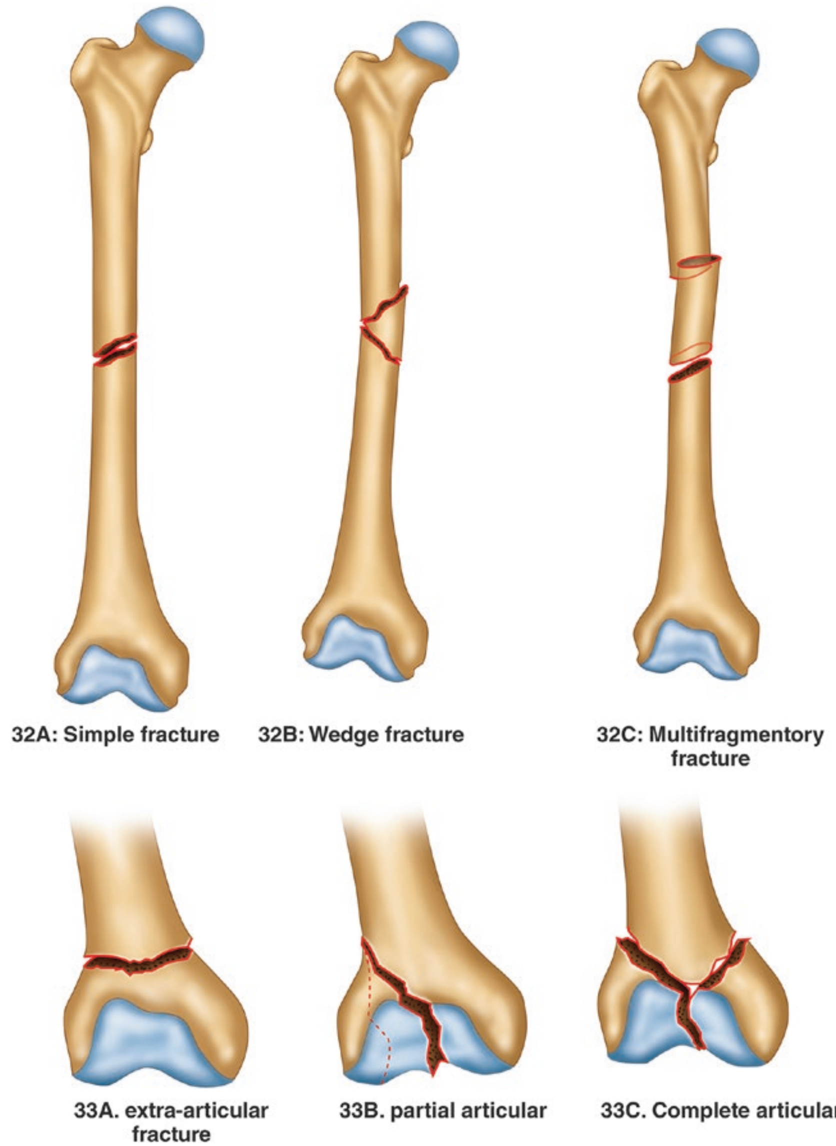
Diagnosis

Femur fracture may be highly suspected or evident on physical examination, but a definitive diagnosis requires imaging [5]. MRI is the most specific imaging test for identifying fractures, but the clinician should start with AP and lateral plain radiographs including the surrounding joints (Figs. 23.12, 23.13, 23.14, and 23.15), which will often be sufficient to diagnose a fracture [2, 26]. With high suspicion for a fracture and negative initial imaging, the clinician should obtain a CT scan. Comminuted fractures (Figs. 23.12, 23.13, and 23.16) or those that involve the lateral wall are thought to be unstable [8]. A high level of clinical suspicion should be considered for other concurrent injuries such as intra-abdominal injuries.

Initial Management

The clinician caring for the athlete with a femur diaphyseal fracture should first follow ATLS protocol and should direct the athlete to emergency care as soon as possible [5, 27, 28]. This should include removing any obstructing clothing, doing a thorough physical examination paying special attention to the neurovascular status, and performing primary and secondary surveys [29]. On the field or in the prehospital setting, splinting and traction (Fig. 23.13) can provide temporary pain relief, immobilize the injury, and decrease hemorrhage [2, 20]. Traction should be avoided in athletes with other bony fractures, unstable knee ligamentous injuries, and open injuries to prevent introduction of exposed, contaminated bone back into the body [2]. Femur fractures have a higher likelihood of neurovascular compromise compared to other fractures in the body [5]. Ensuring hemodynamic stability is paramount as up to 3 liters of blood can be lost into the thigh, predisposing the athlete to hypotension or hemorrhagic shock [2, 29]. The goal of initial management is to stabilize and transport the patient to a hospital as soon as possible. Pain should be managed with opioid medications and a femoral nerve block. The athlete should be taken to

Fig. 23.8 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for diaphyseal and distal femur fractures



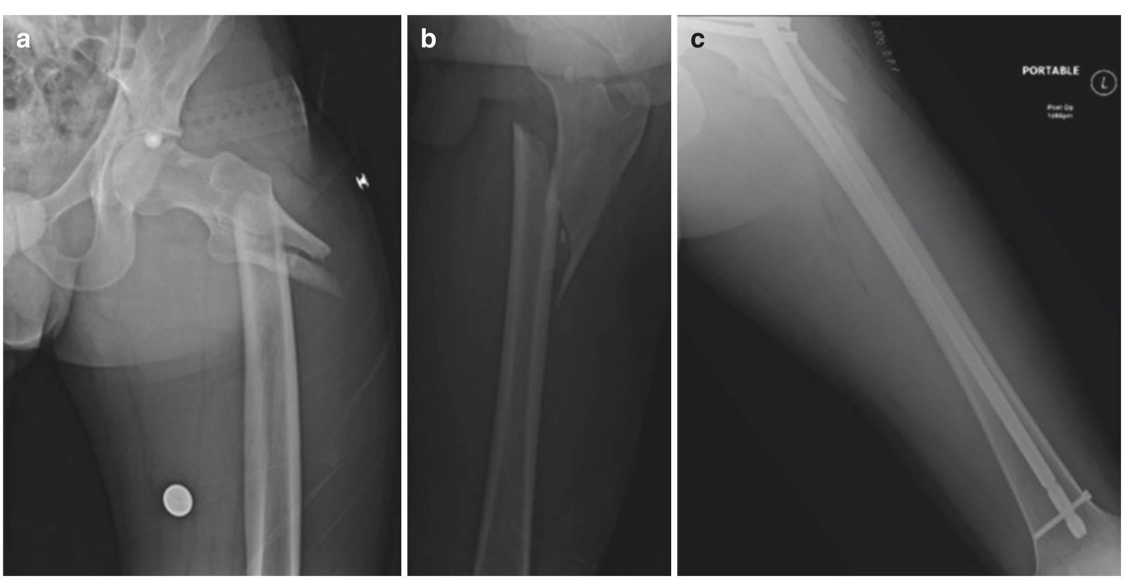


Fig. 23.9 Left displaced and comminuted subtrochanteric diaphyseal femur fracture in a 28-year-old male as the result of a tree strike while skiing (**a**, **b**). Postoperative radiograph shows near-anatomic alignment (**c**)

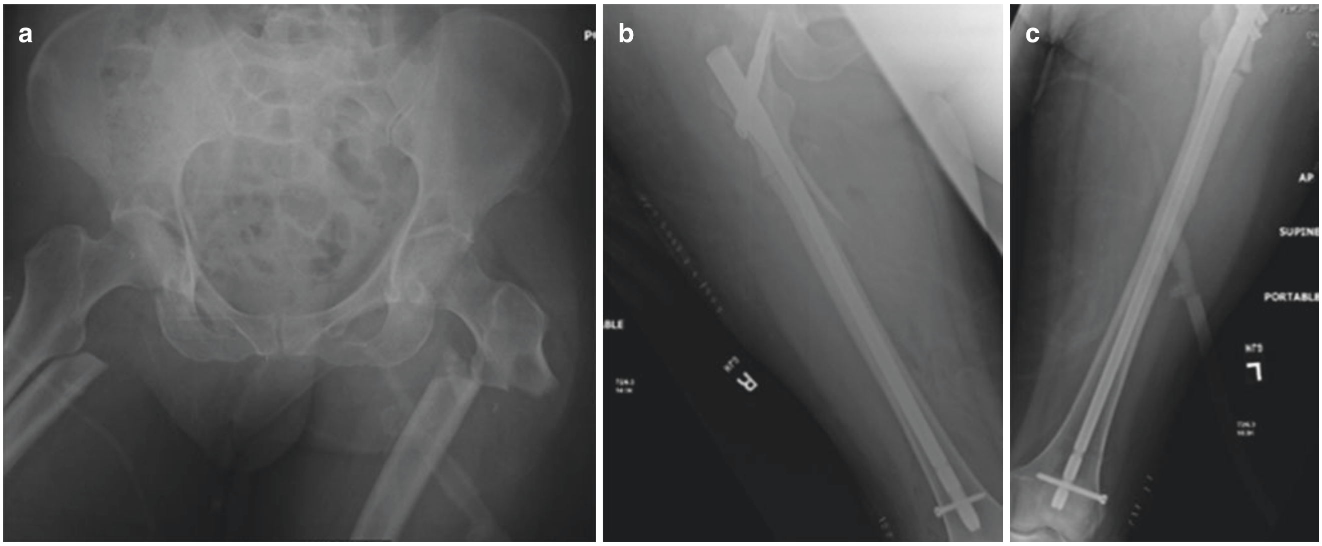


Fig. 23.10 Bilateral displaced and comminuted subtrochanteric diaphyseal femur fractures in a 62-year-old female as the result of a collision with another skier (**a**). Postoperative radiographs show near-anatomic alignment (**b**, **c**)

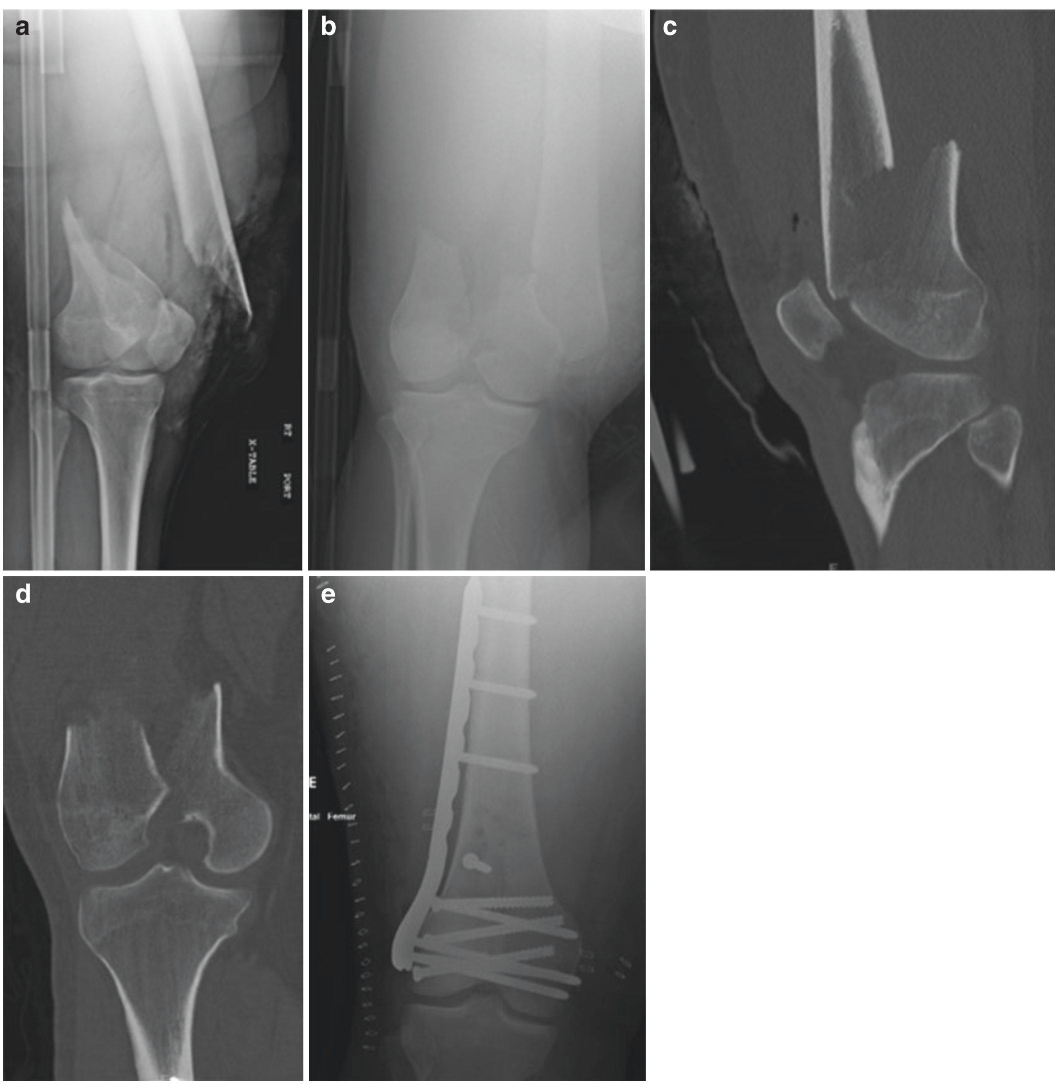


Fig. 23.11 Comminuted, open, intra-articular distal femur and patella fractures in a 23-year-old male as the result of a tree strike while skiing (**a**, **b**). CT scan images demonstrate the extent of the injury (**c**, **d**). Postsurgical plain radiography shows near-anatomic alignment (**e**)

early surgery if indicated [28]. The mortality of femur fractures was previously 80%, but it has decreased dramatically with early intervention and surgery [24].

Indications for Orthopedic Referral

A femur diaphysis fracture is a medical emergency and should have immediate orthopedic referral. If possible, surgery should be performed within 6 hours of the time of the injury as early fixation has been shown to improve morbidity and mortality [8, 24].

Fig. 23.12 Left comminuted diaphyseal femur fractures in a 32-year-old female as the result of a ski injury

Follow-up Care

Athletes with femur fractures can begin partial weight-bearing in the first days after surgery [2]. Complete weight-bearing should not be started for at least 6–8 weeks [2]. Bone healing will take place in 3–6 months. Regular follow-up radiographs should be performed to monitor appropriate healing. As weight-bearing progressively increases with clinical improvement, the athlete should be encouraged to actively heal with progressive increases in range of motion and muscle strengthening [2]. Athletes should not use non-steroidal anti-inflammatory medications for pain control and should follow a healthy lifestyle with a healthy diet [2].

Return to Sports

The majority of patients heal well with internal fixation and are able to return to sports by 6 months [2]. Based on few case reports, professional athletes may return to sports after an isolated femur fracture without any complications within a year. However, athletes may take longer to return to play with any continued pain, weakness, or other residual symptoms [23].

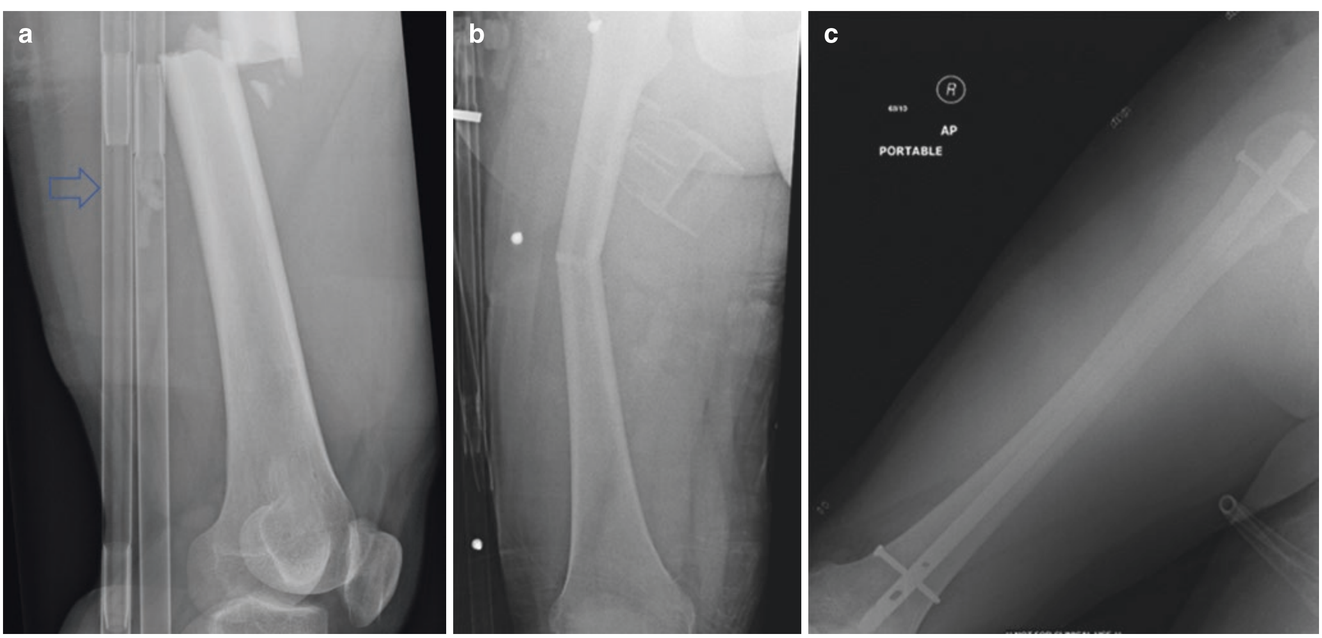


Fig. 23.13 Right comminuted and displaced diaphyseal femur fracture in a 23-year-old male skier as the result of a tree strike (**a**, **b**). An external traction device (open arrow) is in place. Postoperative radiograph demonstrates near-anatomic alignment (**c**)

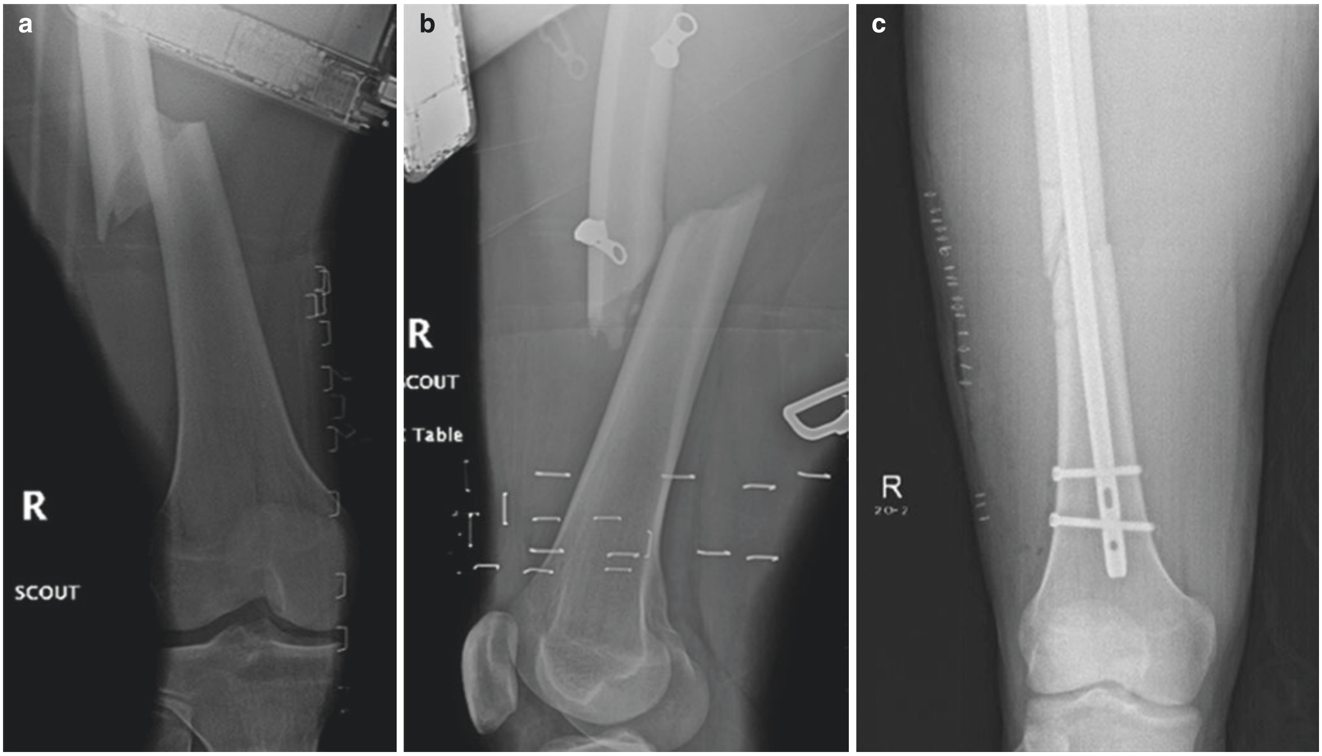


Fig. 23.14 Right displaced diaphyseal femur fracture in a 20-year-old male as the result of a tree strike while snowboarding (**a**, **b**). Postoperative radiograph shows near-anatomic alignment (**c**)

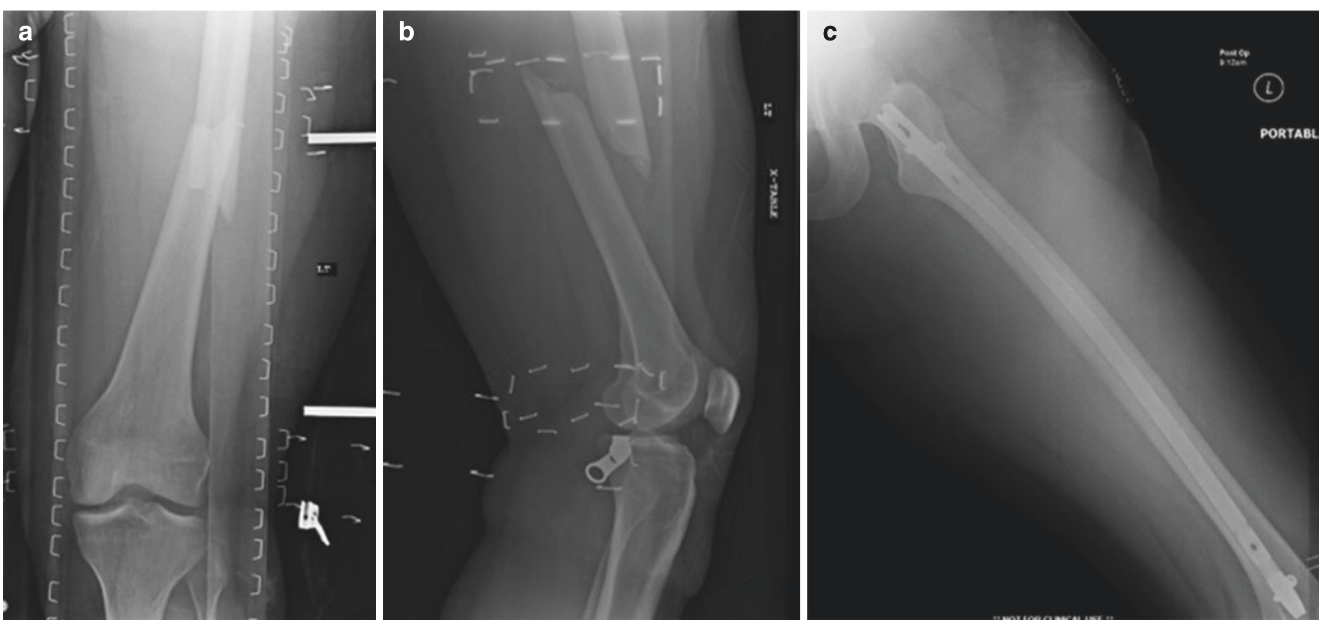


Fig. 23.15 Left displaced diaphyseal femur fracture in a 29-year-old male as the result of a ski injury (**a**, **b**). Postoperative radiograph shows near-anatomic alignment (**c**)

Complications

In the immediate fracture period, diaphyseal fractures can be life- and limb-threatening, as the fracture can be complicated by hemorrhage and fat emboli entering the bloodstream [20]. Athletes may also experience subsequent anterior knee pain, bursitis, and muscle weakness, specifically in hip adductors and quadriceps [23]. Postoperative complications include damage to nearby structures including nerves and vasculature, infection, malalignment, nonunion, procedure failure, and hematoma [24].

Pediatric Consideration

Although uncommon in sports, pediatric femur diaphysis fractures make up a large proportion of traumatic orthopedic operations [30]. Because of the hemodynamic reserve of pediatric athletes, hemorrhagic shock may present as a later and more severe finding than in adults [29]. Pediatric femur diaphysis fractures (Figs. 23.17, 23.18, 23.19, and 23.20) should otherwise be managed similarly to adults [31]. Pediatric patients presenting with a fracture should be evaluated for non-accidental trauma as well as other causes of pathologic fracture [2, 31].

Fig. 23.17 Left subtrochanteric diaphyseal femur fracture in a 13-year-old boy as the result of a tree strike while skiing. Significant fracture pieces overlap and femoral shortening is noticeable

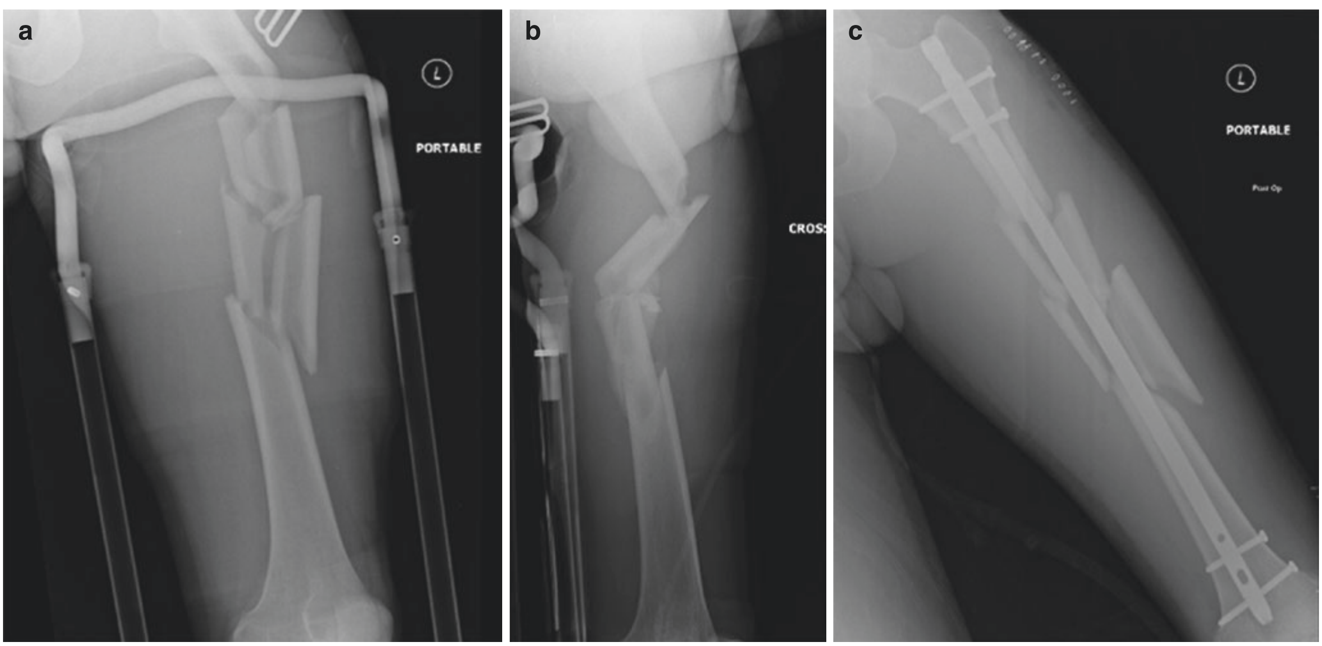


Fig. 23.16 Left comminuted and displaced diaphyseal femur fracture in a 24-year-old male as the result of a car accident (**a**, **b**). Postoperative radiograph shows near-anatomic alignment (**c**)

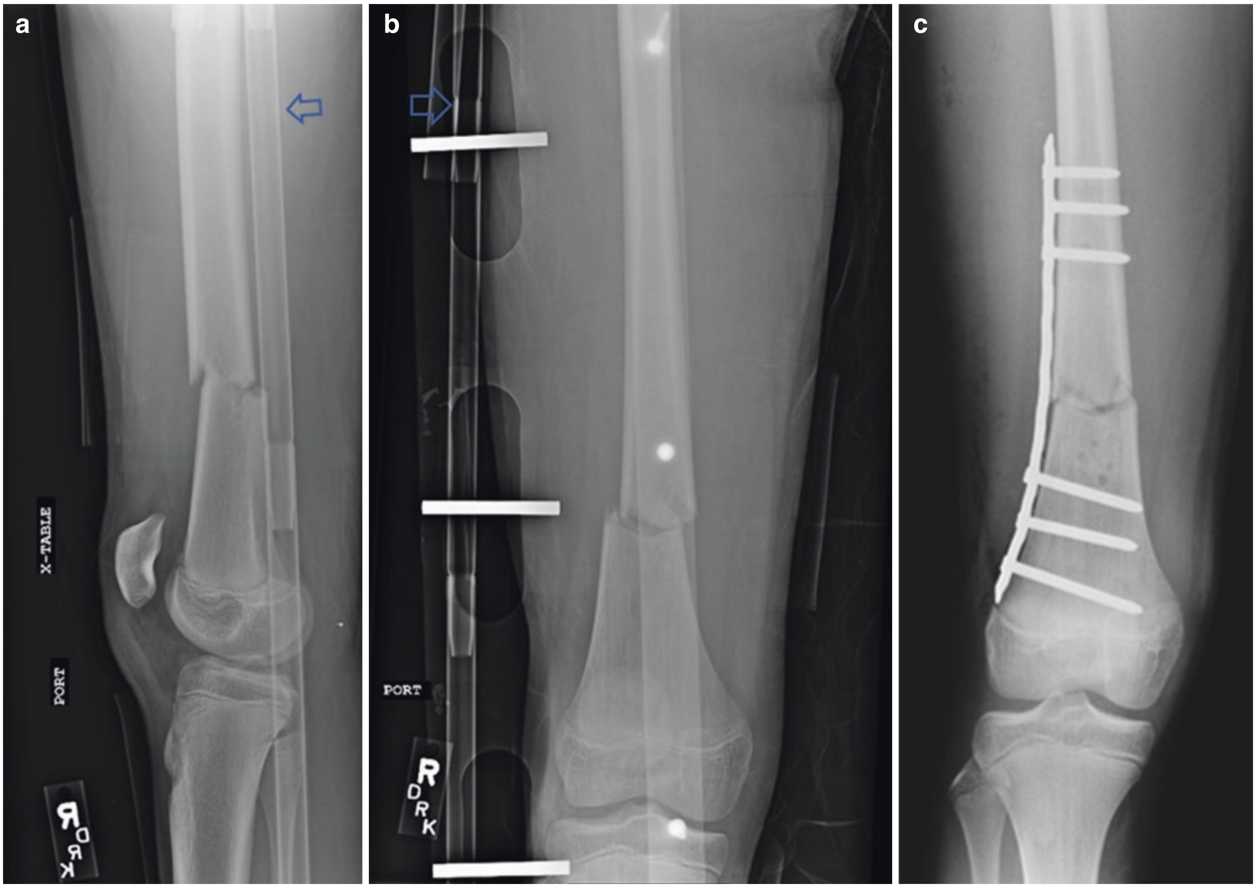


Fig. 23.18 Right transverse distal diaphyseal femur fracture in a 13-year-old boy as the result of a ski injury (**a**, **b**). An external traction device is in place (open arrows). Postoperative radiograph shows near-anatomic alignment (**c**)

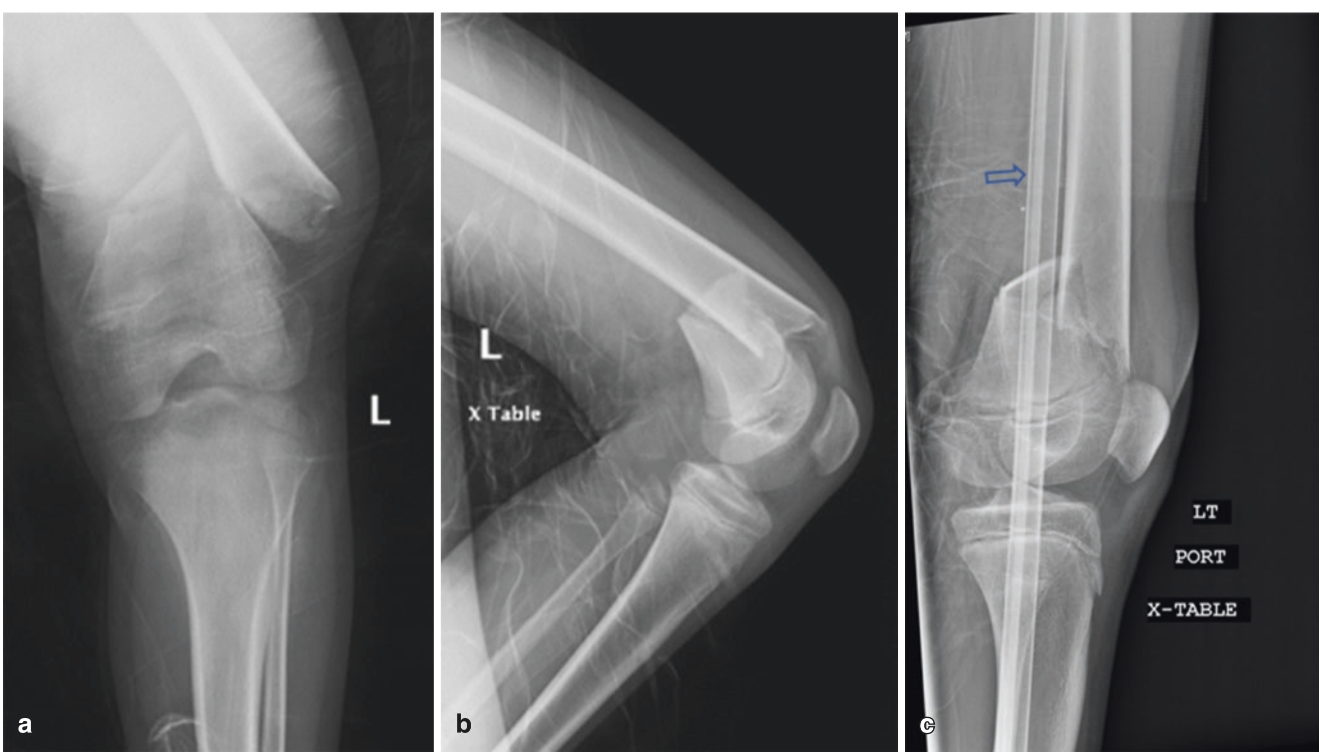


Fig. 23.19 Left displaced distal diaphyseal femur fracture in a 13-year-old boy as the result of a snow tubing injury (**a**, **b**). An external traction device (open arrows) was placed (**c**, **d**). Intraoperative fluoroscopy shows near-anatomic alignment (**e**)

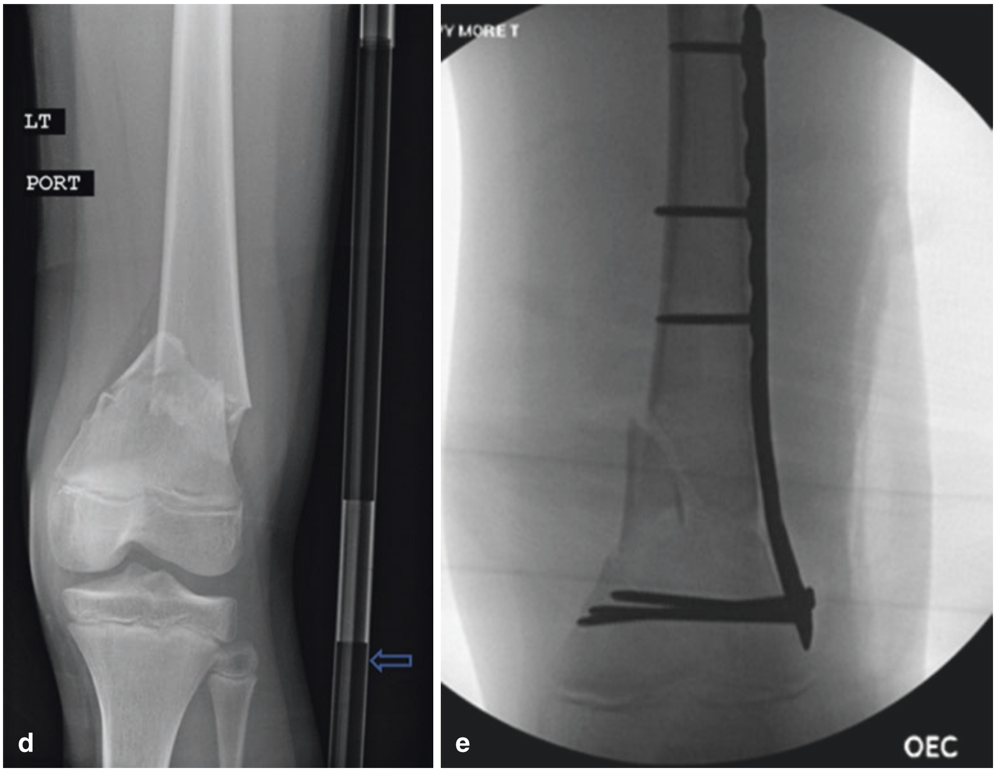


Fig. 23.19 (continued)

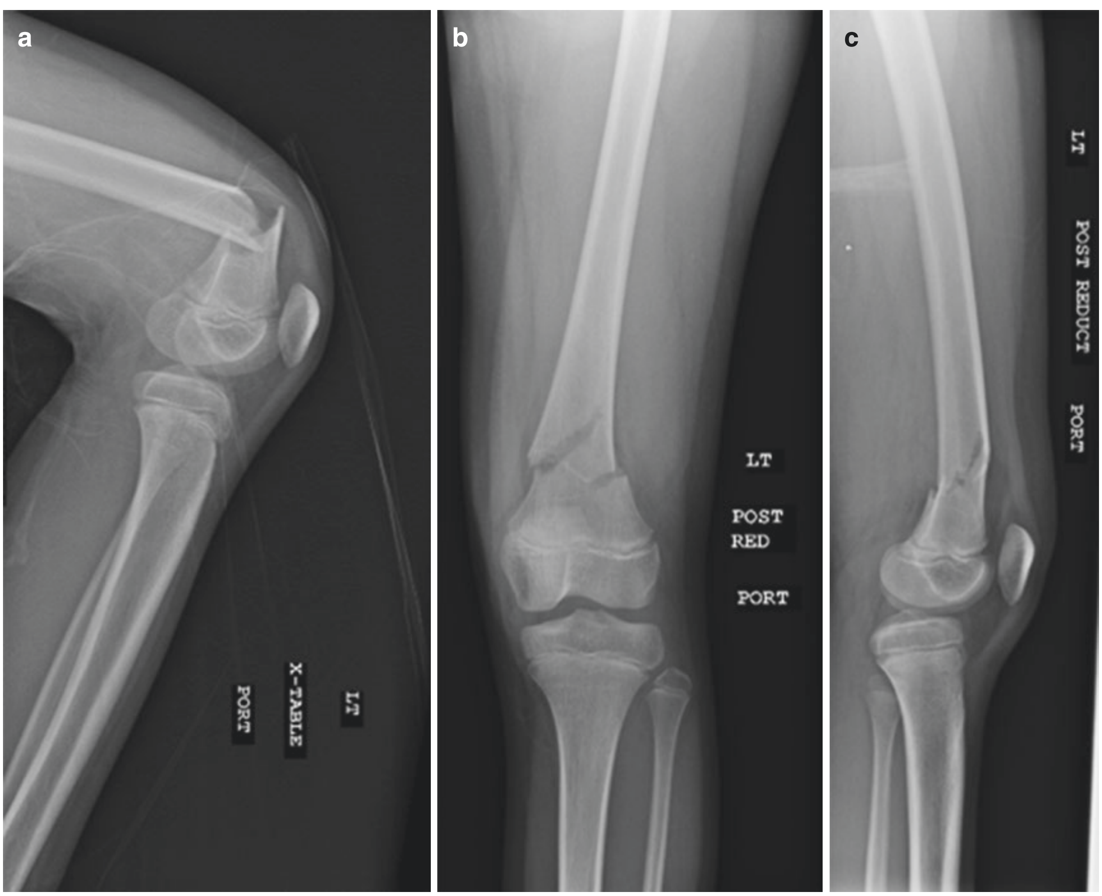


Fig. 23.20 Left displaced and angulated distal diaphyseal femur fracture in a 7-year-old girl as the result of a ski injury (**a**). Postreduction images show improved alignment (**b**, **c**). Intraoperative fluoroscopy reveals near-anatomic alignment (**d**)

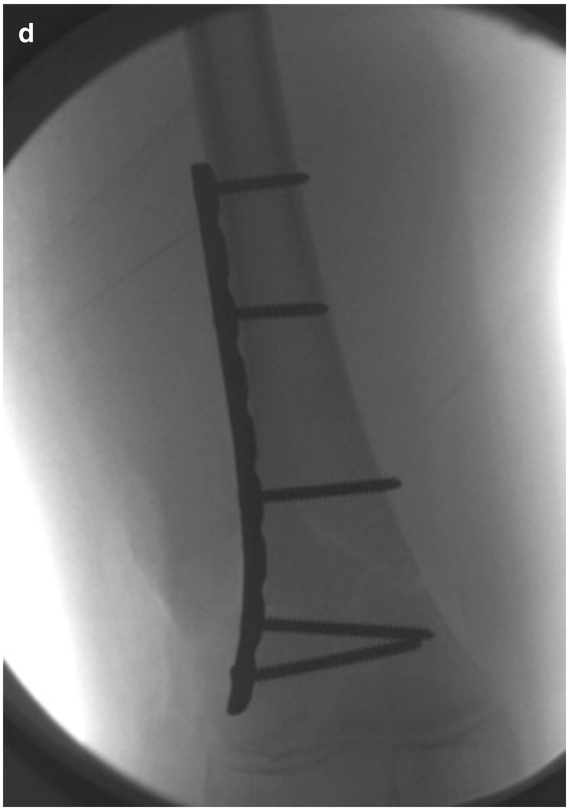


Fig. 23.20 (continued)

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Knee

24

Geoffrey M. Dreher, Utsav Hanspal, David M. Baxter,
and Morteza Khodaee

Key Points

- Knee injuries are common in sports.
- Soft tissues, articular surfaces, and bony structures can be involved in acute knee injuries.
- Many knee injuries in sports require surgeries with long-term for recovery.
- Athletes should not be allowed to return to sport if they still have effusion, limited range of motion, and strength deficits.

Introduction

Knee injuries are very common in sports. Injuries can happen to the soft tissues, articular surfaces, tendons, ligaments, and bony structures. The exact incidence of specific knee injuries in different sports is unknown, but there are risk factors associated with most of these injuries. Soft tissue, ligament (e.g., ACL, PCL, MCL, LCL), and tendon (e.g., patellar and quadriceps) injuries are discussed elsewhere in the book. This chapter details articular cartilage and meniscal injuries as well as fractures and dislocations around the knee.

G. M. Dreher
Department of Sports Medicine, Crozer-Keystone Health Network, Springfield, PA, USA

U. Hanspal
Department of Orthopedics and Sports Medicine, Lourdes Hospital, Binghamton, NY, USA

D. M. Baxter
Department of Sports Medicine, Healthplex Sports Medicine, Springfield, PA, USA

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Anatomy

The knee complex is made up of three joints: tibiofemoral, patellofemoral, and proximal tibiofibular. The tibiofemoral is the largest joint in the body. The knee joint is a complex structure consisting of bones, cartilage, menisci, muscles, tendons, bursae, and ligaments. As a hinge joint, the knee has a limited and unidirectional range of motion (about 0° to 135°), which allows for good stability. Figure 24.1 demonstrates major anatomical structures of the knee.

Soft Tissue Injuries

Soft Tissue Contusion

Knee soft tissue contusion is common in a variety of sports. The exact incidence is unknown as most athletes do not seek medical attention.

Penetrating knee joint injuries are uncommon in sports. However, they may happen in high-speed sports such as skiing (Fig. 24.2). In these cases, an urgent referral to a hospital with the possibility of immediate surgery is recommended. Prophylactic antibiotics and tetanus immunization status review are essential prehospital actions.

Contusion, ecchymosis, hemorrhagic superficial bursitis, and Morel-Lavallée lesions are common. Managements of these injuries are discussed elsewhere in the book.

Acute Articular Injuries

Knee articular injuries consist of a variety of pathologies which have been described for decades. With the addition of more advanced imaging modalities such as MRI and arthroscopy, our understanding and eventually management of these lesions have evolved significantly. There is no universally

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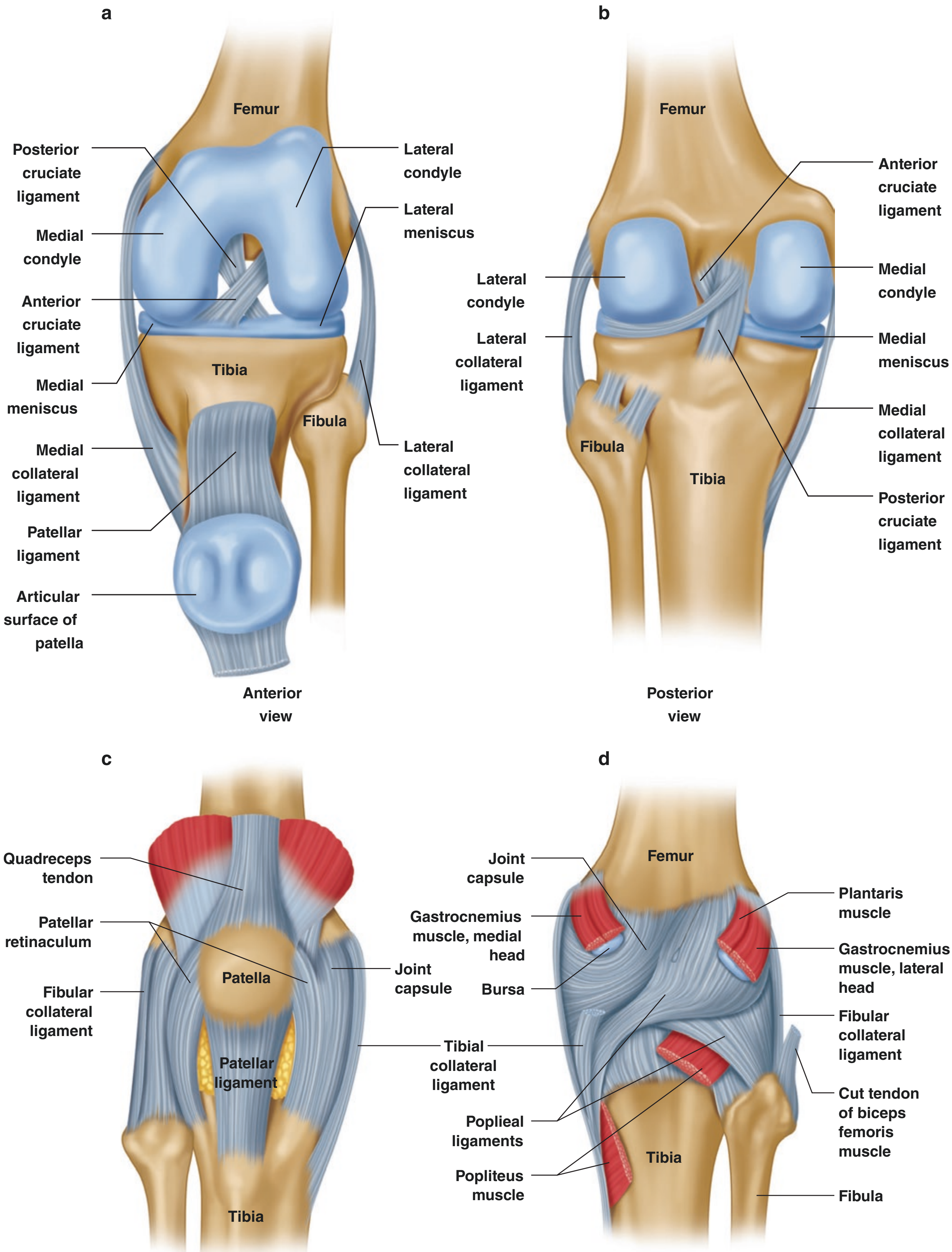


Fig. 24.1 Knee anatomy. Anterior (**a**) and posterior (**b**) articulations. Anterior (**c**) and posterior (**d**) superficial ligaments and tendons attachments

Fig. 24.2 A penetrating lateral knee injury (tree branch) in a 23-year-old male as a result of a tree strike while skiing (**a**, **b**). Plain radiography images (**c**, **d**) show intra-articular air (arrows) in addition to an opacity consistent with the foreign object (open arrows). The branch was removed (**e**) and the wound was tracked to the joint capsule (**f**)

accepted category of these lesions, and there are overlaps among different classifications [1]. These lesions are generally divided into bone bruises, chondral, subchondral, and osteochondral fractures [2]. In general, deeper articular tissue layers are involved in acute osteochondral lesions (e.g., osteochondritis dissecans) compared to chondral lesions. Due to the similarity in the mechanism of injury, diagnosis, and initial management of these lesions, this section will discuss them together.

Mechanism of Injury in Sports

Acute knee articular injuries occur in the setting of a traumatic injury with shear or rotational forces, direct blunt trauma, and patellar dislocation [3, 4]. The most commonly affected area is the medial femoral condyle. Other regions commonly affected include the patellar facets and lateral femoral condyle, as seen in lateral patellar dislocations, and the femoral trochlea. The articular cartilage of the proximal tibial surface has overlying menisci and is much less commonly injured [4].

Epidemiology

The three compartments of the knee (i.e., patellofemoral, tibiofemoral medial, and lateral) are all covered by hyaline cartilage. Hyaline joint cartilage consists of cells, water, and matrix which make it an elastic dense connective tissue. The function of this tissue is shock absorption and to protect the subchondral bone beneath it. A retrospective study of more than 25,000 arthroscopies, which included articular cartilage defects, found that grade II Outerbridge (Fig. 24.3) lesions were the most common [5]. They also found that medial meniscal injuries were

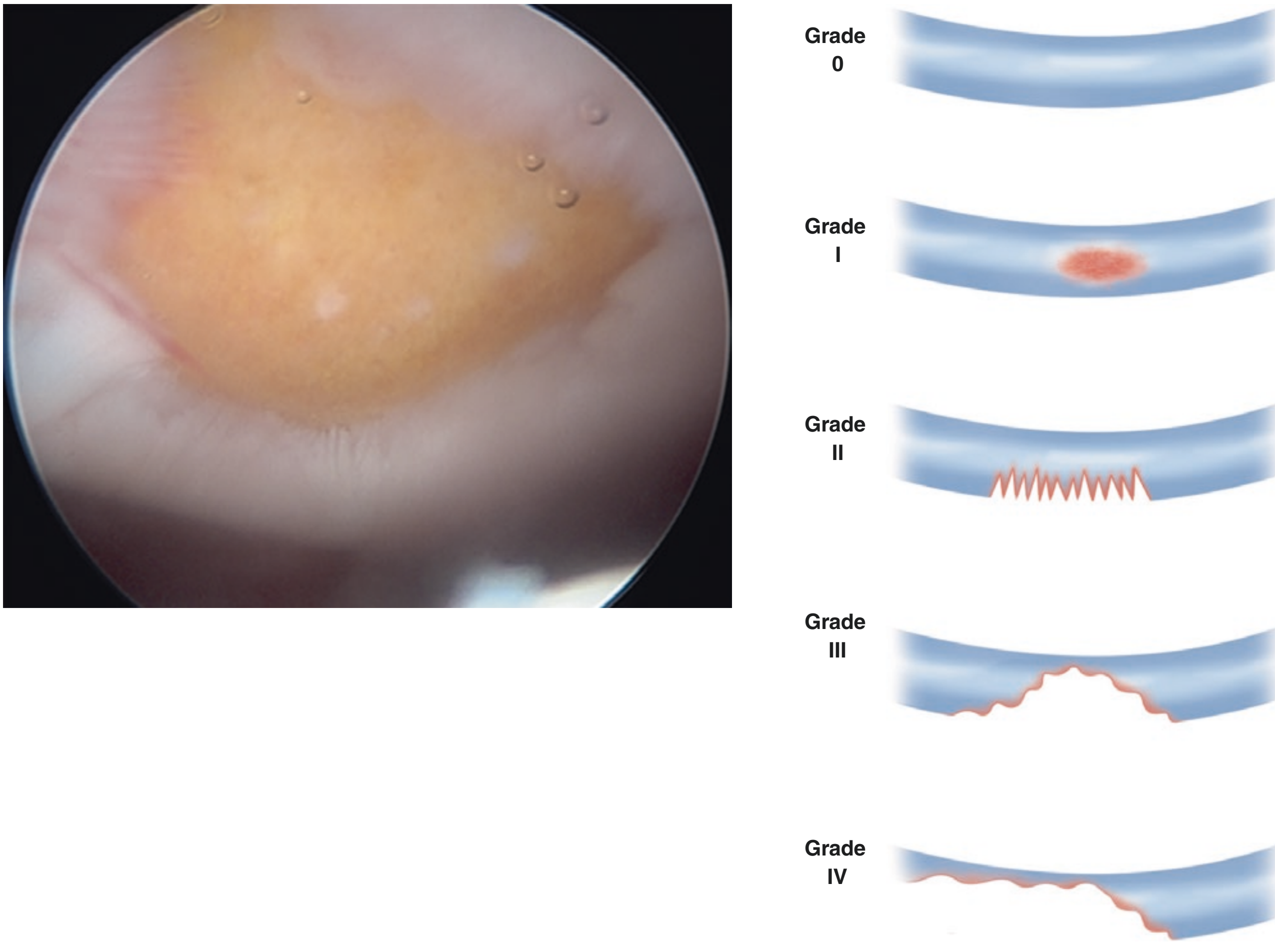


Fig. 24.3 Outerbridge classification (5) of chondral lesions (**a**): grade (0) normal cartilage; grade (I) softening and swelling of cartilage; grade (II) partial-thickness defect with surface fissures extending to <50% of cartilage depth; grade (III) partial-thickness loss with focal ulceration, extending >50% of cartilage depth; grade (IV) full-thickness chondral defect with exposed subchondral bone (**b**)

the most common concomitant injury associated with chondral defects [5]. This study consists of a heterogeneous group and did not delineate acute traumatic from subacute injuries [6]. In the setting of an acute ACL injury with associated chondral defect, there does not appear to be a significant gender predisposition to this injury [7]. Articular cartilage defects affecting the femoral condyles have been observed in up to half of all athletes undergoing ACL reconstruction [8]. In the setting of acute patellar dislocation, the occurrence of concomitant acute chondral defect has been found to be quite high. Nomura et al. found that following an acute traumatic lateral patellar dislocation, 95% of cases had a chondral defect on arthroscopy [9]. The most common site affected in this study was the medial patellar facet and secondly the lateral femoral condyle [9].

Classification

The two most widely recognized systems for classification of chondral lesions are the Outerbridge classification (Fig. 24.3) and the ICRS system (Fig. 24.4) [10]. Outerbridge classification was originally described for patellar chondromalacia, but over time it has been used for other chondral injuries [11]. ICRS classification was developed to provide greater detail of the specific lesion [10, 12–14] (Fig. 24.5).

Clinical Presentation

Acute articular injuries mimic those of meniscal lesions. In the presence of a traumatic injury, pain with weight bearing, mechanical symptoms such as locking, crepitus, and delayed knee joint effusion are all common findings for acute chondral defects [3]. In the acute on-field setting,

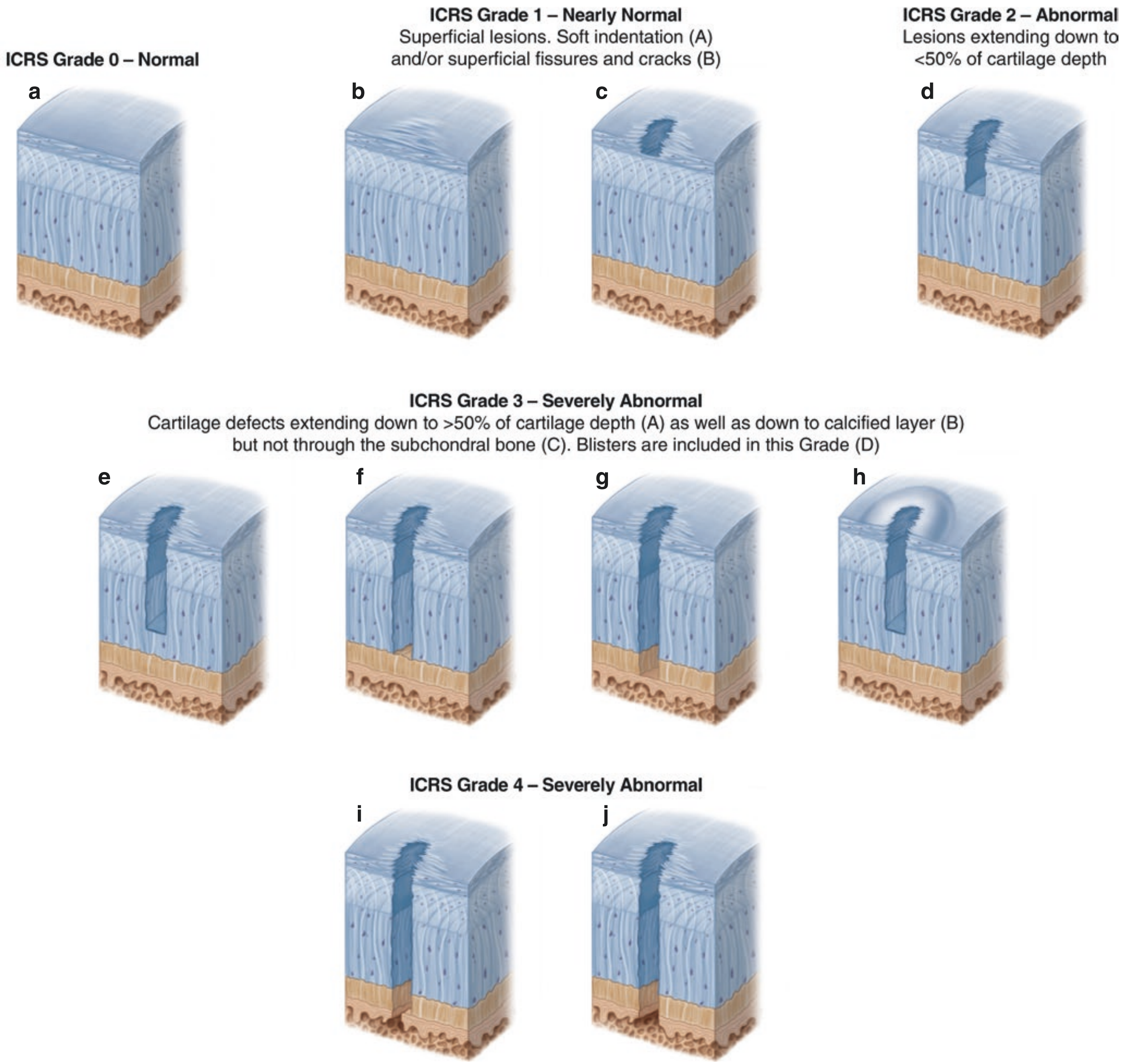


Fig. 24.4 Classification of chondral lesions according to the ICRS system: grade 0) normal cartilage (**a**); grade (1a) superficial lesions/softening (**b**); grade (1b) superficial lesions/softening and/or superficial fissures or cracks (**c**); grade (2) lesion extending <50% of thickness (**d**); grade (3a) lesion extending >50% of thickness (**e**); grade (3b) lesion extending down to the calcified layer (**f**); grade (3c) lesion extending down to the surface of the subchondral bone (without penetration) (**g**); grade (3d) includes bulging of the cartilage around the lesion (**h**); grade (4a) penetration of the subchondral bone but not across the entire diameter of the defect (**i**); grade (4b) penetration across the full diameter of the defect (**j**). (Modified from the ICRS Cartilage Injury Evaluation Package [www.cartilage.org], with permission from the International Cartilage Repair Society [10])

inability to perform full extension is suggestive of a displaced bucket handle meniscal lesion or osteochondral fragment [15].

Diagnosis

In order to establish a diagnosis following an acute knee injury, a combination of clinical history, physical examination, and radiographic studies is essential. Plain radiography should be performed as an initial imaging modality. In addition to standard views (weight-bearing AP, lateral, and sunrise), views such as Rosenberg or tunnel (notch) views should be obtained [8]. Acute cartilaginous lesions are not well visualized on plain radiography. The role of plain radiography in evaluation is to rule out acute fracture and other pathological findings associated with the above symptoms, as in joint space narrowing, subchondral sclerosis, and osteophytes.

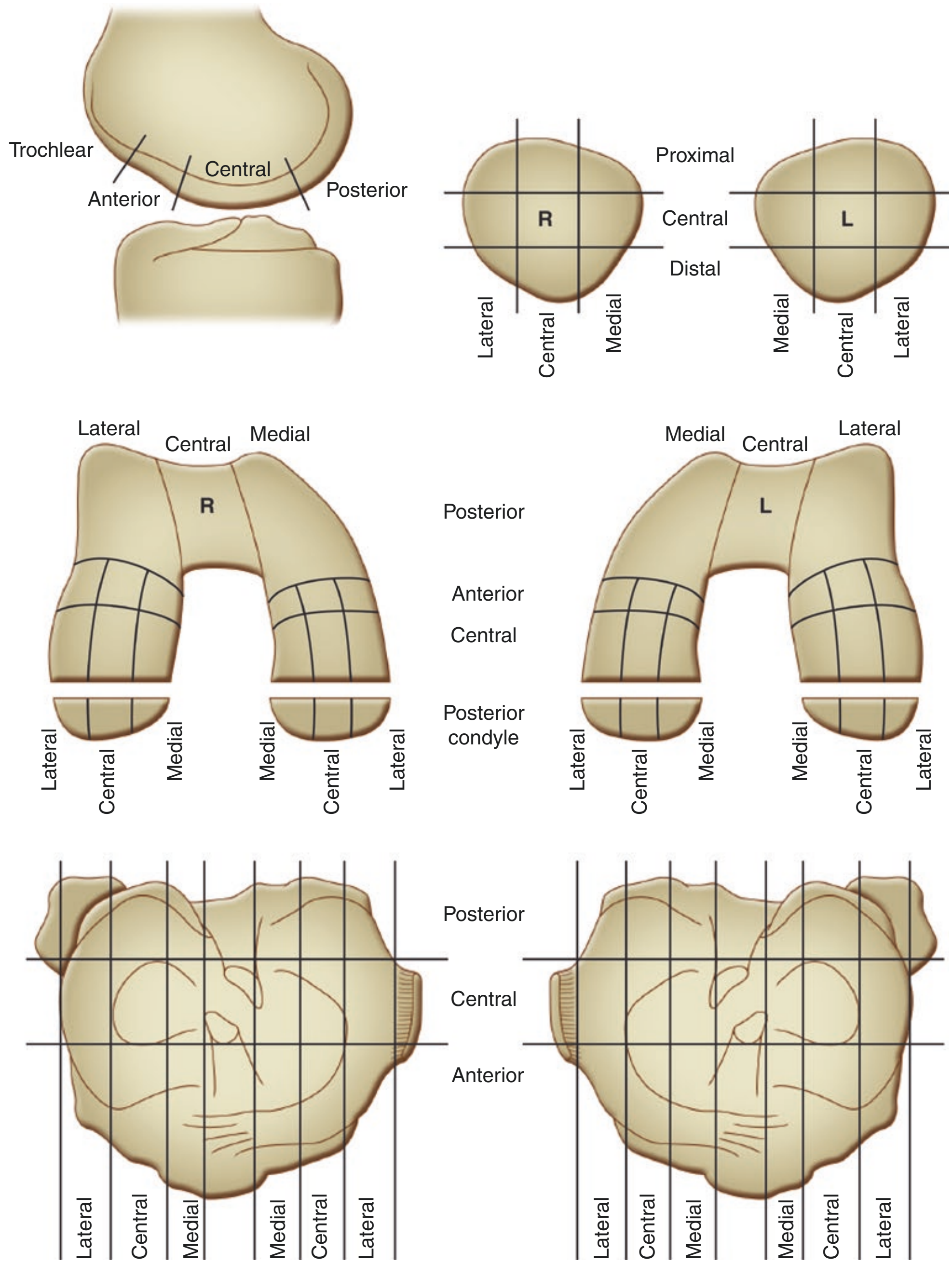


Fig. 24.5 The ICRS knee cartilage lesion mapping system. (Modified from the ICRS Cartilage Injury Evaluation Package [www.cartilage.org], with permission from the International Cartilage Repair Society [10])

Complete chondral/osteochondral lesions with loose bodies in the joint space may be seen on plain radiography. MRI is the imaging modality of choice but may underestimate size of lesion (Fig. 24.6). The gold standard for intra-articular evaluation of pathological conditions of the knee is arthroscopy (Fig. 24.3b) [3, 13].

Initial Management

Initial on-field assessment of acute knee begins with valgus and varus testing at full extension to evaluate for tibiofemoral dislocation [15, 16]. Assessment of neurovascular status, skin inspection, and identification of areas of tenderness should be performed. If the acute examination reveals a stable knee with suspected articular cartilage injury, the knee should be immobilized, and patient should be placed on crutches to avoid bearing weight on the affected extremity.

Conservative management of acute chondral lesions may include nonsteroidal anti-inflammatory medications, intra-articular steroid and hyaluronic acid injections, and focused physical therapy to maintain adequate range of motion and strengthening of the affected limb. In specific instances, unloader braces of the affected compartment may be of benefit as well [6, 8, 13].

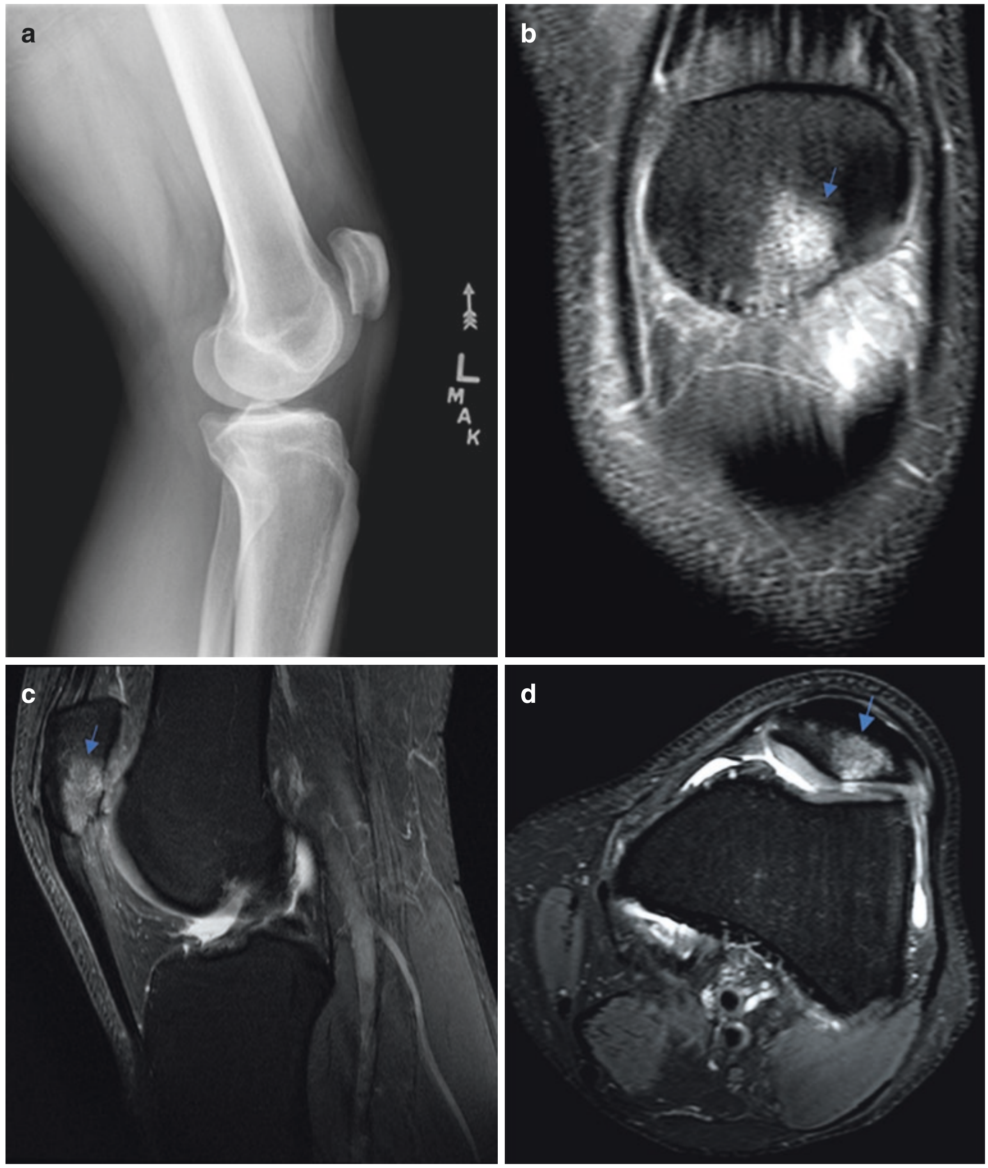


Fig. 24.6 Knee pain in a 31-year-old soccer player as a result of a fall during the game. Plain radiography was unremarkable (**a**). MRI revealed a focal full-thickness cartilage damage along the inferior lateral patellar facet (**b**–**d**). The well-defined margins (arrows) in T2-weighted MRI images (**a**–**c**) suggest a traumatic etiology

Indications for Orthopedic Referral

In the case of acute traumatic injury with significant concomitant ligamentous injury, an orthopedic referral is indicated. Patients with suspected higher-grade lesions or those with mechanical symptoms also need referral for evaluation and most likely for arthroscopy [2]. Bekkers et al. conducted a systematic review finding that the age of patient, size of lesion, and activity level of patient were the main factors when looking at outcome of articular cartilage repair surgery [17]. Lesions >2.5 cm should be treated with more advanced techniques, such as autologous chondrocyte implantation (ACI) or osteochondral autologous transplantation (OATs) [17]. In smaller lesions <2.5 cm, microfracture is considered an appropriate first-line treatment option [17]. The caveat to microfracture treatment is that active patients have poorer response to this treatment option [17]. Patients ≤30 years benefit from surgical repair compared with those >30 years of age [12, 17, 18]. In the setting of an acute ACL injury, the presence of a femoral condyle bone contusion visible on MRI has been found to be indicative of a cartilage deterioration, which should be considered in management of the acute knee injury [5].

Follow-Up Care

Post-injury management includes a period of non-weight-bearing for up to 8 weeks, again depending on concomitant injury. Rehabilitation with focus on optimizing early range of motion is a mainstay following an injury. Proper rehabilitation following surgical procedure is a critical factor in helping return to sport and preinjury activity levels [8].

Return to Sports

Return to athletic activity is multifactorial. Certain sports have heavier demand on impact and weight distribution across the joint, most specifically soccer, basketball, and football. These sports require more caution with return following cartilage injuries. Other factors in return to sport include age of injured individual, lesion size and grade, concomitant injuries, length of preoperative interval, and activity level [2, 8].

Complications

Progressive joint degeneration has been found to commonly occur with acute cartilaginous injuries. Isolated cartilage lesions have poor spontaneous repair potential and, if left untreated, can lead to progressive global cartilage loss over time. A lack of vascularization in this tissue leads to blunting of the inflammatory response and, therefore, less ability for healing. Those who are physically active and young are faced with the greatest risk of long-term complications [2, 3, 8].

Pediatric Consideration

In the setting of an acute traumatic injury in the skeletally immature patient, chondral lesions are the most prevalent injuries followed by meniscal and ACL injuries [19]. There is no statistically significant difference in the prevalence of chondral injury before and after physeal closure [19]. Joint injury in the pediatric patient appears to be the strongest indicator for the development of osteoarthritis [20]. Hemarthrosis in the setting of trauma with a stable knee on examination in the skeletally immature is a sign of an acute chondral defect [3]. In a retrospective study evaluating pediatric patients with ACL tears, the presence of medial or lateral meniscal tears meant the patient was more likely to have an associated chondral injury in the affected compartment than those who did not have meniscal tears [21].

Acute Meniscal Injuries

The medial and lateral menisci are located between the tibia, medial, and lateral femoral condyles (Fig. 24.1a). The menisci serve multiple roles in the healthy knee. They are important for shock absorption, load distribution, joint stability, lubrication, limiting extreme flexion and extension, controlling knee movements, and proprioception [22, 23]. Blood supply to the peripheral third of the meniscus is derived from the superior and inferior branches of the lateral and medial geniculate arteries.

Mechanism of Injury in Sports

Acute meniscal injuries occur most commonly with a rotational force around a planted foot [24]. In the setting of axial loading and rotational forces, significant amount of shear forces can be generated between the tibial and femoral condyles. This can happen in sports requiring jumping, landing, and knee rotations (e.g., skiing). In a study of high school athletes, among soccer players, the largest proportion of meniscal injuries occurred with a planted foot and rotational force. For both boys' and girls' basketball and girls' volleyball, the most common specific activity causing injury was jumping and landing [24]. Traumatic tears can be associated with concomitant chondral or ligamentous injury [22, 24].

Epidemiology

Meniscal pathology in younger patients is most likely secondary to an acute traumatic event, with degenerative findings most common in individuals over 30 years of age. More than one third of all meniscal tears are associated with an ACL injury. The peak incidence of meniscal injuries for men is between 21 and 30 years of age and in women is between 11 and 20 years of age [25]. In a study of meniscal injuries among US high school athletes, using a sample of over 21,000 athlete exposures, Mitchell et al. found an injury rate of 5.1 per 100,000 exposures [24]. Football had the highest injury rate (42.4%). Girls' soccer, girls' basketball, and boys' wrestling accounted for the highest rates among all sports [24]. More than two third of injuries occurred in males, skewed by the high rates in football players [24]. In gender-comparable sports, however, the injury rates were 5.5 in females compared to 2.5 in males per 100,000 exposures [24]. In this study, 15% of all knee injuries were meniscal, with noncontact injuries accounting for 38% of meniscal injuries [24].

Classification

The ISAKOS classification of meniscal tears released in 2016 is a form of classification that provides uniform documentation of meniscal pathology, with sufficient inner-observer reliability among surgeons [23]. (Table 24.1).

The first category in the ISAKOS classification system is tear depth, which refers to partial- or complete-thickness

Table 24.1 Simplified summary of ISAKOS classification of meniscal tears description categories [23]

Category	Descriptors
1. Tear depth	Partial or complete
2. Location/rim width	Zone 1, 2, 3
3. Radial location	Anterior, middle, posterior
4. Central to popliteus hiatus?	Yes or no
5. Tear pattern	Horizontal, radial, longitudinal, flap, complex
6. Quality of meniscus tissue	Degenerative, nondegenerative, undetermined
7. Tear length	In millimeters, maximum length reported

Reprinted from Wadhwa et al. [23], with permission from Elsevier

involvement of the meniscus (Fig. 24.7). This refers to either through superior and inferior surfaces of the meniscus or inner and outer surfaces. The second category is location and rim width. These are three zones (Fig. 24.7b) that define how far a tear extends into the meniscus. The third category looks at three defined sections of the meniscus, each approximately one third of the total meniscus (Fig. 24.7b). Of note, this system does not define the terms classically used such as posterior horn, body, and anterior horn [23]. Category 4 is for lateral meniscal tears. If the tear extends either completely or partially in front of the popliteal hiatus, it is considered central to the popliteus hiatus. Tear pattern is the 5th category of description and is the most extensive of all the categories. Longitudinal tears are those that run parallel to the circumference of the meniscus, separating the meniscus into inner and outer segments (Fig. 24.8). These types of lesions are most common in the younger patient with acute injury, such as ACL rupture [26]. A bucket handle tear is when there is vertical displacement of the meniscus in a longitudinal tear, which extends into the joint space (Figs. 24.8 and 24.11). Horizontal tears occur parallel to the tibial plateau and is differentiated from a horizontal flap tear that contacts the meniscus obliquely without extending to the apex. Radial tears are defined as originating from the inner free edge of the meniscus and may progress to displaced complex tears (Fig. 24.9) [23].

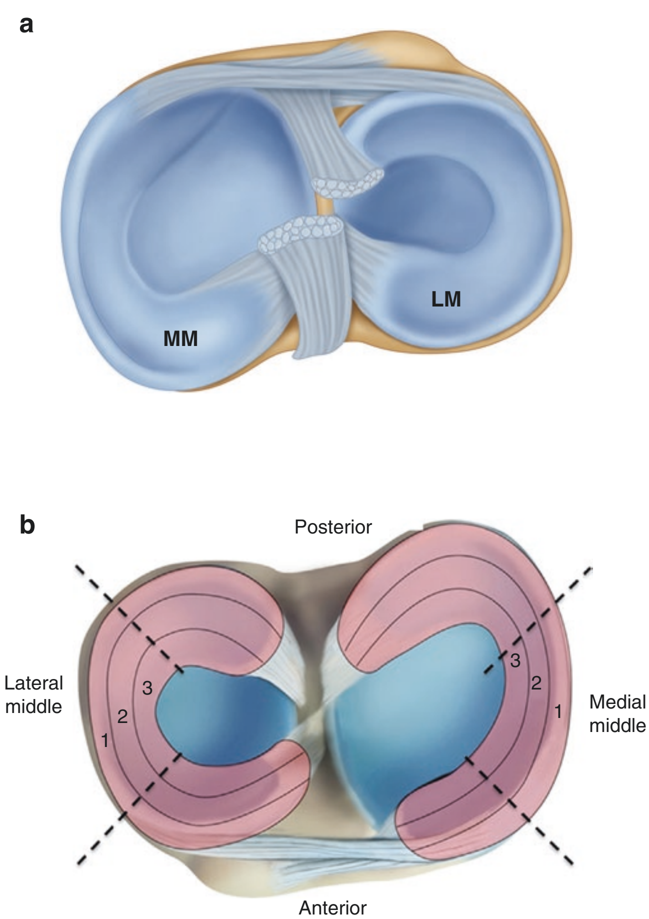


Fig. 24.7 Schematic view of the medial (MM) and lateral menisci (LM) (**a**). Illustration of rim width involvement and imaginary division of the menisci into the anterior, middle, and posterior parts for ease of reporting showing the zonal distribution in MM and LM as defined in the ISAKOS classification (**b**). (Reprinted from "Wadhwa et al. [23]," with permission from Elsevier [23])

Clinical Presentation

Meniscal injuries may present with localized joint-line pain, which is exacerbated by torsional weight-bearing movements. Pain is most commonly referred to as a sharp stab but may also be described as a dull ache lasting several hours after inciting movements. A small effusion may be present on examination, with difficulty placing the knee into full extension [22, 25]. Mechanical symptoms, such as locking of the knee, suggest a fragment of a bucket-handle tear preventing full extension. Other mechanical symptoms, such as catching, giving way, and grinding, are less closely associated with meniscal pathology [22, 25]. Pseudo-locking is represented by inability to do full extension, either from effusion or pain inhibition. The frequency and severity of symptoms are related the location, size, and mobility of the lesion [22].

Diagnosis

Under the correct clinical settings as above, a full examination of the knee with a comprehensive ligamentous evaluation is the first step in confirming the diagnosis. Special examinations may be useful in the clinical diagnosis of a meniscal tear. McMurray's test has been historically performed, but its sensitivity and specificity are not great [22, 27, 28]. Apley's test also has been reported to have high specificity with the modest sensitivity [22, 25, 27–29]. The Thessaly test is the most clinically useful physical examination finding, with sensitivity and specificity of reported up to 90% in detecting meniscal injury [22, 25, 27, 29, 30]. However, there is no one single physical examination test that can reliably detect a meniscal tear [22, 25, 27, 29, 30]. Plain radiography is ordered to evaluate for loose bodies, occult fracture, and OCD lesions. MRI is a highly accurate imaging modality for the evaluation of acute meniscal injuries (Figs. 24.10 and 24.11). The gold standard for the diagnosis of meniscal tears is arthroscopy [22, 25, 27, 29, 30].

Initial Management

A comprehensive knee examination should be performed for concomitant injuries, and if meniscal injury is suspected, the patient should be placed non-weight-bearing on crutches until follow-up evaluation. Conservative management including physical therapy is a reasonable initial manage-

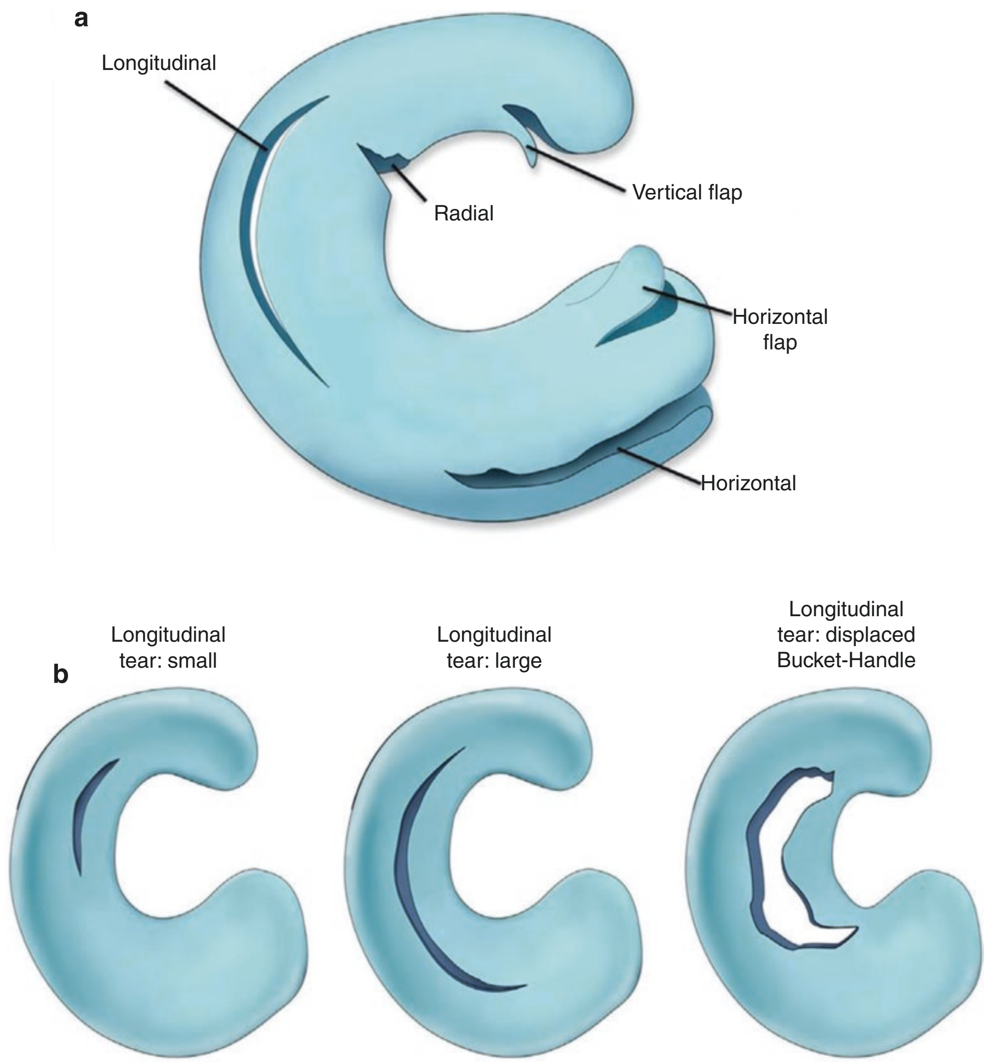


Fig. 24.8 Illustrations showing appearances and orientations of different meniscal tears (**a**). Notice larger longitudinal tear extending into the anterior and posterior parts of the meniscus can get displaced as a bucket-handle tear (**b**). (Reprinted from "Wadhwa et al. [23]," with permission from Elsevier [23])

ment option for many meniscal injuries [22, 25]. Current literature agrees that exercise therapy should be recommended for patients with degenerative meniscal lesions and patients older than 40 years of age [22, 25]. For patients with traumatic meniscal tears and in younger patients, however, the evidence is inconclusive [22, 25, 31]. Location in the vascularized region of the meniscus and size of the defect play a role in the decision to pursue surgical repair [22, 25, 31]. Vertical tears that are <5 mm in length in the peripheral one third of the meniscus have the optimal factors for nonoperative management [22, 25, 31, 32].

Indications for Orthopedic Referral

The absolute indication for referral to an orthopedic surgeon is locking of the knee, suggestive of a bucket-handle tear (Fig. 24.11). This loss of joint function necessitates surgical intervention [22, 32, 33]. Mitchell et al. reported that the majority (64%) of meniscal injuries, ultimately, required surgical intervention [24]. Concomitant injuries were reported in half of knees with meniscal injuries, with the ACL being the most common additional structure injured [24]. Multiple studies reported that approximately one third of ACL injuries have associated meniscal tears [24, 34, 35]. In the young athlete, nonoperative management has a high failure rate, and therefore surgical repair is often recommended [36].

Follow-Up Care

In patients that are selected for nonoperative management of acute meniscal tears, the mainstay of management is physical therapy. Although there are no absolute guidelines on the amount or duration of physical therapy following injury, a twice-weekly individualized exercise program for 8–12 weeks has been studied as a sufficient course of recovery [22, 31]. Focus of therapy should be to improved range of motion, muscle strengthening, and proprioceptive exercises [22, 31]. Nonsteroidal anti-inflammatory medications may be recommended for 8–12 weeks following injury. Although

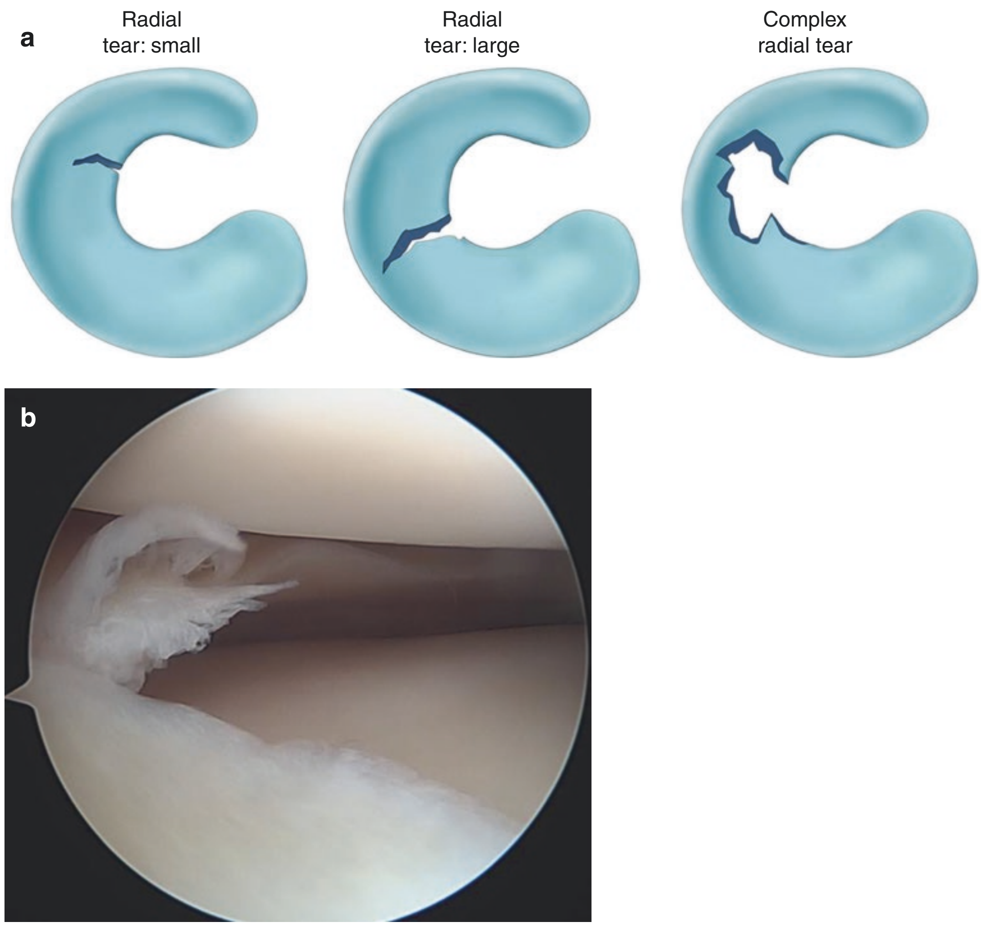


Fig. 24.9 Illustration of radial tear and its complex variant (**a**). (Reprinted from "Wadhwa [23]," with permission from Elsevier [23]). A radial lateral meniscal tear is visible during arthroscopy (**b**)

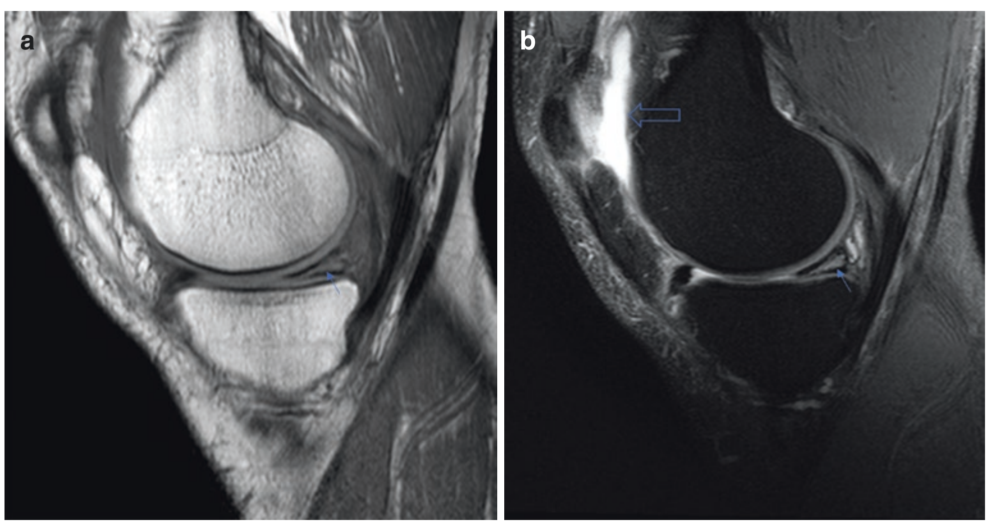


Fig. 24.10 A large horizontal oblique tear (arrows) of the body and posterior horn of the medial meniscus present in T1-weighted (**a**) and T2-weighted (**b**) MRI images in a 41-year-old male as a result of a twisting knee injury. T2-weighted MRI (**b**) also shows a significant knee joint effusion (open arrow)

commonly practiced, there is little evidence for the effectiveness of bracing following meniscal injury, and this is not currently recommended [22].

Should the patient require surgical fixation, rehabilitation following surgical meniscal repair is generally broken down into three phases. The initial postoperative window is from 3–5 days postsurgery extending out to 4–6 weeks postsurgery [37]. The main goals of this phase is to protect the post-surgical knee, reduction of effusion, regaining extension, and improved leg control [37]. Phase 2 occurs over the next 2 months and focuses on regaining range of motion, gait normalization, and control on single leg stance [37]. The final phase that is approximately 3 months postoperative works on pain-free return to sport-specific exercise [37].

Fig. 24.11 A bucket-handle-type medial meniscus tear (arrows) in a 26-year-old male as a result of twisting his knee during yoga

The overall rehabilitation process is far more extensive than this small snapshot and is beyond the scope of this book.

Return to Sports

Following surgical repair of traumatic meniscal lesions, return to sports on the preinjury level has been found to be quite high (90%) [38]. Delay of return to sports following surgical repair varies in the literature, with timetables between 4 and 6 months quoted [38]. These results seem to be consistent between professional and mixed-level athletes, although the failure rate was higher in mixed-level over professional athletes [38]. Return to play is usually shorter in patients with post-meniscectomy than in both meniscal repair and meniscal allograft transplantation, making it the more favorable procedure for athletes in season [39]. Patients with lateral meniscus have a longer recovery period following meniscectomy when compared with those after medial meniscectomy [40]. Patients with lateral meniscectomy have higher risk of complications than patients with medial meniscectomy and a higher chance of undergoing repeat arthroscopy [40]. For athletes with nonoperative management, return to sport activities should be guided by symptoms and ability to perform specific physical activity [41]. Rehabilitation should focus on functional activities related to sport, range of motion, proprioception, and strengthening of surrounding musculature [41].

Complications

Pain and swelling are the common complications for surgically repaired lesions of the meniscus [40]. Advanced degenerative disease is well studied following surgical removal of damaged parts of meniscal tissue. Conservative management should be the mainstay of treatment for degenerative meniscal tears and tears in the older population [35, 42]. Traumatic injury of the meniscus is associated with the development of osteoarthritis of the affected knee as well. The combination of a torn meniscus and surgical resection alters the transmission of load across the tibiofemoral articulation and increases the stress across the articular cartilage. Large resections of meniscus in arthroscopy worsens the long-term prognosis for development of degenerative disease as well [34, 42, 43]. Delayed ACL reconstruction increases the likelihood of a worsening of an existing medial meniscal tear [44].

Pediatric Consideration

The pattern of meniscal injury varies based on the age at time of injury. A retrospective cross-sectional study compared meniscal tears of children to those of adolescents (defined as having closed growth plates) [45]. The children group were less likely to have associated ligamentous injury [45]. They were, however, more likely to have discoid meniscus tears and lower BMI [45]. Complex tears were the most common meniscal injuries found in both groups, favoring those of male gender and those with higher BMI [45]. Meniscal injuries were more common in the skeletally mature patients. In this study, half of meniscal tears were ultimately treated with surgical repair [45].

Fractures Around the Knee

Femoral Condyles Fractures

The femur is the longest bone in the body and serves as the attachment site (both origin and insertion) for over a dozen muscles. It is also among the strongest bones in the body. As a result, fractures of the distal femur tend to be very complex injuries [46]. The femoral condyles are formed from a flaring of the distal end of the femur [47]. The anterior surface of the femoral condyle articulates with the patella to form the patellofemoral joint or anterior knee compartment [47]. On the posterior aspect of the distal femur, the condyles make way for the intercondylar fossa or notch, which provides an attachment site for the cruciate ligaments [47, 48]. Laterally, the condyles have epicondyles, which serve as attachment sites for the collateral ligaments [47]. Medially, slightly proximal (superior) to the medial epicondyle lies the adductor tubercle, which serves as an attachment for the adductor magnus muscle [47]. In terms of vasculature, the popliteal artery runs in the posterior knee and begins approximately 10 cm proximal to the knee joint and is a continuation of the femoral artery [46–48].

Mechanism of Injury in Sports

Femoral condylar fractures occur as a result of high-energy trauma in sports such as skiing and motor sports. Most of the femoral condyle fractures in sports are limited to stress fractures.

Epidemiology

Fractures of the distal femur including the femoral condyles comprise less than 1% of all fractures and are far less common than fractures of other regions of the femur particularly the shaft and proximal femur [49, 50]. Major fractures of the femur and its condyles typically have a bimodal distribution. They are always a result of high-energy impacts in young individuals such as high-velocity motor vehicle accidents or falls from heights [47, 49, 50]. They are also seen in the elderly with osteoporotic bones and result from falls, usually from standing [47]. Given the high amount of energy required to cause distal femur fractures in young patients, it is likely that the injury is not isolated, but one of many problems for the trauma victim, making these fractures complex injuries.

Classification

The AO/OTA classification for distal fractures groups them into extra-articular, partial articular fracture, and complete articular fracture (Chap. 23; Fig. 23.2b) [51]. They are also subclassified based on their displacement, comminution, and pattern of fracture [51].

Clinical Presentation

Due to the severity of many of these fractures, thigh deformity is usually present. Often time, patients have multiple traumatic injuries. Distal pulses should be checked in any patients with possible condylar fractures.

Fig. 24.12 Medial epicondyle fracture in a 21-year-old female pedestrian as a result of a direct trauma by a car

Diagnosis

Plain radiography of the hip, femur, and knee is the first step in diagnosis of distal femoral fractures (Fig. 24.12). As most of these fractures are serious, patient's management and transfer to higher level of care should not be delayed for obtaining additional images. More advanced imaging such as CT scan may be needed for preoperative planning (Figs. 24.13, 24.14, and 24.15).

Initial Management

Unless there is a small epicondylar or avulsion fracture, most patients' situation should be taken seriously [47, 50]. Because of the mechanism and severity of femoral condylar fractures, all patients with major intra-articular fractures should be stabilized and transferred to the nearest emergency department. Depending on the location of the fracture, a knee immobilizer seems to be appropriate in most cases. ATLS algorithm should be applied as hemorrhagic shock may occur.

Indications for Orthopedic Referral

In general, all femoral condylar fractures should be referred to an orthopedic surgeon. Only a small percentage of patients with femoral condylar fractures may be managed nonoperatively [47, 49, 50]. In the absence of any other major injuries, a nondisplaced and partially intra-articular fracture may be managed nonoperatively with immobilization (i.e., cast or splint) [47, 49, 50].

Follow-Up Care

If the patient is managed nonoperatively, a clinical evaluation and repeat plain radiography in 2 weeks are recommended [47, 49, 50]. Frequent follow-ups with early range of motion exercises and physical therapy as tolerated are also recommended [47, 49, 50].

Return to Sport

When an athlete has completed the treatment plan, they can start a return-to-sports protocol as long as they are pain free, and their plain radiographs demonstrate appropriate healing.

Complications

Depending on the location and type of the fracture, fat embolism, hemorrhagic shock, neurovascular complications, nonunion, and early degenerative joint disease may occur [47, 50].

Pediatric Considerations

Fracture of the femoral condyles are very rare in children, particularly as a result of sporting activities. Children with femoral condyle fracture should be managed similar to adults with initially following the ATLS algorithm.

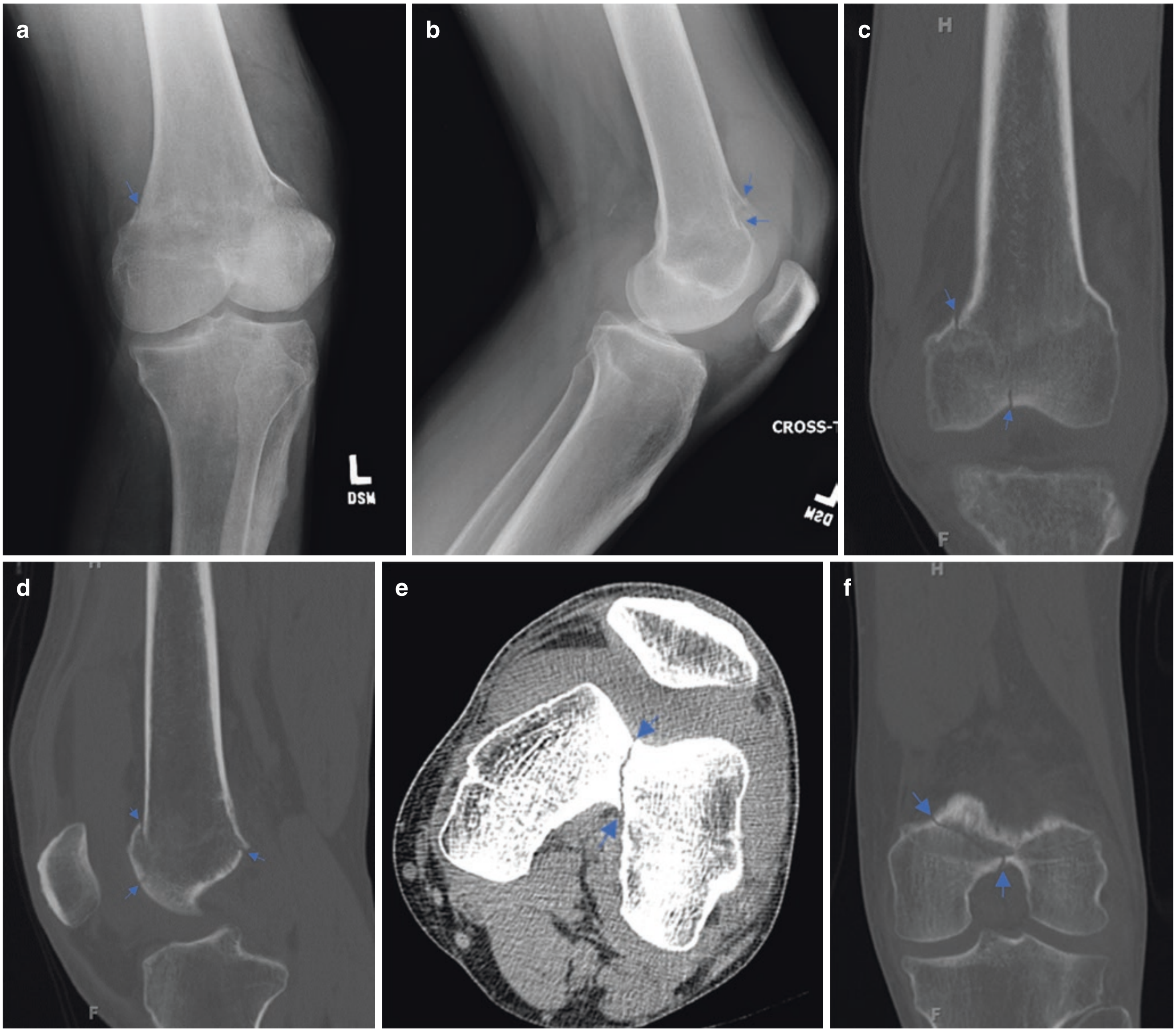


Fig. 24.13 Nondisplaced, intra-articular, medial femoral condyle (intercondylar) fracture (arrows) in a 53-year-old male as a result of a fall (**a**, **b**). CT scan images demonstrate the extent of the fracture (**c**–**f**)

Patella Fractures

The patella is a triangular bone with a proximal base and a distal apex. The proximal part serves as an attachment site for the quadriceps tendon, which comprises tendons from the rectus femoris, vastus medialis, vastus intermedius, and vastus lateralis [52, 53]. This tendon encapsulates the patella and distally becomes the patellar tendon (ligament) inserting on the tibial tuberosity [54]. Part of the vastus medialis tendon extends medially forming the medial retinaculum attaching distally to the medial condyle of the tibia. Similarly, part of the vastus lateralis tendon forms the lateral retinaculum and inserts to the lateral condyle [53, 54]. The patella is the largest sesamoid bone in the body. By definition, a sesamoid bone is a bone embedded within a tendon or muscle. It functions similar to other sesamoid bones in that it is thought to protect the quadriceps tendon from abrasion. In addition, it also improves the biomechanical leverage by acting as a fulcrum allowing for better force distribution during knee extension [54, 55]. Further, it also protects the knee joint from direct trauma and provides nourishment for the articular cartilage of the distal femur [54, 55]. If the medial and lateral retinacula are intact following a patella fracture, the patient may retain the ability to actively extend the knee. The patella is vascularized by the anastomosis of the genicular arteries of the knee. The entry point of the arteries lies in the middle and distal patella. Thus, fractures through the middle can compromise blood

Fig. 24.14 Comminuted, open, intra-articular distal femur and patella fractures in a 23-year-old female as a result of a tree strike while snowboarding (**a**, **b**). CT scan images demonstrate the extent of the fracture (**c**, **d**). Postoperative radiograph shows near-anatomic alignment (**e**)

supply to the proximal patella increasing risk for avascular necrosis [53–55]. Normally, in over 80% of individuals, the patella arises from a single center of ossification. However, in about 20% of individuals, two to three centers of ossification may exist. When these centers fail to fuse with the main patella, the patella can then develop as two separate pieces known as bipartite patella (Fig. 24.16) [54]. The incidence of bipartite patella is reported to range from 0.2% to 6% and is more common in males compared to females [54].

Mechanism of Injury in Sports

Most fractures of the patella occur from direct forces on the patella [54, 55]. This includes falls onto a flexed knee or the knee striking a dashboard during a motor vehicle accident.

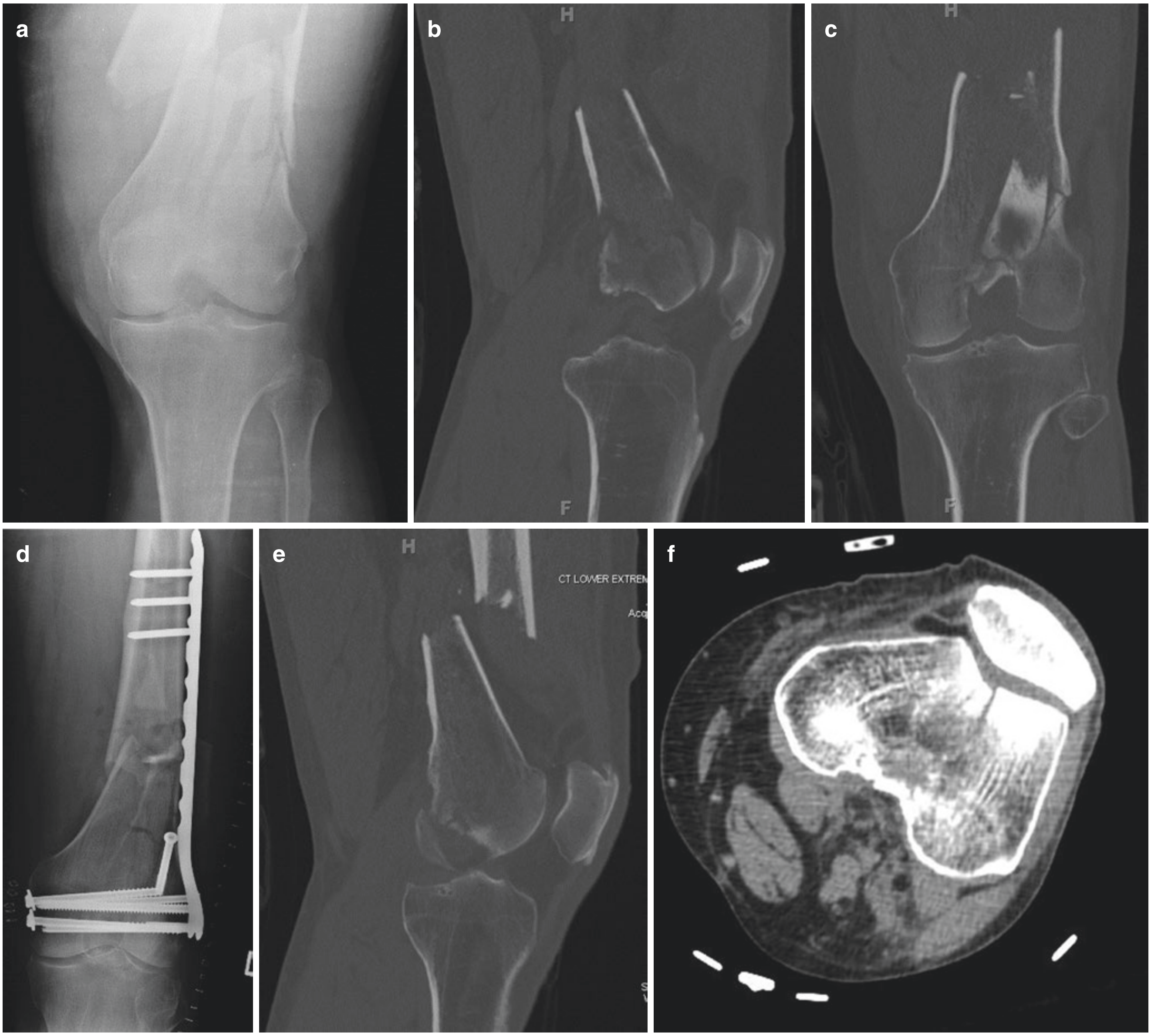


Fig. 24.15 Displaced distal femoral shaft and displaced, intra-articular, and comminuted distal femoral fractures in a 65-year-old male as a result of a car accident (**a**). CT scan images demonstrate the extent of the fracture (**b**–**e**). Postoperative radiograph shows near-anatomic alignment (**f**)

Patella fractures are said to be displaced if there is >2 mm articular step-off or > 3 mm diastasis (i.e., separation of the fractured segments) [54–56].

When associated with a fall, they are more likely to cause fractures in the elderly due to their osteopenic bones and propensity to fall. Direct trauma, whether from falls or motor vehicle accidents, generally causes comminuted stellate fractures [54–56].

In sports, it commonly occurs from sudden and forceful flexing of the quadriceps (e.g., jumping from a moderate height or sudden stopping from full sprint) or direct trauma from an object (e.g., tree strike during ski injuries) [54–56]. Avulsion of the patella can occur if the force generated by the quadriceps exceeds the strength of the bone. These are more likely to be displaced if the lateral and medial retinacula are also torn as a result. Indirect trauma such as from a jump generally causes a transverse patella fracture usually involving the central or distal third of the patella, and there is often associated disruption of the medial and lateral retinacula. Diastasis of the fractured patella may occur with the force of the contracting quadriceps [54–56]. Stress fractures are uncommon but can be seen in athletes.

Epidemiology

Patella fractures account for approximately 1% of all skeletal injuries in both adults and children and have a bimodal dis-

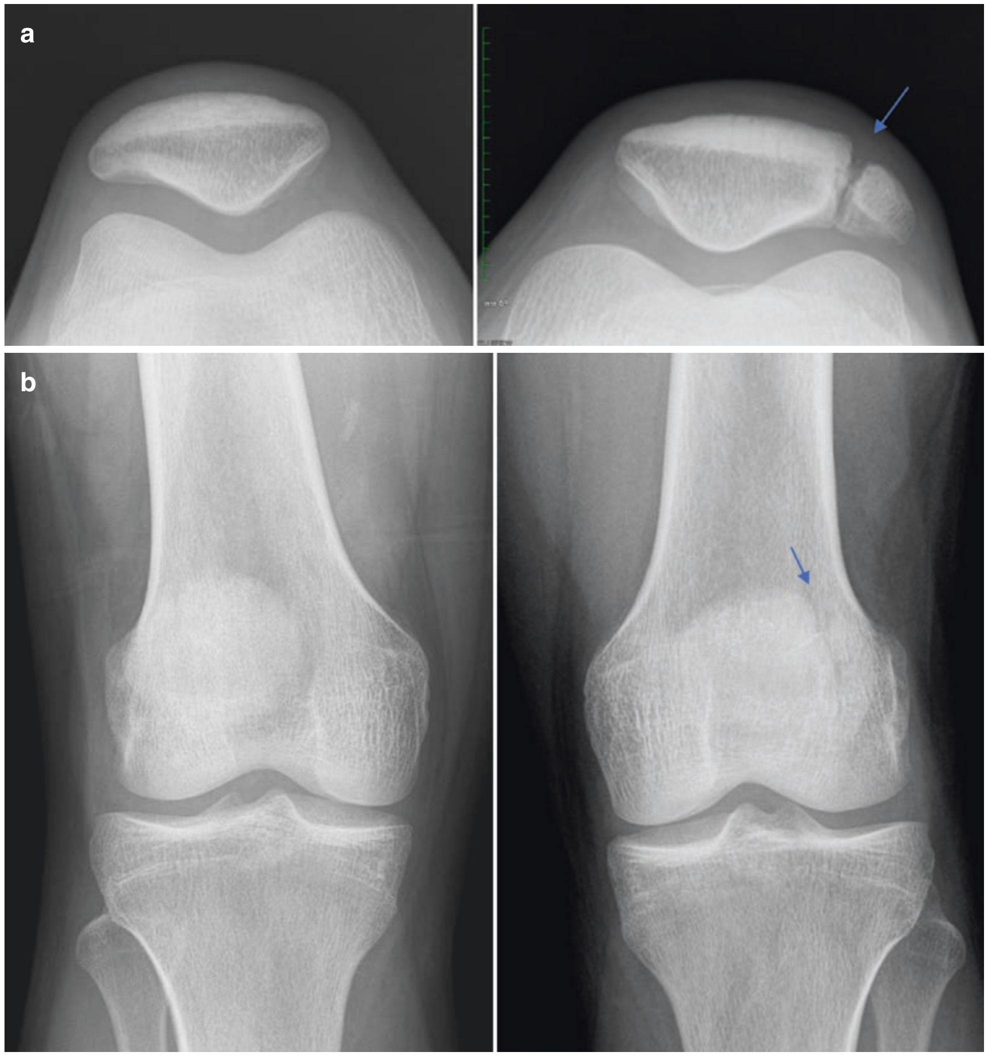


Fig. 24.16 Unilateral bipartite patella (arrows) in a 36-year-old male as an incidental finding (**a**, **b**)

tribution [53, 57]. In males, the incidence of patellar fractures is highest between the ages of 10 and 19 years; while in females, the incidence is highest between the ages of 60 and 80 years [57]. Although serious when they occur, fortunately they are not very common with an incidence rate of 13 per 100,000 individuals [57].

Classification

There are few classification systems for patellar fractures [55, 58, 59]. The AO/OTA classification for patella fractures groups them into extra-articular, partial articular sagittal fracture, and complete articular fracture [59]. The general description of patellar fractures is based on descriptive anatomy and includes terms like transverse, stellate, comminuted, longitudinal or marginal, proximal or distal pole, and osteochondral (Figs. 24.17, 24.18, 24.19, and 24.20) [55, 58, 59]. Transverse fractures are the most common type [53, 55]. Fractures of the patella are also classified on the fracture pattern as either nondisplaced or displaced [57]. Displaced fractures are defined by an articular step-off greater than 2–3 mm and a diastasis between 1 and 4 mm [57]. Based on one classification system, patellar fractures have seven major fracture subtypes which include nondisplaced transverse, lower or upper pole (Fig. 24.21), vertical, multifragmented nondisplaced, multifragmented displaced (Figs. 24.14, 24.18, 24.19, and 24.20), and osteochondral [57].

Clinical Presentation

The clinical symptoms of a patellar fracture include pain and local swelling in prepatellar bursa and in the knee joint (Fig. 24.22a). In case of diastasis, a defect is palpable and often an intra-articular effusion (hemarthrosis) is present [52, 53, 57]. Generally, an extended knee cannot be raised against gravity due to disruption of the extensor mechanism [52, 53, 57]. However, when the extensor mechanism is preserved, the active extension of the knee joint is possible but limited due to a reduction in strength likely from pain inhibition [52, 53, 57].

Diagnosis

The diagnosis is suspected based on history including trauma to the knee such as a fall on a flexed knee and pres-

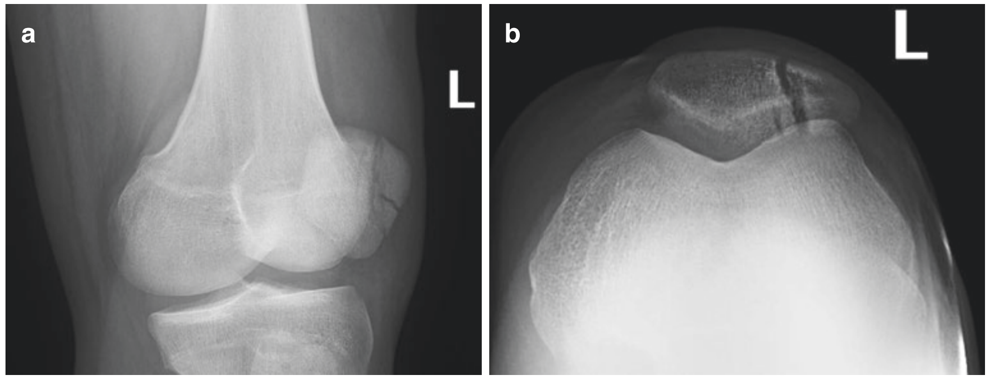


Fig. 24.17 Left comminuted and mildly displaced patella fracture in a 22-year-old male as a result of a tree strike while skiing (**a**, **b**)

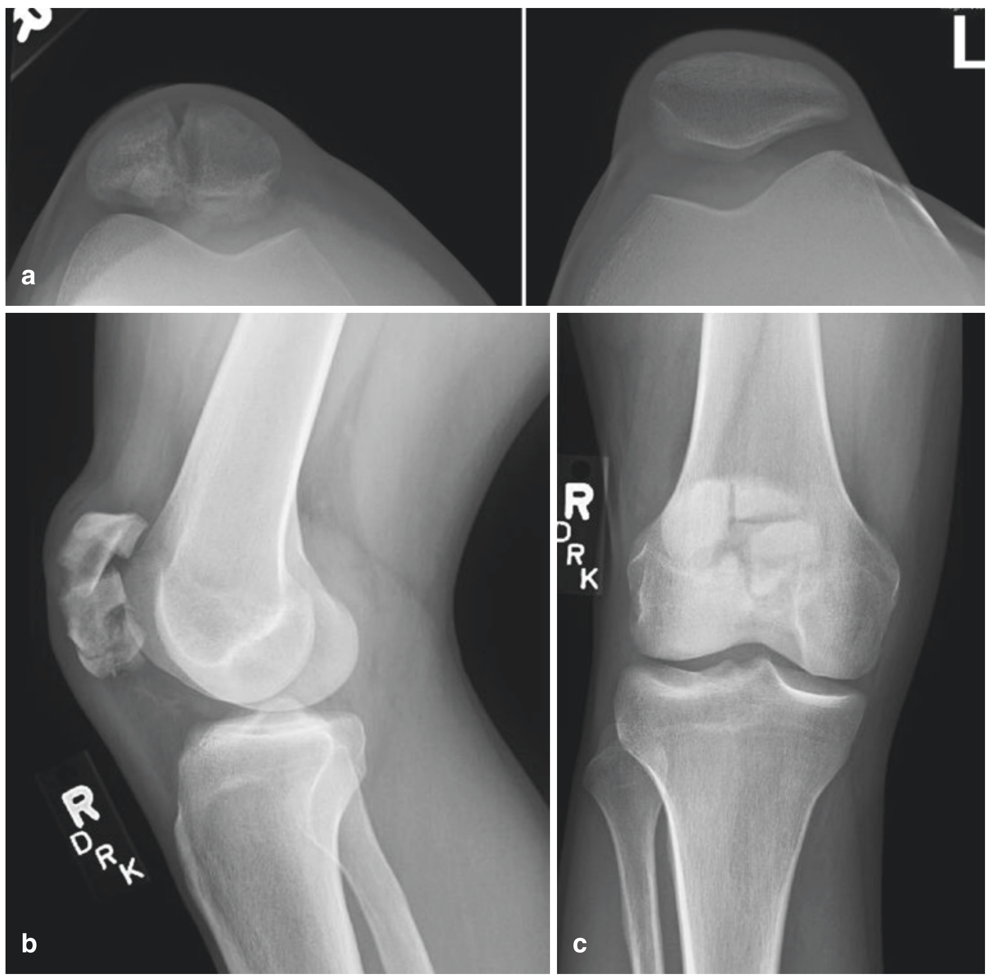


Fig. 24.18 A displaced and comminuted right patellar fracture in a 25-year-old male as a result of a tree strike while skiing (**a**–**c**)

ence of clinical findings of anterior knee swelling and tenderness of the patella [52, 53, 57]. The diagnosis is confirmed via plain radiography in the majority of athletes [52, 53, 55, 57]. Anteroposterior, lateral, and sunrise or merchant views are normally sufficient; however, keep in mind that a sunrise view can be difficult to obtain in patients with patellar fractures due to limited flexion from knee pain and swelling.

In general, if the patient's ability to extend the knee is preserved, it suggests a nondisplaced fracture [52, 53, 55, 57]. Most authors agree that the inability to actively extend the leg at the knee indicates disruption of the extensor mechanism and requires surgical intervention [4]. Because bipartite patellae can mimic acute fracture on plain radiographs, comparison radiographs of contralateral knee can be useful in distinguishing this for patients with the congenital variant [52, 55]. However, possibility of a unilateral bipartite patellae cannot be excluded (Fig. 24.16).

During examination, close attention should be paid to possible soft tissue damage, since even minimal wounds

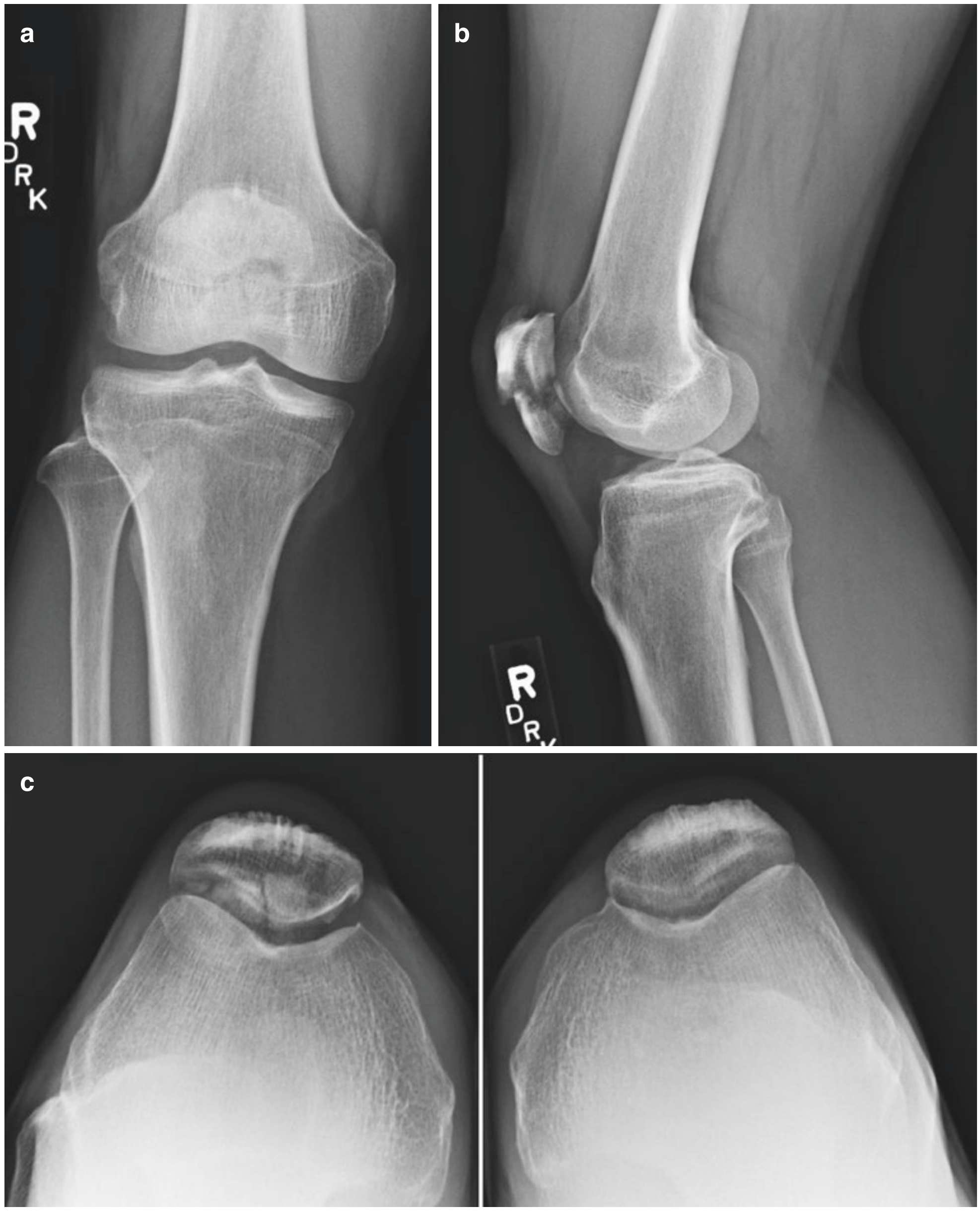


Fig. 24.19 A displaced and comminuted patella fracture in a 70-year-old male as a result of a tree strike while skiing (**a**–**c**)

with opening of the prepatellar bursa correspond to an open fracture and require surgical repair (Fig. 24.23) [52, 55].

Initial Management

There is no established protocol for nonoperative management in the literature [57]. However, the generally accepted acute management of athletes with patellar fractures is accomplished by placing the injured knee in a knee immobilizer, locked in full extension. Care should be exercised to not hyperextend the knee as this places unnecessary additional stress on the patella. Nondisplaced, marginal vertical fractures do not require immobilization.

Indications for Orthopedic Referral

All fractures that are displaced (i.e., >2 mm articular step off) or manifest diastasis (i.e., >3 mm separation of fractured fragments) require referral for surgery [52–55, 57]. In addition, all comminuted fractures of the patella regardless of diastasis or displacement require surgical referral [52–55, 57]. Athletes with evidence of disruption of extensor mechanism including avulsion fractures with tendon rupture; one third of all patella fractures and all open fractures must be referred to an orthopedic surgeon [52–55, 57].

Follow-Up Care

Nondisplaced, marginal vertical fractures can be treated with activity modification for 4–6 weeks and rehabilitation. Cylinder casts should be placed approximately 7 days post-injury so as to allow time for swelling to subside [52–55, 57]. The cast must extend from the groin to just proximal to the malleoli. The cast is maintained for 4–6 weeks. While a cast is required for children, a knee immobilizer may be used for highly compliant

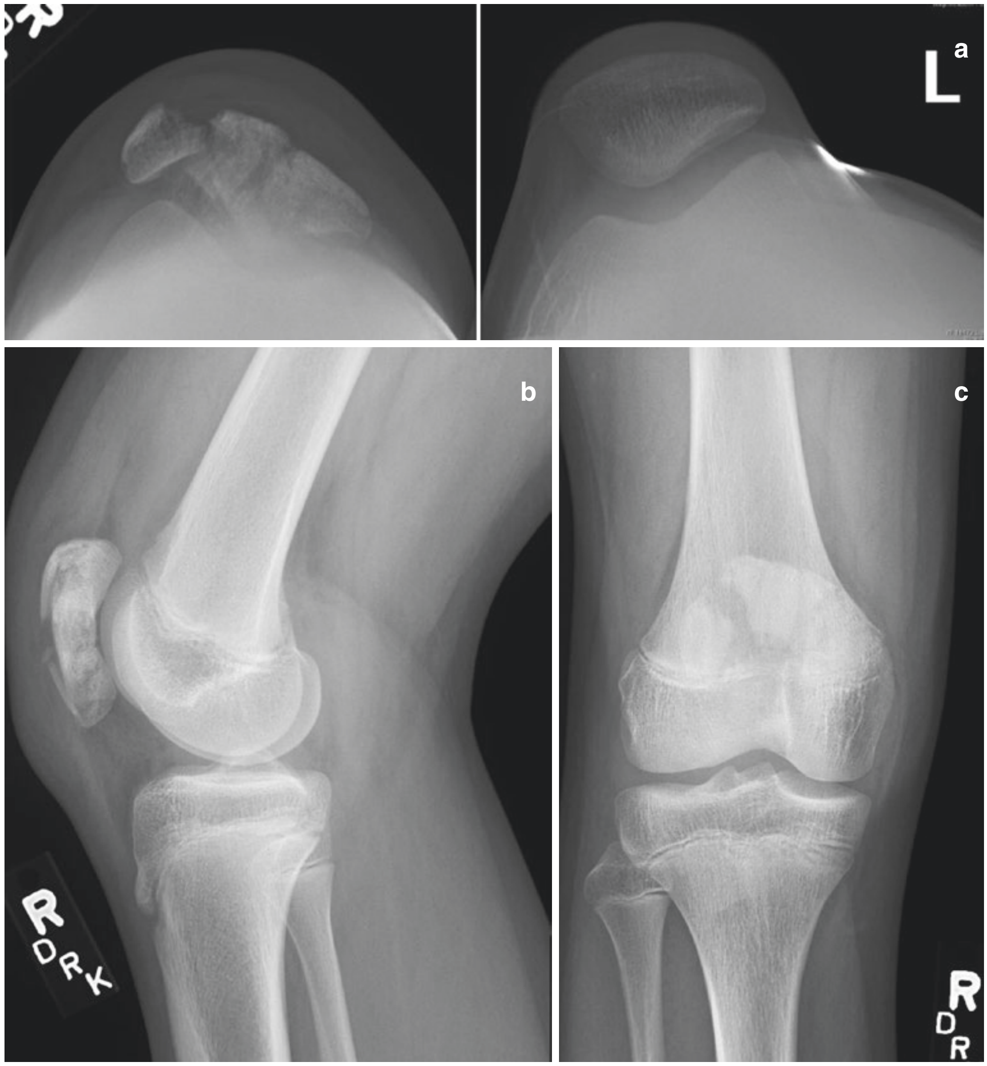


Fig. 24.20 A displaced and comminuted right patella fracture in a 15-year-old male as a result of a tree strike while skiing (**a**–**c**)

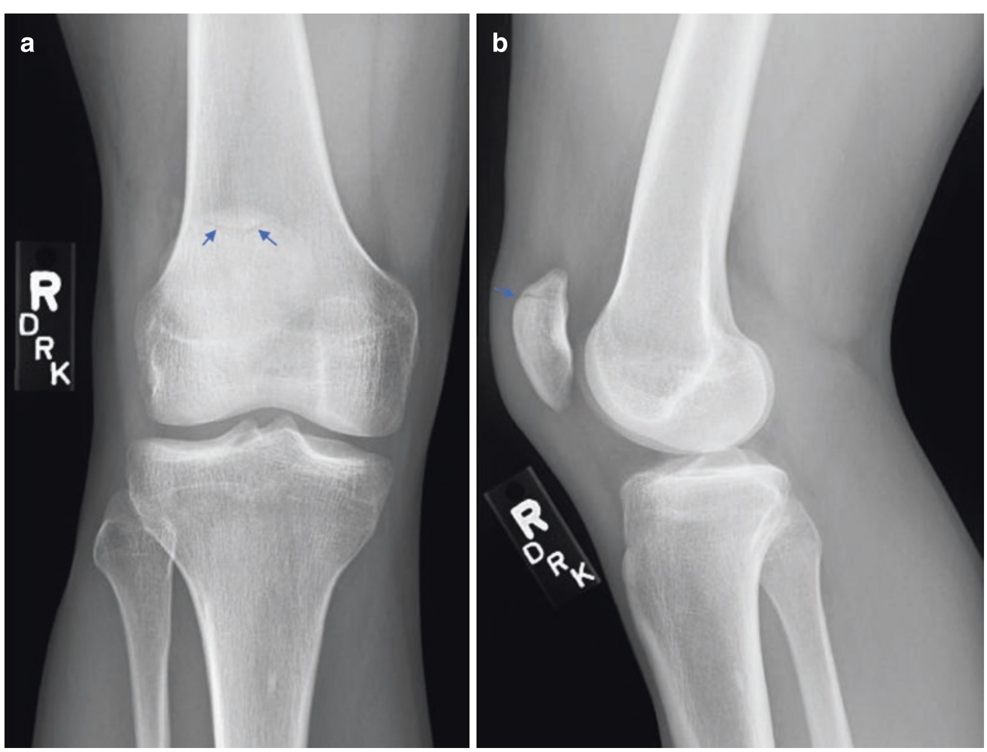


Fig. 24.21 A nondisplaced proximal pole fracture (arrows) of the right patella fracture in a 28-year-old male (**a**, **b**)

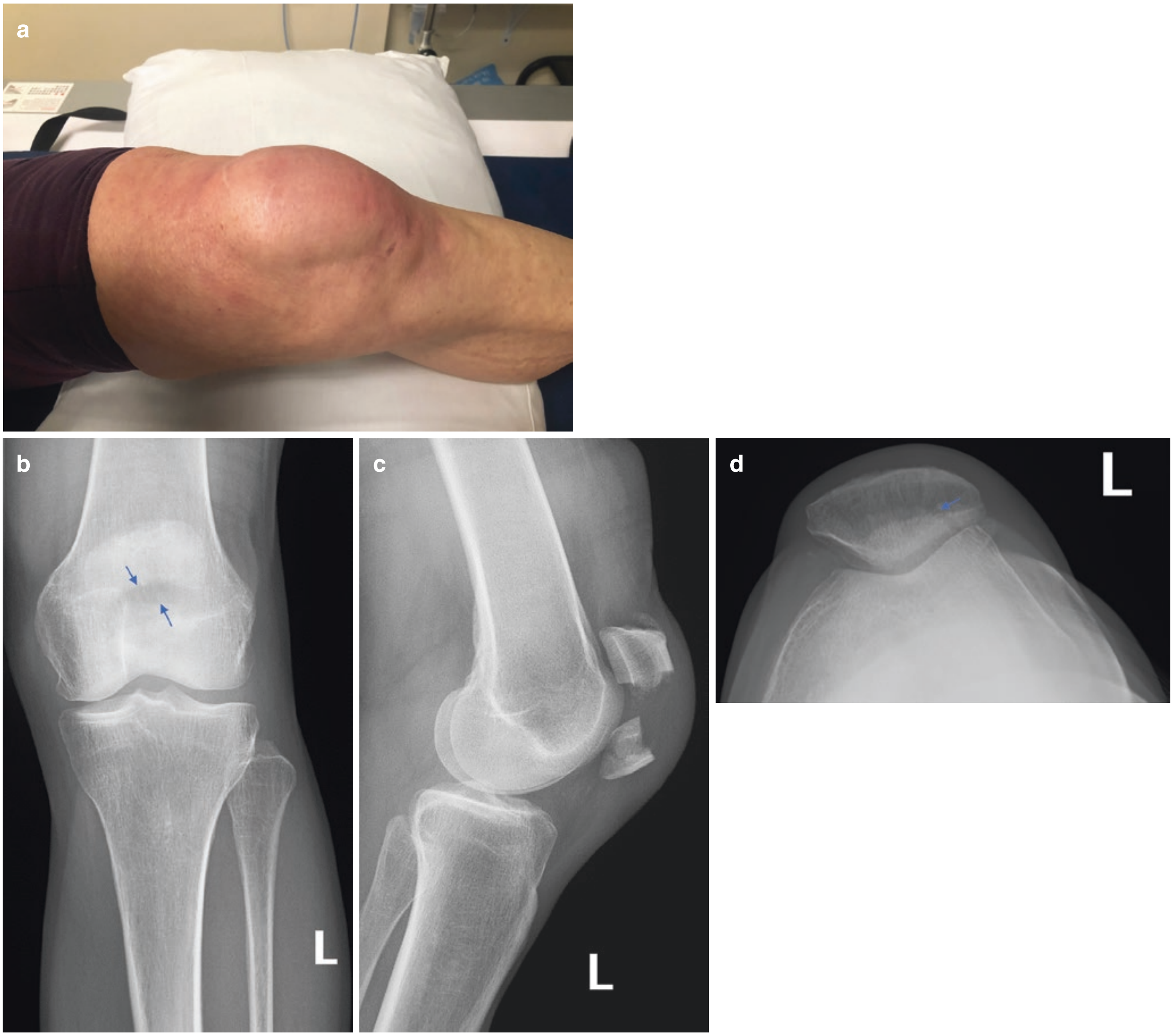


Fig. 24.22 Left patella transfer fracture in a 59-year-old female as a result of a tree strike while skiing. Prepatellar hemorrhagic bursitis is evident on observation and lateral view (**a**, **c**). Fracture line (arrows) is present in AP (**b**) and sunrise (**d**) views as well

adult patients [52–54, 57]. Patients should be treated with activity modification for 4–6 weeks and progressive strengthening and range of motion exercises [52–54, 57]. Follow-up radiographs should be obtained 2 weeks post-injury to ensure that the fracture remains nondisplaced and then again at 4–6 weeks to assess for fracture healing indicated by callus formation. Once bone callus is noted on radiographs, the cast can be safely removed and full recovery can be expected in 8–10 weeks. Upon removal of the cast, knee rehabilitation with physical therapy including range of motion exercises and progressive strengthening exercises can begin. Patients should begin with isometric quadriceps contraction exercises. Begin with two sets daily while maintaining contraction for 10 seconds per set. This can be progressed as tolerated to increase both the number of sets and duration of isometric contractions. Straight-leg raises are also beneficial to maintain both quadriceps and hip flexor strength. These are performed by lying supine with both legs extended. Each leg is then elevated to 30° while keeping knee extended. This position is held for 5–10 seconds and slowly returned to the starting position. This can be repeated 10 times per set and started with two sets daily. Again, this can be progressed as tolerated to increase both the number of sets and duration. Surgical literature notes that closed methods often yield poor results because of lengthy immobilization, which results in peri-patellar muscle weakening despite leg raising exercises [52–54, 57]. Athletes must be removed from contact sports for the entire 8–10-week duration.

Return to Sports

Contact sports can be resumed 8–10 weeks after initial injury, but this is variable and depends upon individual progression with rehabilitation and strengthening. Care should

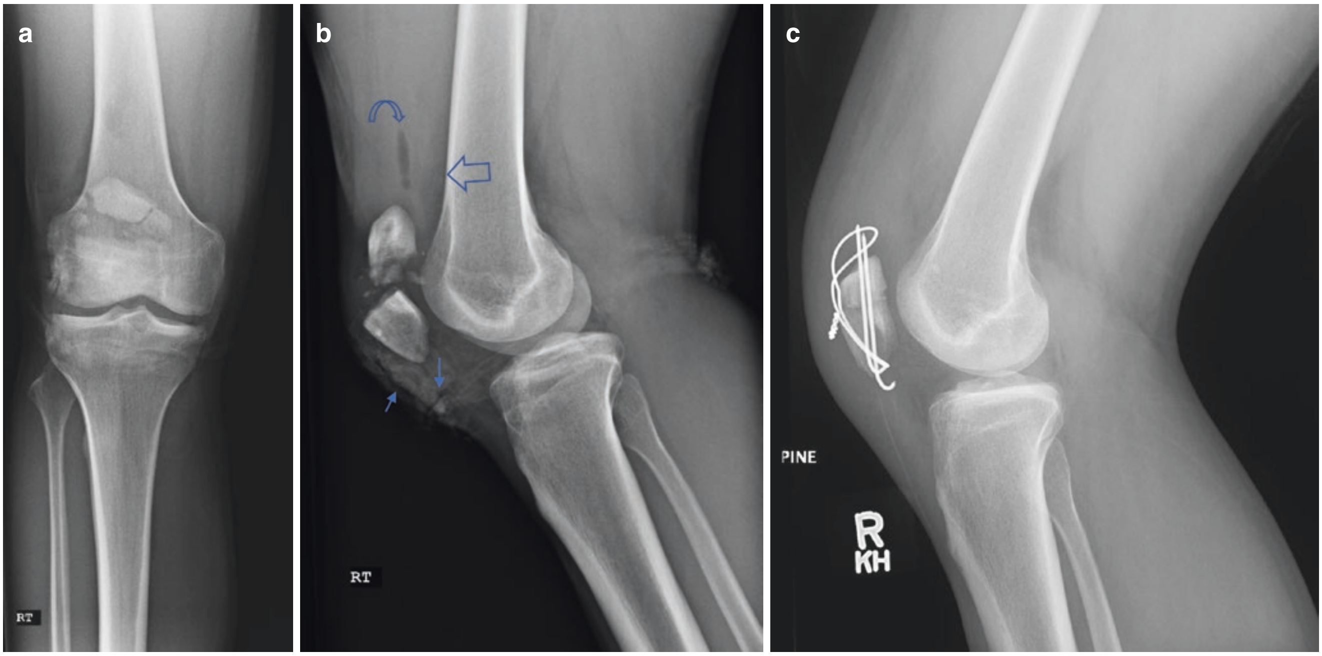


Fig. 24.23 Right open, displaced, and comminuted patella transfer fracture in a 24-year-old male as a result of a direct trauma (**a**, **b**). Significant hemarthrosis (open arrow), intra-articular air (curved arrow), and subcutaneous air (arrows) are present. Postoperative lateral view shows near-anatomic alignment (**c**)

be taken to work in cohesion with the physician, athletic trainer, physical therapist, and athlete. Thus, athletes should not return to full competitive sport until knee motion is fully restored, strength is at least 90% of the uninjured leg, and they have passed a functional, sport-specific assessment [52, 53, 57].

Complications

Perhaps the most common complication is traumatic cartilage damage, which often leads to post-traumatic retropatellar arthrosis [55]. Comminuted fractures of the patella may require partial or total patellectomy, which often leads to permanent disability of the knee [52, 55]. While not common, superficial and deep infections may occur, especially in open fractures [55].

Patellar fractures are known to be associated with hemarthrosis and other lower extremity trauma such injuries of the hip, femur, and tibia. It is therefore important to evaluate these areas for additional injuries. Further, patellar fractures can be associated with avascular necrosis, which is associated with both operative and nonoperative management although the risk is higher with operative repair [52, 54, 55, 57]. This usually presents as sclerosis of the proximal or distal portion of the patella on radiographs months after repair. Athletes with comminuted patellar fractures are also at a higher risk of developing early arthritis, and individuals in which pain limits rehabilitation may develop adhesions (e.g., arthrofibrosis), which usually need lysis with arthroscopy [52, 54, 55, 57].

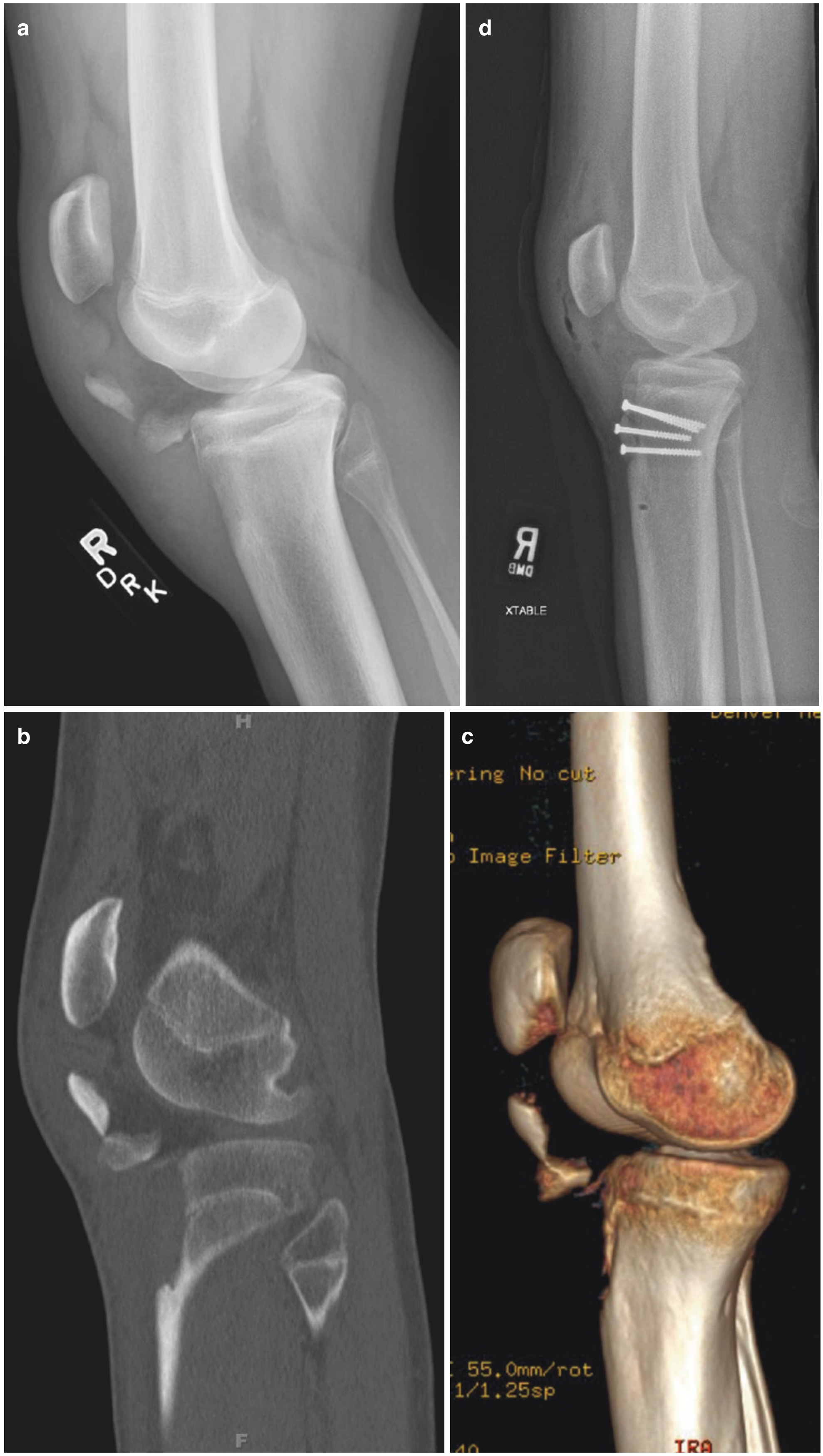
Pediatric Considerations

Pediatric patella fractures are uncommon (Fig. 24.20). The classification, mechanism of injury, and management are usually the same as adult's fractures. Although rare, children are more likely to have avulsion fractures of the patella also known as sleeve fractures of the patella [54]. These occur in less than 0.5% of patellar fractures. A sleeve fracture causes rupture of the extensor tendon and, along with it, avulsing a piece of the cartilaginous patella (in children who are skeletally immature). It also peels away the periosteum. When the periosteum is peeled away with a fragment of the underlying bone still attached, it is called a sleeve avulsion. These can occur at the superior pole of the patella as well as the inferior pole; however, most involve the superior pole [54]. The management of these injuries is similar to patellar or quadriceps tendon ruptures and typically requires surgery [53, 54].

Tibial Tubercle Avulsion Fractures

Tibial tubercle avulsion fractures are rare sports injuries that mainly occur among adolescents before the closure of the apophysis at age 13–17 years. It is more common among male adolescents [60]. History of Osgood-Schlatter's disease is a risk factor for this injury [60]. The mechanism of injury is usually an abrupt contraction of the knee extensor mechanism (patellar tendon). This usually happens after landing from a high jump in sports such as basketball, volleyball, and skiing (Fig. 24.24). It can also happen when a forceful knee

Fig. 24.24 An avulsion fracture of the tibial tubercle as a result of landing from a high ski jump in a 17-year-old male (**a**). CT and 3D CT reveal the extent of the injury (**b**, **c**). Tibial tubercle was reattached using pins (**d**)



extension abruptly stops due to kicking an object (e.g., block a punt in American football). On physical examination, the athlete is unable to extend his or her knee. Local tenderness is usually present and a knee effusion may be present. Diagnosis is usually made by plain radiography (Fig. 24.24). However, CT is usually performed to evaluate the extent of the injury (Fig. 24.24). Unless the fracture is nondisplaced, athletes with avulsion fracture of the tibial tuberosity require surgical intervention [60].

Tibial Eminence (Spine) Fractures

Tibial eminence is the insertion point for the ACL. Tibial eminence avulsion fractures are rare in sports and mainly occur among active pediatric population aged 8–17 years (Fig. 24.25) [61, 62]. In rare cases, it can occur in adults (Fig. 24.26) [63]. It is more common among males than females [61–63]. The mechanism of injury is usually from a fall while the knee is flexed (e.g., fall from a bicycle or fall while skiing) [61, 62]. Associated injuries such as meniscal tear, capsular strain, MCL and LCL sprain, and knee dislocations (Fig. 24.27) are common [61–63]. On physical examination, athlete has difficulty ambulating. Knee effusion is common. Lachman test may be positive. Diagnosis is usually made by plain radiography (Figs. 24.25, 24.26, and 24.27). CT and MRI imaging are recommended to evaluate the associated injuries [61–63]. Unless the fracture is nondisplaced, athletes with tibial eminence fracture should be referred to an orthopedic surgeon for possible surgical intervention [61–63].

Fig. 24.26 Tibial eminence (spine) avulsion (arrow) fracture in a 68-year-old female as a result of a ski injury

Fig. 24.25 Left tibial eminence (spine) avulsion fracture (arrow) and a Segond fracture (open arrow) in a 17-year-old male skier

Tibial Plateau Fractures

The knee is the largest joint in the body and the tibia is the major weight-bearing bone of the knee joint [46, 66]. To support this weight-bearing function, the tibia is wider proximally and forms the medial and lateral condyles [46]. These condylar portions are relatively flat and form the tibial plateau, which comprises its weight-bearing aspects. The region between the condyles called the intercondylar eminence serves as attachment sites for several soft tissues and ligaments such as the menisci and cruciate ligaments [46, 67]. The medial and lateral tibial plateaus articulate with the medial and lateral femoral condyles, respectively. The lateral tibial plateau is smaller and comparatively weaker than the larger, thicker, and thus stronger medial tibial plateau [46, 67]. Consequently, the lateral tibial plateau is more frequently fractured compared to the medial condyle [46, 67].

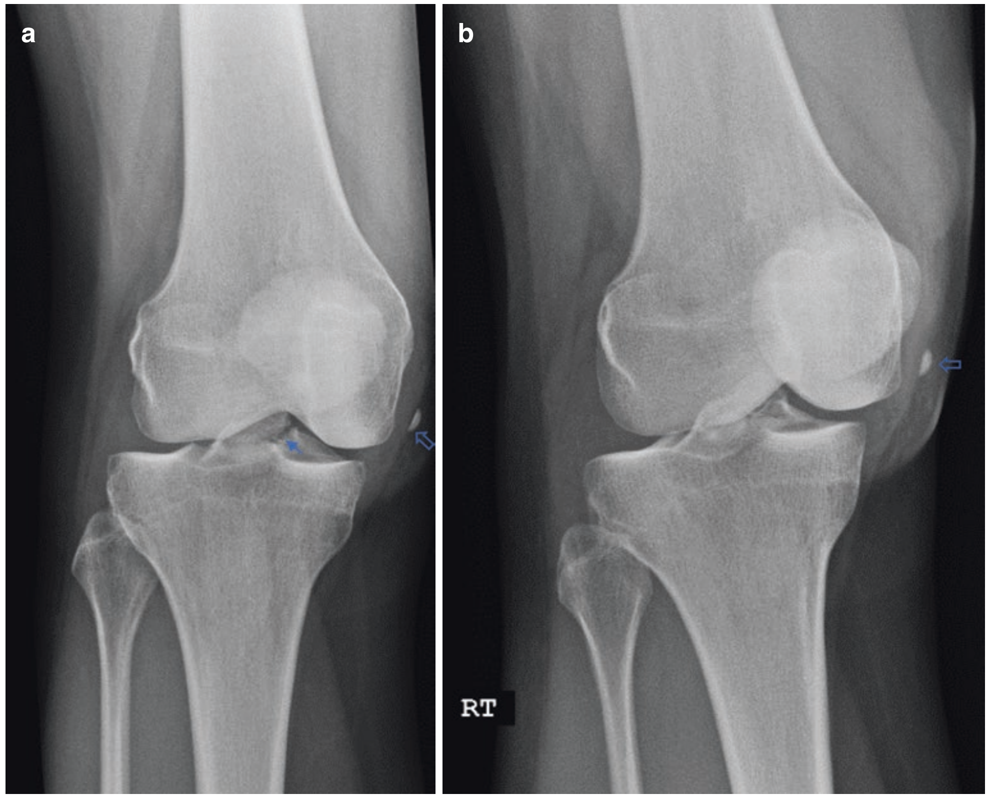


Fig. 24.27 A posterolateral tibia subluxation, tibial eminence (arrow), and medial epicondyle fracture (open arrow) in a 59-year-old female as a result of a ski injury (**a**, **b**)

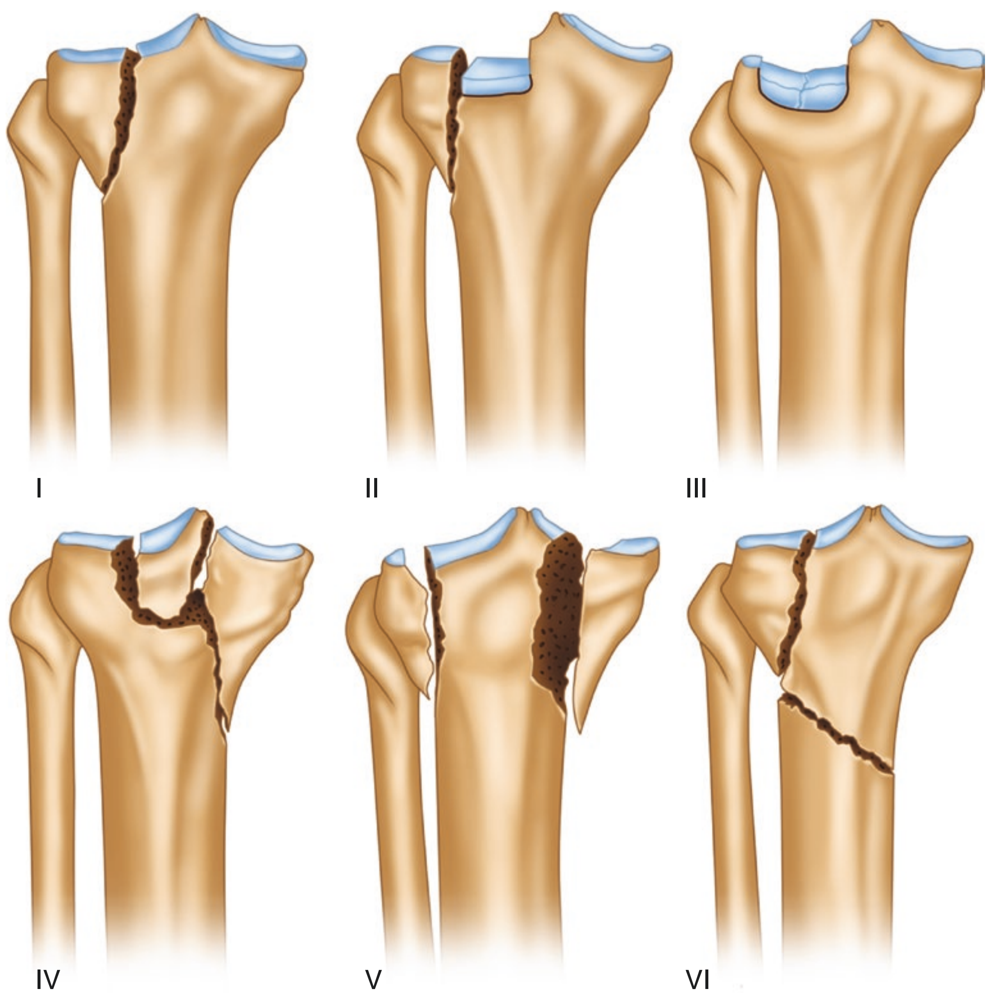


Fig. 24.28 The Schatzker classification for tibial plateau fractures [64]

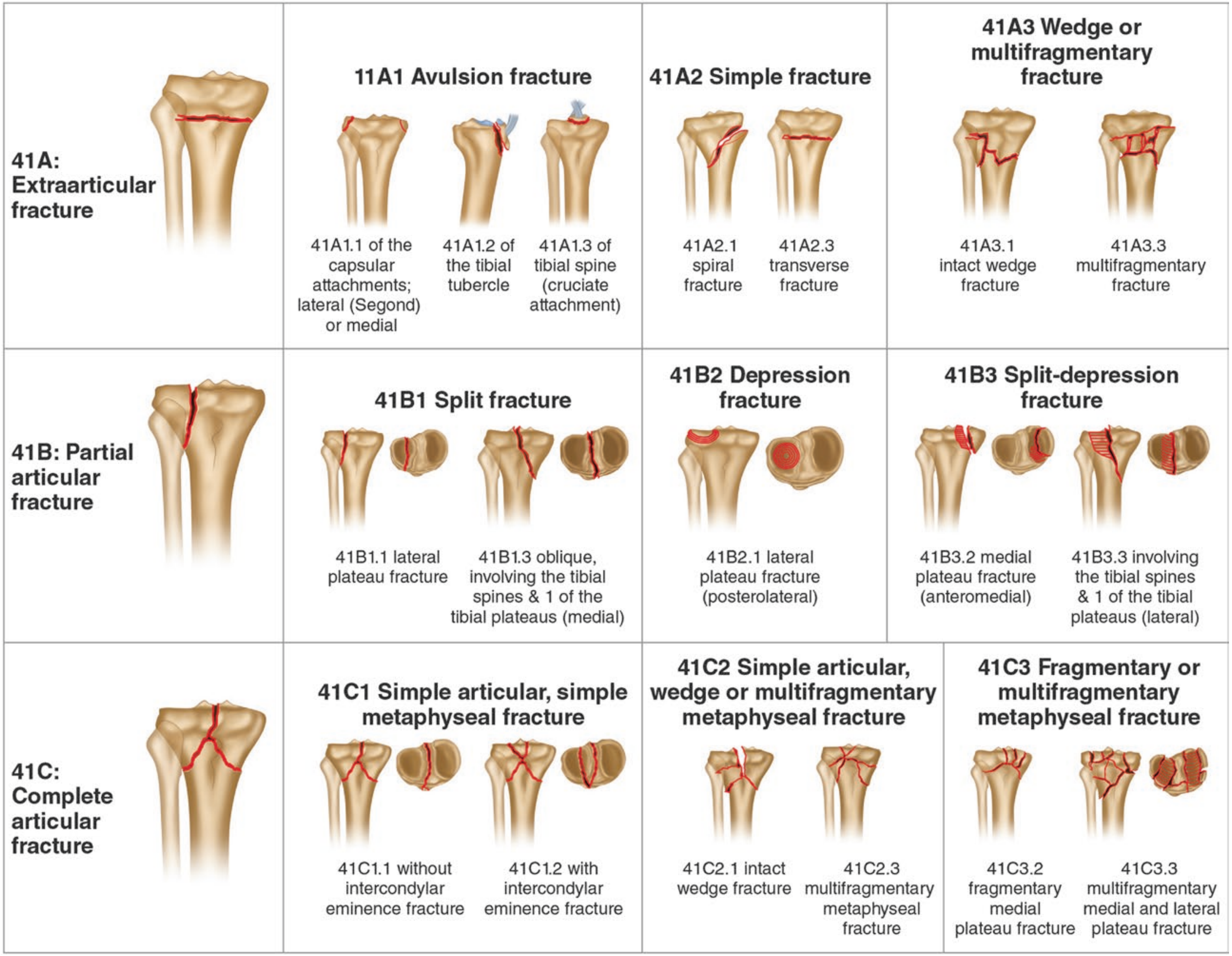


Fig. 24.29 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for diaphyseal and distal femur fractures. (Adapted from Ref. [65])

Mechanism of Injury in Sports

Because of its role in acting as one of the major weight-bearing bones, it is a very strong bone, and thus it is important to be cognizant that its fracture requires significant trauma [46, 67, 68]. These can comprise direct blows to the knee with forces directed medially, laterally, or axially including knee hyperextension injuries. In relation to sport, collision during contact sports involving hyperextension of the knee is the most common cause. In many cases, the medial or lateral femoral condyle acts as an anvil imparting a combination of both shearing and compressive force to the underlying tibial plateau leading to fracture of the tibial plateau [46, 67, 68].

Epidemiology

While tibial plateau fractures are not very common, accounting for only 1% of all fractures, they are a common long-bone fracture. They are also a source of significant morbidity accounting for >70,000 hospitalizations and 800,000 office visits [69]. Most of the tibial plateau fractures are related to motor vehicle accidents and falls, while 5–10% are sports related with the highest rates in skiers [46, 68].

Classification

The most common method of classifying tibial plateau fractures is the Schatzker system [46, 64]. This system divides tibial plateau fractures into six types based upon fracture fragment anatomy and fracture pattern (Fig. 24.28). Types IV, V, and VI are associated with higher-energy trauma and are more likely to result in compartment syndrome [46] (Table 24.2).

The AO/OTA classification for proximal tibia fractures groups them into extra-articular, partial articular fracture, and complete articular fracture (Fig. 24.29) [65]. They are also subclassified based on their displacement, comminution, and pattern of fracture [65].

Table 24.2 Schatzker classification system for tibial plateau fractures [64]

Type	Description
Type I	Wedge or split of lateral tibial plateau Usually results from valgus and axial forces No compression of underlying bone due to strong cancellous bone Seen in younger patients
Type II	Wedge or split of lateral tibial plateau with compression Similar mechanism as Type I but underlying bone may be osteoporotic More common in older patients
Type III	Pure compression fracture of lateral tibial plateau Results from axial force Depression is usually laterally or centrally but can occur anywhere
Type IV	Fracture of the medial plateau Results from varus or axial compressive forces Can include split, compression, or both Remember, medial tibial plateau fractures require stronger forces than lateral tibial plateau fractures
Type V	Split elements of both medial and lateral tibial condyles May also include medial or lateral articular compression Usually results from pure axial forces while knee is in extension
Type VI	Complex bicondylar fracture Condylar components separate from diaphysis Depression and compression of the fracture segments are required Requires the highest energy impact

Clinical Presentation

A patient with a tibial plateau fracture commonly presents with nonspecific findings and unclear history unless it was associated with a specific event such as skiing, soccer, fall, or motor vehicle accident [67]. They will have a painful and swollen knee with a history of injury in some traumatic event [67, 70]. Depending on the extent and type of the fracture, athlete may be able to bear weight on the affected leg [67, 70]. Despite nonspecific findings and unclear history, it is important to distinguish between high- and low-energy impacts. Injuries involving high-energy impacts have a greater likelihood of causing additional damage to neurovascular structures, menisci, ligaments, and compartment syndrome [67, 70]. Nerves and vessels that reside within the anterior and the deep posterior compartments can be damaged from significant swelling in these compartments and result in neurovascular compromise. Therefore, while examining the athlete with suspected tibial plateau fractures, it is important to attempt to examine these structures albeit swelling, spasm, and guarding may limit the accuracy of examination. In all cases, careful attention must be paid to peripheral pulses, neurological function, and the status of the compartments of the injured extremity [67, 70].

Diagnosis

Fractures of the proximal tibia occur most often from direct trauma to the region at or just distal to the knee. The diagnosis is usually made by plain radiographs. Standard radiographs for suspected proximal tibial fracture include anterior-posterior (AP), lateral, intercondylar notch, and oblique views (Figs. 24.30, and 24.31) [46, 67]. When clinical suspicion of fracture is high but plain radiographs are equivocal, CT scan better defines the fracture (Figs. 24.32, 24.33, and 24.34). It can also provide additional information regarding the extent and pattern of both articular and extra-articular components of the fracture [70]. MRI may demonstrate bone bruising and associated soft tissue injuries such as meniscus or ligamentous injury [71, 72]. While not important for diagnosis of the tibial plateau fracture, MRI is important for prognosis, management, and surgical planning for concomitant injuries often associated with tibial plateau fractures.

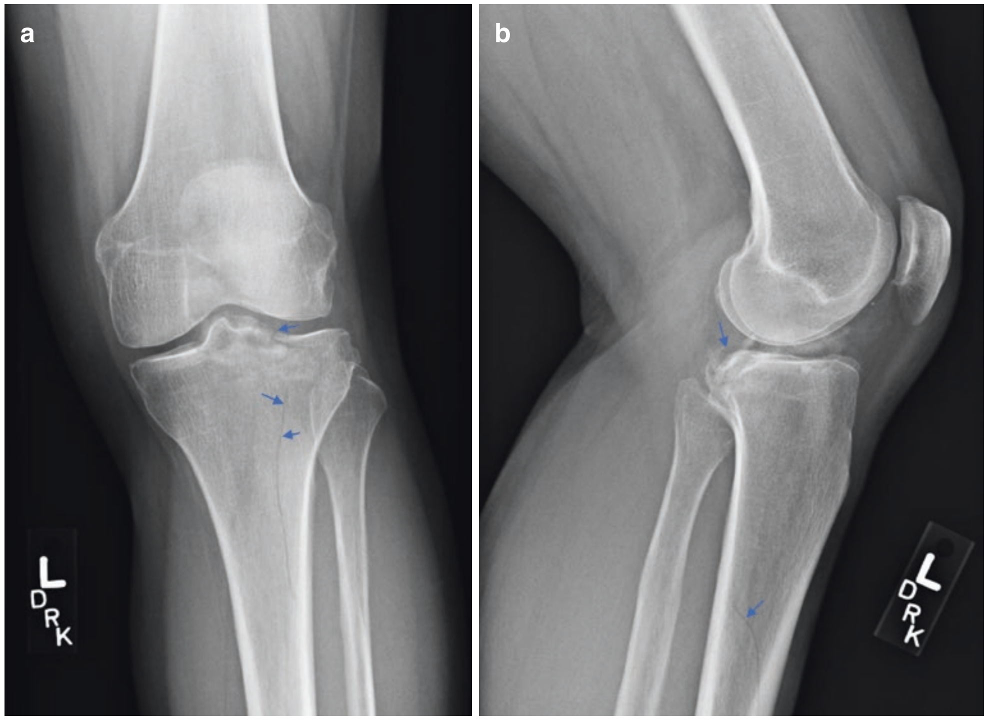


Fig. 24.30 A nondisplaced, intra-articular proximal tibia (lateral plateau) fracture in a 67-year-old female (**a**, **b**)

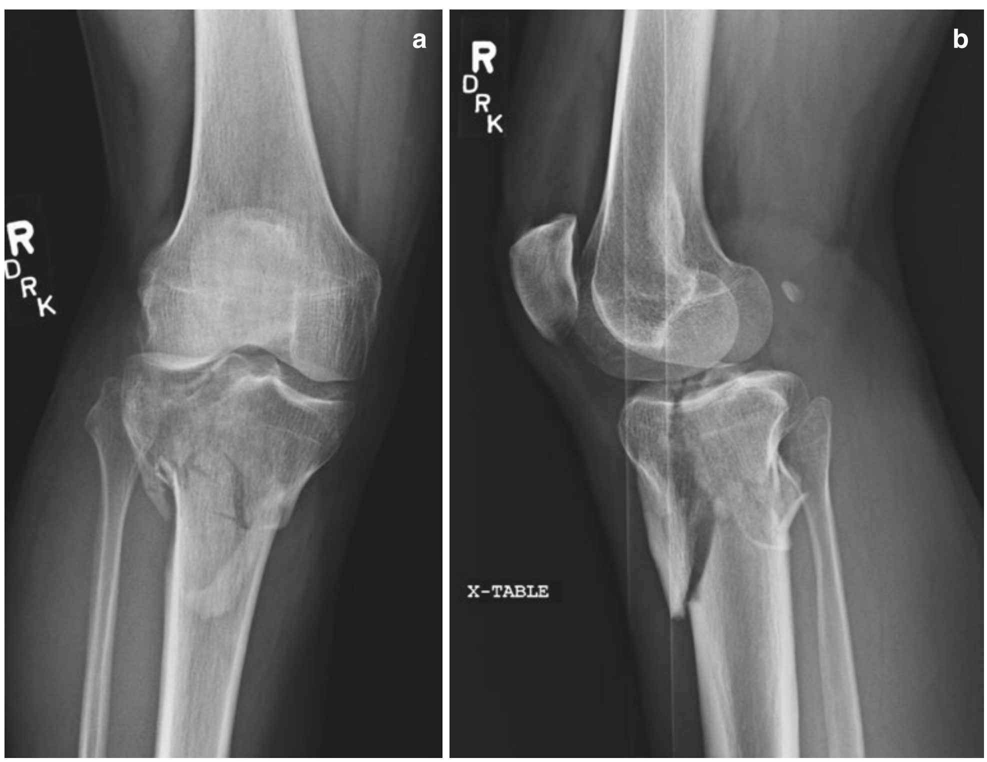


Fig. 24.31 A displaced, comminuted, and intra-articular proximal tibia fracture in a 38-year-old male (**a**, **b**)

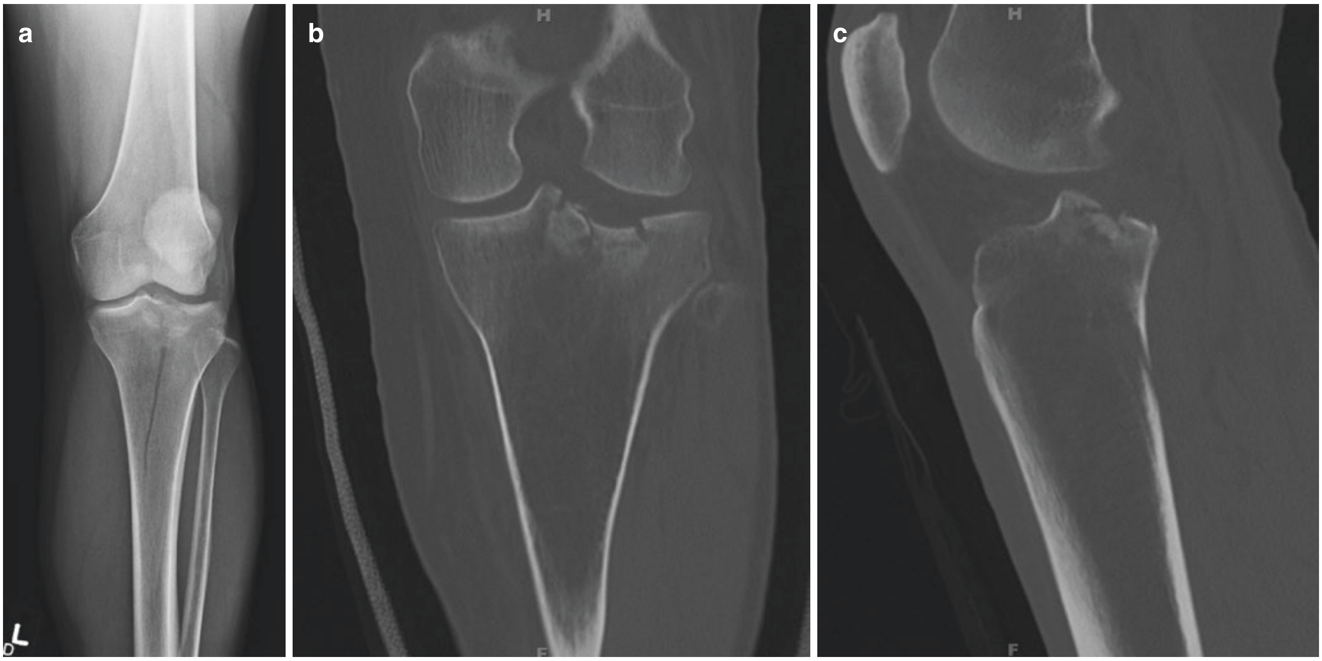


Fig. 24.32 A nondisplaced proximal tibia (lateral plateau) fracture in a 29-year-old male as a result of a ski injury (**a**–**c**)

Initial Management

The initial management of all tibial plateau fractures includes ice, compression, analgesia, splinting of the knee in near-full extension, elevation of the leg above heart level, and strict non-weight-bearing status.

Indications for Orthopedic Referral

Patients with open fractures, neurovascular compromise, extensive intra-articular, or multifragmentary metaphyseal fracture (e.g., Schatzker ≥IV or AO/OTA 41C3) should be immediately transported to higher level of care with possibility of surgical intervention [46, 67, 69–71].

The majority of tibial plateau fractures require surgical treatment with open reduction and internal fixation (Figs. 24.35, and 24.36) [46, 67–71]. Lastly, an orthopedic surgeon should evaluate fractures with any degree of displacement, compression, and those associated with high-energy trauma.

Follow-Up Care

Fractures that are stable in extension and are minimally displaced may be responsive to cast immobilization or bracing with early range of motion and delayed weight-bearing as tolerated [46, 67, 69–71]. In addition, nondisplaced fractures in low-demand individuals such as elderly and osteoporotic patients may be treated nonoperatively [46, 67, 69–71].

All athletes undergoing closed treatment should ideally have plain radiographs performed every 2 weeks for the first 6 weeks to ensure appropriate alignment and healing [46, 67, 69, 70]. Varied protocols exist for repetitive imaging during the initial phase. Another approach is to repeat plain radiographs every week for 3 weeks and then on a 2–3-week basis depending on radiographic appearance [46, 67, 69, 70]. Activities should be restricted for 4–6 months [21]. Partial weight-bearing in the brace can begin once there is evidence of adequate radiographic healing demonstrated by the presence of bone callus [46, 67, 69, 70]. After brace fitting, the patient returns weekly for the first 3 weeks following injury. If there is no displacement at 2 weeks, the patient begins working on knee flexion in the brace with a goal of achieving 90° by 4 weeks [46, 67, 69, 70].

Return to Sports

Athlete should have almost complete range of motion of the knee and be pain free to start the return-to-sports protocol. As with most injuries, once the affected extremity has dem-

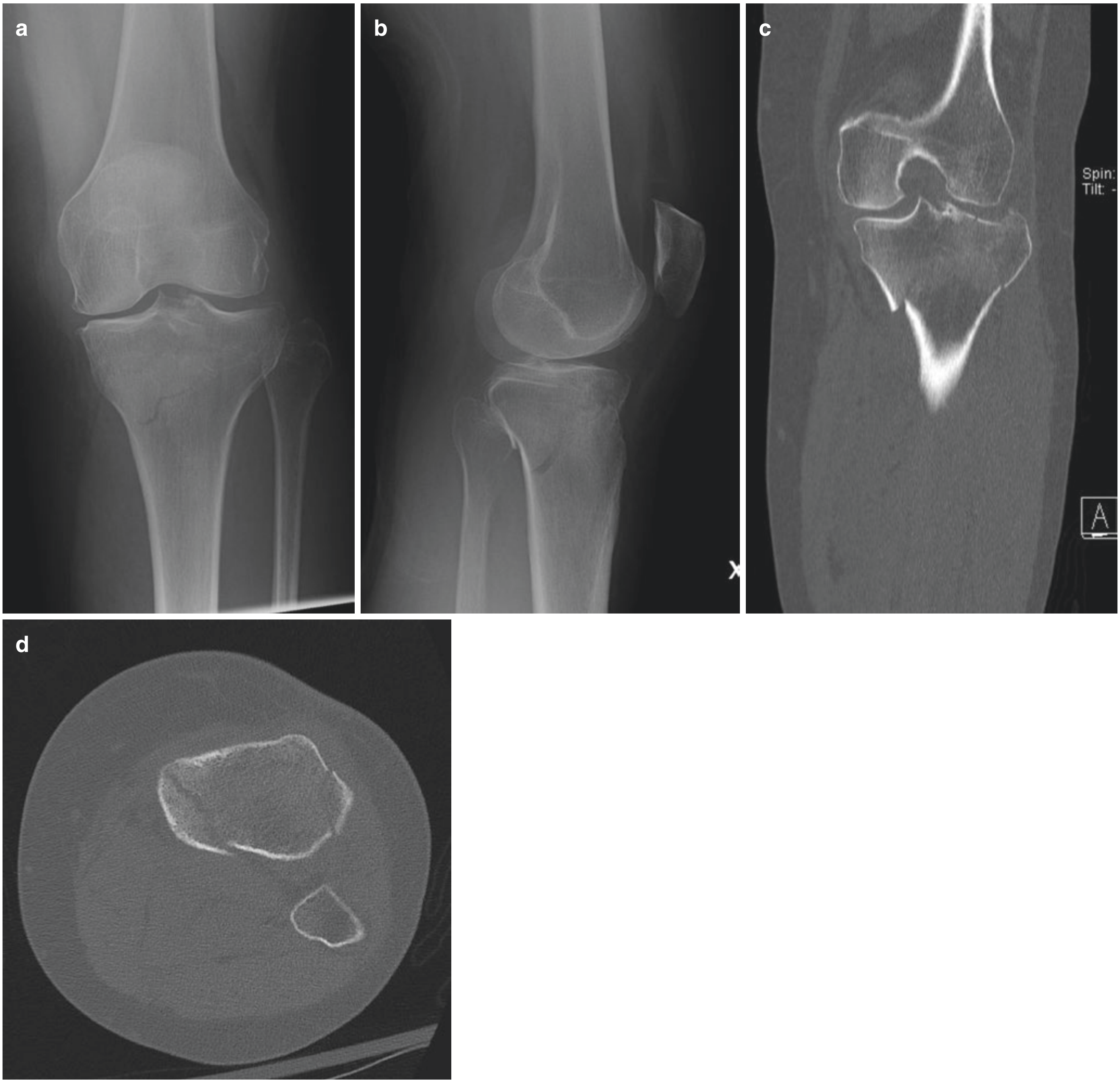


Fig. 24.33 A mildly displaced proximal intra-articular medial and lateral tibia fracture in a 62-year-old female as a result of a car accident (**a**, **b**). CT scan reveals the extent of the injury (**c**, **d**)

onstrated about 90% of its strength and function compared to the unaffected extremity, an athlete may begin sport-specific rehabilitation.

Complications

A major long-term complication of tibial plateau fractures is the development of osteoarthritis of the affected knee [73]. In a large population-based cohort study, patients treated surgically for tibial plateau fractures have an increased likelihood of total knee replacement compared to control group (LR 5.3, 95% CI 4.58–6.11) [74]. At 10 years, 7.3% of the patients required total knee replacement [74]. As discussed previously, acute compartment syndrome is the most common, significant short-term complication associated with tibial fractures [46, 67, 70]. This is a limb-threatening emergency, and immediate orthopedic consultation is required when the diagnosis is suspected. Due to the long-term immobility, patients with tibial plateau fractures are at increased risk for venous thromboembolic events (VTE) such as DVT and PE.

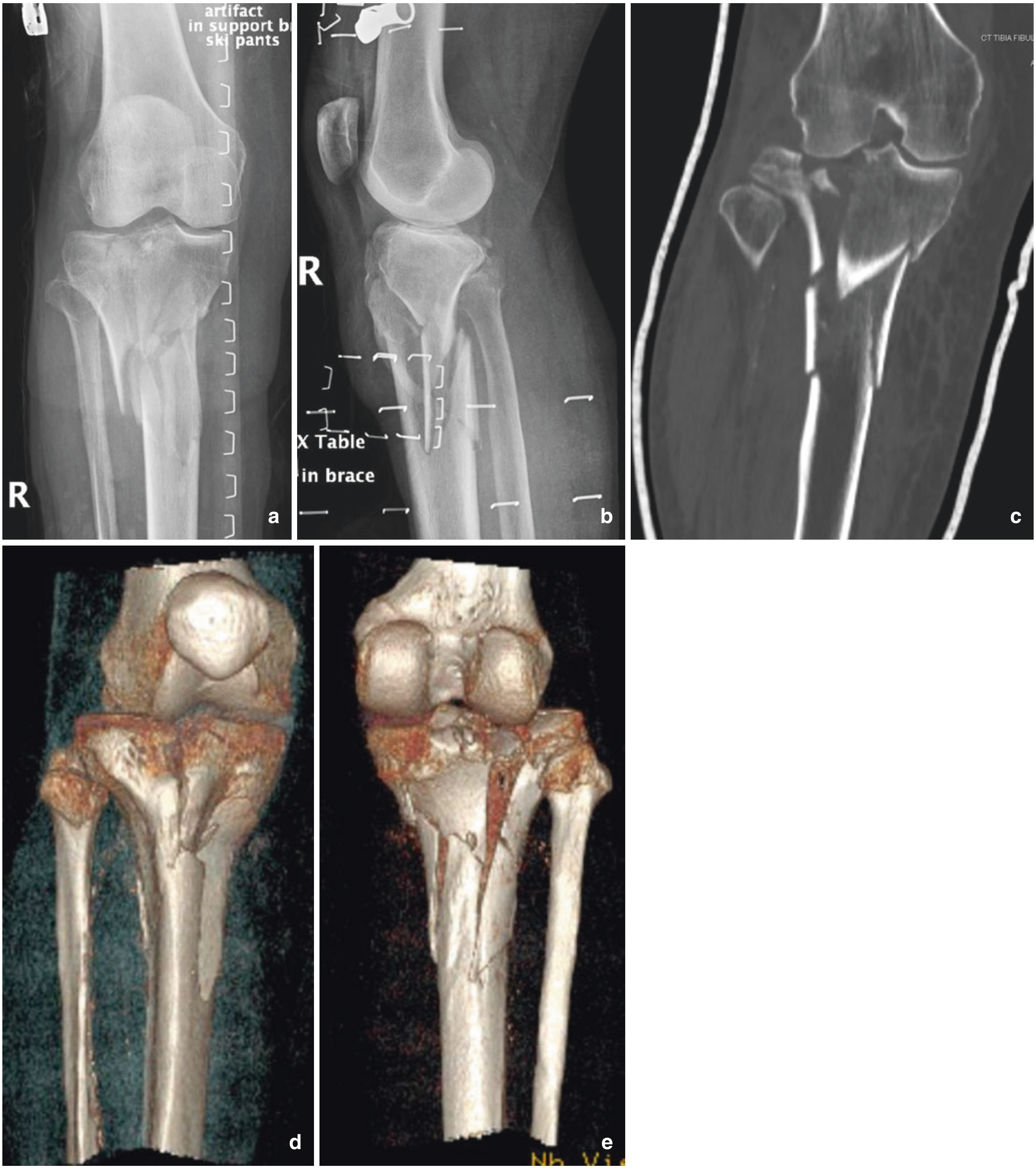


Fig. 24.34 A displaced, intra-articular, and comminuted proximal tibia (medial and lateral plateau) fracture in a 75-year-old female as a result of a ski injury (**a**, **b**). CT and 3D CT reveal the extent of the injury (**c**–**e**)

Pediatric Considerations

Fractures of the proximal tibia are uncommon in children. They include intra-articular fractures, physeal fractures, apophyseal avulsion fractures, and proximal tibial metaphysis greenstick fractures. Hyperextension is the main cause of proximal tibial physis fractures [75]. Avulsion fracture of the tibial tubercle is usually caused by a forceful knee extension mechanism such as landing from a height (Fig. 24.24). Intra-

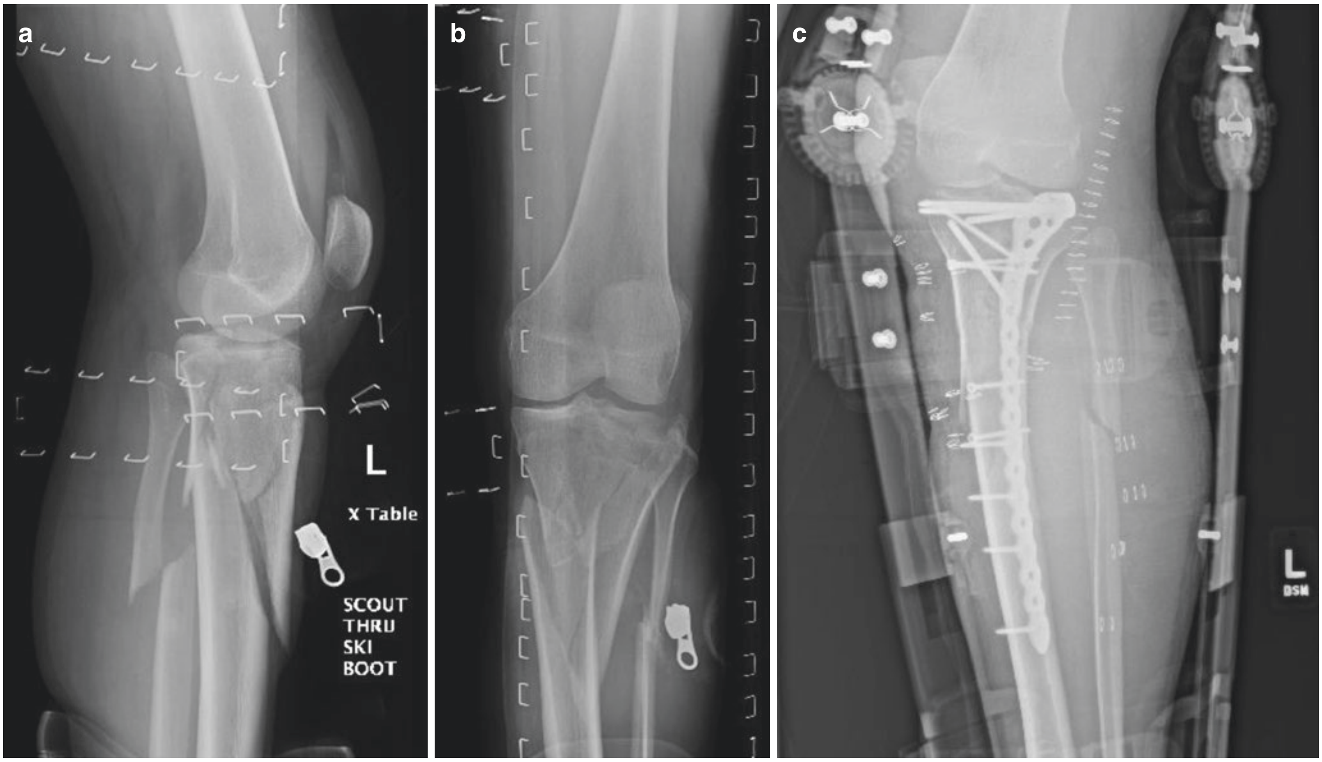


Fig. 24.35 Intra-articular, displaced, and comminuted proximal tibia and fibula fractures in a 24-year-old male skier (**a**, **b**). Postoperative image shows near-normal alignment (**c**)

Fig. 24.36 Intra-articular, displaced, and comminuted proximal tibia (lateral plateau) fracture in a 68-year-old male as a result of a ski injury (**a**). CT scan reveals the extent of the fracture (**b**, **c**). Postoperative image shows near-normal alignment (**d**)

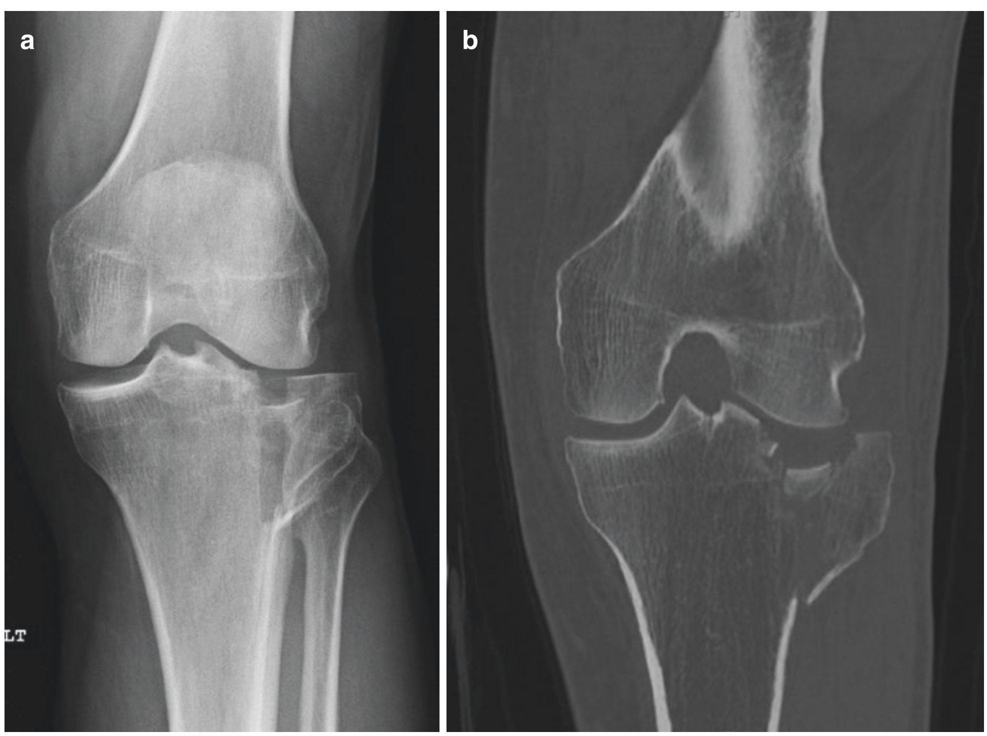
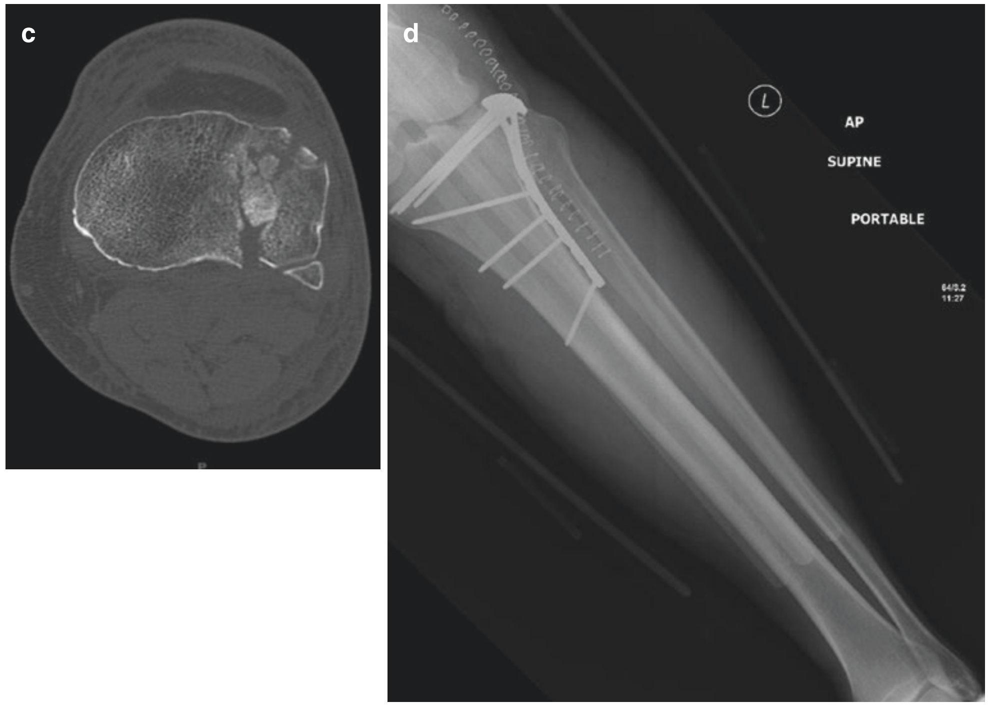


Fig. 24.36 (continued)



articular tibial plateau fractures are rare and usually from high-energy axial loading of the knee such as high-energy sports [75]. Children with femoral condyle fracture should be managed similar to adults with initially following the ATLS algorithm.

Proximal Fibula Fracture

Isolated proximal fibula fractures are rare in sports. Proximal fibula fractures often occur in combination with tibial plateau or distal tibia (Maisonneuve) fractures. Most isolated fractures are nondisplaced (Fig. 24.37) or are avulsion fractures. The mechanism of injury is usually a direct trauma. Management is similar to fibular shaft fractures (Chap. 25). Clinicians should be cognizant of possible associated neurovascular injuries.

Dislocations Around the Knee

Knee (Tibiofemoral) Dislocation

Mechanism of Injury in Sports

Knee dislocations are defined by disruption of the tibiofemoral articulation. The majority of knee dislocations occur as a result of high-velocity injuries, such as motor vehicle accidents. However, they are becoming more frequent in sports, such as skiing, football, and soccer [76–80]. Injury occurs as a result of hyperextension or extreme pivot-shift maneuvers [81, 82]. They also occur with direct traumatic contact or falls [83].

Epidemiology

Knee dislocation is an uncommon injury, occurring during sports at a rate of 29 per one million person-years [84]. They commonly occur to males in the second or third decade of life [76, 78, 80, 85, 86]. The majority of knee dislocations occur in winter sports with the highest risk in skiing (Fig. 24.38); however, there are an increasing number of cases in football and soccer [77–80, 84]. Vascular and neurologic injuries frequently accompany knee dislocations, particularly with injuries to two or more ligaments. A high suspicion and concern for neurovascular injury is essential as more than half of knee dislocations reduce spontaneously and may not be evident on initial examination [16, 86]. Injuries to other intra-articular structures are common with the meniscus being involved in 37–55% and cartilage in 28–48% of dislocations [76, 78, 87].

Classification

Historically, knee (tibiofemoral) dislocations were classified according to the amount of tibial displacement in relation to the femur. As a result, there were five classifications: ante-

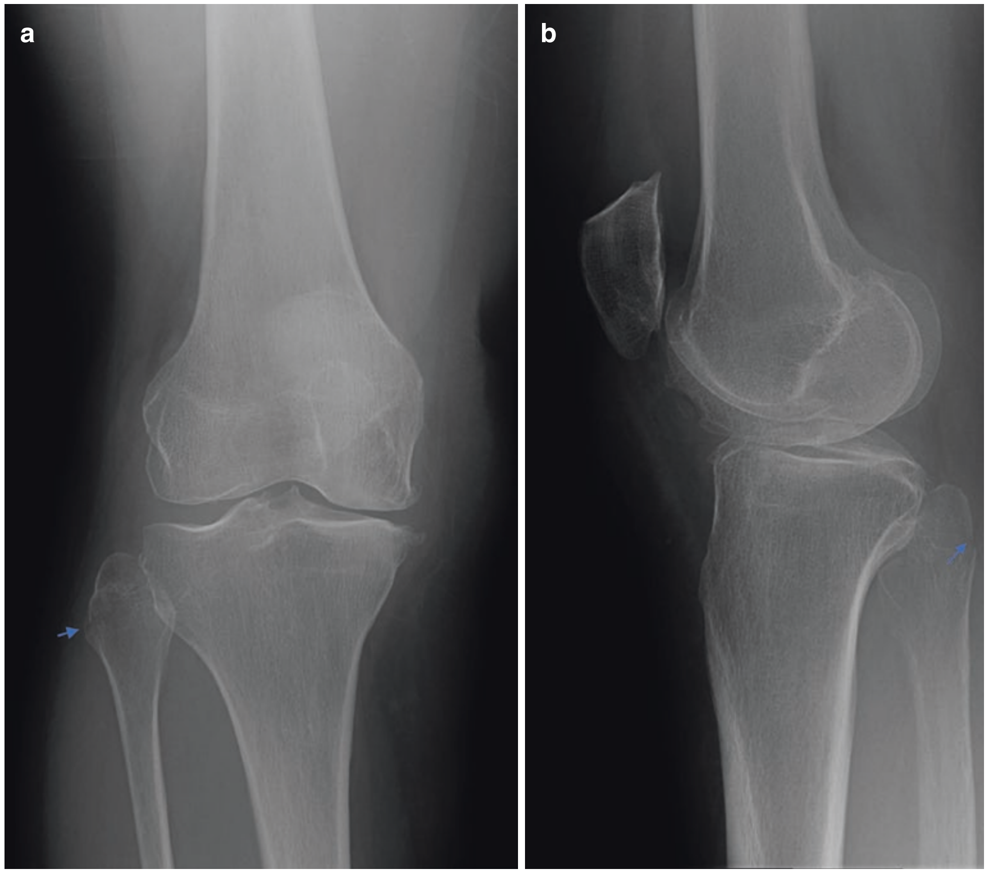


Fig. 24.37 An isolated nondisplaced proximal fibula fracture (arrows) in a 62-year-old female as a result of a direct trauma as a pedestrian in a car accident (**a**, **b**)

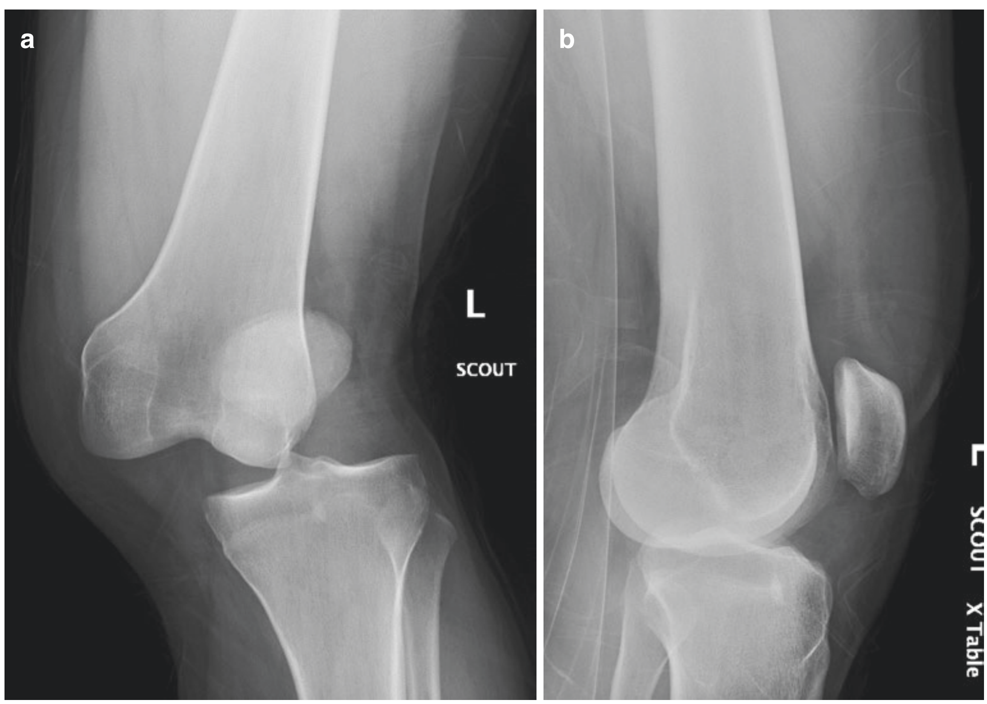
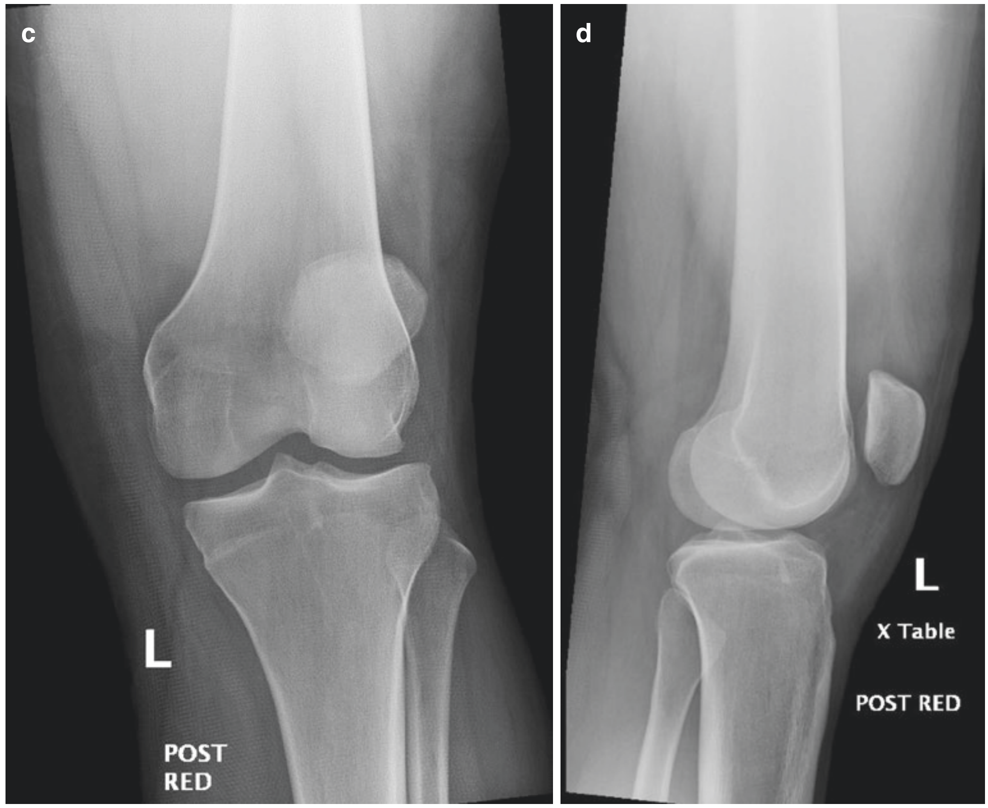


Fig. 24.38 Left knee dislocation (**a**, **b**) in a 26-year-old male as a result of a ski injury. Postreduction images show normal bony alignment (**c**, **d**)

Fig. 24.38 (continued)



rior, posterior, medial, lateral, and rotatory. Of these, anterior dislocation are the most common followed by posterior dislocation [81, 88]. This directional classification system was highly limited, especially since it did not account for spontaneous reductions, associated knee structure injuries, or risk of local vascular or neurologic injury [86]. Consequently, Schenck developed a new and now more widely accepted classification system [89]. In his system, knee dislocations (KD) are categorized by number of knee ligaments disrupted. KD I indicates a single cruciate ligament injury, which is rare, while KD II is a bicruciate ligament injury [80]. If one collateral ligament is injured with a bicruciate injury, it is classified as KD III, followed by an M (MCL) or L (LCL) indicating laterality of medial or lateral collateral ligament disruption. A KD IV signifies a bicruciate and bilateral (MCL and LCL) collateral ligament injury. Lastly, KD V is a knee dislocation with periarticular fracture [86, 89]. The most common type of knee dislocations is KD III-M at 52% followed by KD III-L at 28% [78].

Clinical Presentation

Dislocations of the knee with gross deformity are obvious; however, the majority of knee dislocations reduce spontaneously; therefore, high suspicion is needed with any multi-ligament knee injury [86, 90]. The athlete will likely have immediate noticeable effusion given intra-articular injury, but hemarthrosis is dependent upon the degree of capsular disruption and may not always be apparent on physical examination [91]. The vast majorities of knee dislocations are closed and most commonly occur with injury to three ligaments, KD III [78]. A careful neurovascular examination must be completed, as knee dislocations are associated with nerve injuries in 14–25% and vascular injuries in 5–22% of cases [76, 78, 84, 92]. The most common nerve injured is to the common peroneal nerve running behind the fibular head with patients presenting with motor dysfunction as loss of ankle dorsiflexion, foot drop, and sensory dysfunction as numbness to anterior lower lateral leg and dorsal surface of the foot [76, 92, 93]. The most common vascular injury is to the popliteal artery with patients showing vascular compromise signs of absent pulses, expanding hematoma, hemorrhage, or bruit [94]. Neurovascular injuries typically occur together, but can happen in isolation, and most often occur with KD III-L or posterolateral corner injuries [78, 92]. Of those with vascular injuries, 80% will need surgery and 5–20% might require amputation [76, 92, 95].

Diagnosis

Given that the majority of knee dislocations reduce spontaneously, the diagnosis must be considered whenever two or more ligaments are injured, especially bicruciate injuries. Prompt recognition and appropriate management is crucial as this can be a limb-threatening injury [86]. If available, immediate AP x-ray should be obtained to assess direction of dislocation, likelihood of successful reduction, and any associated fractures (Figs. 24.27, 24.38, and 24.39) [90]. In any instance of reduction, if possible, x-rays should be performed before and after reduction to assess alignment (Fig. 24.38) [96]. Further neurovascular examination is warranted after realignment in any dislocation. In acute setting, vascularity should be prioritized [91, 92]. While the gold standard for vascular assessment is angiography (Fig. 24.39), there is a debate as to whether angiography should be performed on all knee dislocations. Many argue angiography should be done on all knee dislocations given possibility of undetected intimal tears, even with normal distal pulses, as patient are at high risk of amputation if diagnosis is missed [16, 90, 92]. Given the invasiveness of angiograms, cost, risk of iatrogenic injury, and contrast exposure, others recommend that a selective angiography algorithm based on pulses and ankle-brachial indexes (ABI) is more appropriate [92, 94, 97]. A vascular algorithm develop by Nicandri et al. recommends immediate intraoperative angiogram with any hard vascular signs such as absent pulses, expanding hematoma, hemorrhage, or bruit [94]. Without hard physical signs, check ABI, and if <0.90, then proceed to angiography to evaluate for vascular compromise. However, if the patient has palpable pulses and ABI >0.90, there is high sensitivity that vasculature is intact and no immediate angiography is warranted. In these patients, a minimum of 24-hour monitoring with frequent neurovascular checks is necessary [94]. When clinically stable, MRI of the knee should be performed to fully assess boney, ligamentous, and cartilage injuries and to help direct further treatment [90, 98].

Initial Management

The approach to a dislocated knee should be to check neurovascular status, obtain x-rays (if possible), and attempt reduction [79]. If an on-field dislocation is suspected and there is no gross deformity, varus and valgus testing at full extension should be performed. If gapping is appreciated, it is indicative of probable dislocation, encompassing a capsular injury with at least 1 collateral and 1 cruciate ligament injury [16, 94]. At this point, assessment for neurovascular injury is the priority. In the event of absent distal pulses, immediate reduction should be attempted to restore circulation [90, 94]. This is accomplished via gentle inline traction with attempted knee extension [91]. If distal pulses are palpable, there is no consensus that on-field reduction should be attempted. Some believe imaging should be obtained first and any required reductions should be performed under anesthesia [16, 79, 96]. Prereduction imaging often aids in the identification of an irreducible knee dislocation. On physical examination, an irreducible knee may be appreciated if you have difficulty with an attempted reduction or notice a defect or “buttonhole” underneath the medial femoral condyle [99]. Irreducible dislocations require intraoperative reduction under anesthesia; this occurs in about 13% of knee dislocations [85]. After any reduction or attempted reduction, a neurovascular exam should be performed to reevaluate for any change [16]. The affected leg should be splinted or externally fixated and immediately transferred to hospital. Strength testing should not be performed as it can cause further damage to local structures [83]. In the emergency room, imaging and continued monitoring of vascular status should be prioritized as described above. If no emergent surgery is indicated, the knee should be immobilized in a hinge brace between 15° and 20° of flexion, to prevent re-subluxation or dislocation, with every 2-hour vascular checks for 48–72 hours [79, 82, 94, 96]. Initial management of a knee dislocation centers on identification, successful reduction, temporarily stabilization, appropriate evaluation, and repeat neurovascular examinations [82]. Then further imaging, such as MRI, and other special tests to check integrity of surrounding knee structures can be conducted.

Indications for Referral to Orthopedic Surgery

Given the high risk of neurovascular compromise, all knee dislocations should be reduced and admitted to the hospital for serial examinations. There should be direct communication to the emergency department to indicate probability of tibiofemoral dislocation as well as need for vascular investigation. Emergent surgery is required if there is an irreducible knee, vascular compromise, compartment syndrome, extensor mechanism ruptures, displaced menisci, displaced fractures, or lateral sided rupture of structures that need acute repair such as the iliotibial band, biceps femoris, or lateral collateral ligament [96, 100]. If vascular injury has occurred, a vascular surgeon should conduct intraoperative angiography and repair within 6–8 hours to limit the risk of amputation [88, 94].

Follow-Up Care

After passing of the acute emergent period, the full extent of injury can be appreciated and definitive treatment plan established. While there is an ongoing debate over surgical approach, management is primarily dictated by the injured structures, and operative management has been proven to have better outcomes than nonoperative care, especially in the young, athletic population [79, 82, 83, 91, 96, 100]. Controversies over surgical approach include repair versus reconstruction, acute versus delayed treatment, single versus staged surgeries, arthroscopic versus open treatments, and use of allografts [82].

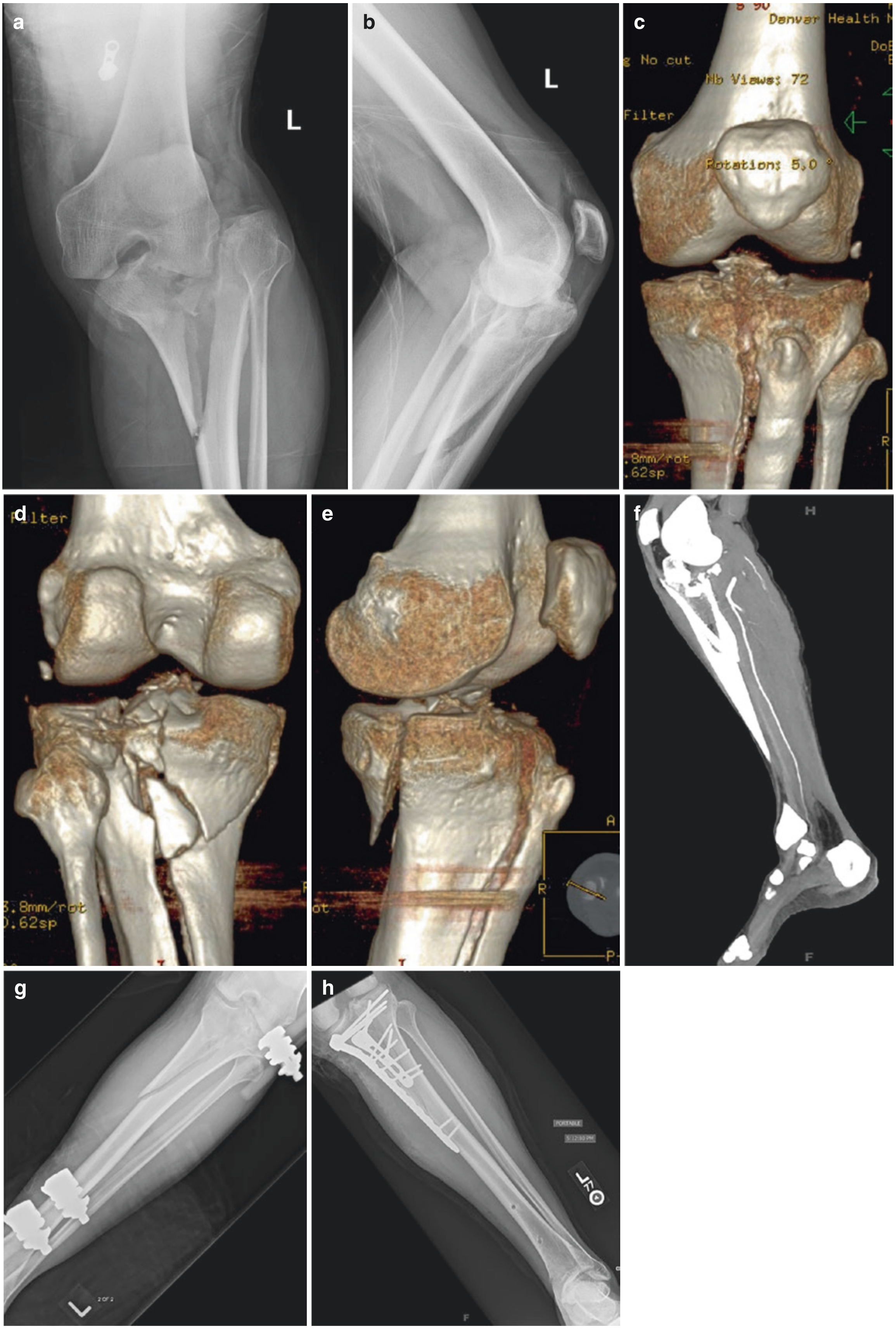


Fig. 24.39 Knee dislocation associated with displaced, comminuted, and intra-articular proximal tibia fracture in a 47-year-old male as a result of a snowboarding injury (**a**, **b**). Postreduction 3D CT images show the extent of the injury (**c**–**e**). CT angiogram did not show any arterial injuries (**f**). Proximal tibia fracture was treated with internal fixation (**g**) first and then with ORIF (**h**)

Neurologic outcomes must also be considered in all knee dislocations, with the highest occurrence in KD III-L and posterolateral corner injuries [78]. While the majority (87%) of patients with incomplete common peroneal palsy will recover, injury to the common peroneal nerve with persistent foot drop has worse functional outcomes [91, 93]. Treatment includes physical therapy with a focus on ankle foot orthosis and range of motion rehabilitation. Only 38% of those experiencing full common peroneal palsy will recover, and these patients should be monitored with nerve conduction studies at 6 weeks and 3 months post-injury. Surgery should be offered to those with a poor prognosis, typically posterior tibial tendon transfer [93].

Return to Sports

A study by Hirschmann et al. looked at long-term outcomes and return to sport in surgically repaired athletes, who showed better outcomes for early, open complete single-stage reconstruction within 40 days of injury [77]. Additionally, KD III-M cases do better than KD III-L in the long term. Overall, almost 80% of athletes returned to sport, but only 33% returned to preinjury baseline level [77]. After initial management, an intense, graded rehabilitation course such as tiered treatment plan is indicated [83].

Complications

Complications of knee dislocations are dependent not only upon the initial extent of injury but also upon the treatment and management of injury. Conservative treatment places the patient at risk of an unstable knee joint with limitations to return to play. Patients who elect for surgical management most commonly complain of chronic joint stiffness and pain [100]. As most knee dislocations are treated surgically, the highest long-term complications are reduced range of motion, with a flexion deficit, and chronic pain [77, 101]. The majority of patients can expect to return to activities of daily living; however, return to sport is more variable depending on specific associated injuries [76, 82].

Pediatric Considerations

The majority of knee dislocations, which includes high-velocity traumas, occur in young individuals in ages between 20 and 30 years [78–80, 85, 86]. However, in sports the majority of knee dislocations occur at a younger age between 10 and 19 years [84]. Therefore, suspicion should be increased in high-velocity or high-impact sporting events. Regardless, there is no change in management based on age.

Patellofemoral Dislocation

Mechanism of Injury in Sports

Patellofemoral or patellar dislocations occur when the patella disarticulates with the trochlear groove of the femur; this most commonly occurs laterally [102]. The majority of patellar dislocations occur in sports [103–105]. Dislocations can occur with or without contact; however, noncontact mechanisms are significantly more common, accounting for up to 93% of dislocations [105]. Contact or trauma dislocations encompass falls on a flexed knee or collision with a lateral force onto the patella [105]. Noncontact dislocations occur when a planted flexed knee has a valgus force applied, typically causing asymmetric quadriceps contraction and a laterally displaced patella [105, 106]. Patellar subluxation is similar to patellar dislocation [107]; however, it requires less force and often happens in individuals with patellar instability. It usually self-reduces which makes the diagnosis challenging [108].

Epidemiology

Patellar dislocations most commonly occur in cutting or pivoting sports and dance with an annual incidence of 23–42 per 100,000 person-years [103–105, 109]. Females between the ages of 10–17 years old have the highest risk of patellar dislocations; however, the overall annual incidence is equal between males and females [103–105, 109–112]. The average age for first dislocation is between 18 and 21 years old [103, 106, 109, 112–114]. The overall re-dislocation rate is about 30%, again with the highest reoccurrence in females 10–17 years old at 36% [109, 110, 113, 115]. The most common risk factors for patellar dislocations are trochlear dysplasia, patella alta, and age less than 18 years old [104, 105, 108, 109, 111, 116].

Classification

Patellar dislocations almost always occur laterally and are the focus of this section [102]. Lateral patellar dislocations can occur with or without contact [105]. Medial, superior, and intra-articular dislocations are rare [102, 117]. A nonlaterally displaced patella is iatrogenic, traumatic, or secondary to degenerative changes [102, 117]. As the patella dislocates laterally, it typically results in injury to the medial stabilizing ligaments (up to 63% of dislocations) such as the medial retinaculum and medial patellofemoral ligament [103, 113]. The next most common associated injury is chondral defects, found in up to 44% of dislocations [103, 109, 112, 113]. The chondral injury more commonly occurs on the patella than the femur [113]. There are also smaller associated injuries, which include meniscal tears in up to 4% and ACL tears in 1% [105, 113].

Clinical Presentation

An on-field patellar dislocation presents with an obvious laterally displaced patella deformity usually in a flexed knee (Fig. 24.40). There will likely be immediate effusion and tenderness around the medial retinaculum. Reduction should be attempted, and if successful, all other knee ligaments should be assessed by physical examination [118]. Palpation should also focus on any noticeable defects in the vastus medialis or adductor muscles [119]. If a dislocation presents in your office after reduction, a comprehensive knee examination should be completed focusing on patellar hypermobil-

Fig. 24.40 Left lateral patellar dislocation in a 44-year-old female as a result of a ski injury (**a**, **c**, **e**). Postreduction images show normal alignment (**b**, **d**, **f**)

ity and apprehension with medial and lateral movements bilaterally [119]. The majority of dislocations, especially if traumatic, will present with effusion or hemarthrosis [103, 105, 118, 120, 121]. Patellar dislocations are the second most common cause of traumatic hemarthrosis, only behind ACL injuries [120].

Diagnosis

Diagnosis of a patellar dislocation can be accomplished through careful history and physical examination. History will typically describe the patellar dislocation and requisite relocation. The physical examination, as described above, will be most significant for effusion, patellar

apprehension, and medial retinaculum tenderness [119]. To confirm a suspected diagnosis, further imaging should be obtained. Initial radiographs should include AP, lateral, and sunrise (Figs. 24.40, and 24.41) [112, 119, 121]. Plain radiography should focus on patellar alignment as well as reveal osteochondral fragments [112]. However, radiographs have limited ability to correctly identify osteochondral fragments for which MRI provides better diagnostic performance [122]. Therefore, there is a debate on obtaining an MRI for first-time patellar dislocations. One perspective proposes all first-time dislocations require MRI to rule out osteochondral defect, while others suggest MRI only if there is high likelihood of defect as seen with hemarthrosis or mechanical symptoms [112, 119, 121, 122]. Typical MRI findings after lateral patellar dislocation include injury to the medial patellofemoral ligament as well as marrow edema in the anterolateral femoral condyle and inferomedial patella [105, 122].

Initial Management

If an on-field patellar dislocation is identified, the first step should be attempted reduction. Reduction is accomplished with medial patellar pressure, to manipulate the patella over the lateral femoral condyle, while gently extending the leg [123]. Once reduced, the athlete's knee can be immobilized and crutches can be used to weight-bear as tolerated [103]. Postreduction radiographs should be obtained to confirm normal alignment and evaluate associated injuries (Figs. 24.40 and 24.41). If reduction cannot be accomplished, then the athlete should be sent to the emergency department for imaging and orthopedic evaluation for reduction under sedation or in the operating room.

Indications for Orthopedic Referral

First-time patellar dislocations should be treated conservatively [112, 118, 119]. However, there are several indications for surgical intervention after any patellar dislocation. These include osteochondral fracture or major chondral lesion, substantial disruption of the medial patellar stabilizers (medial

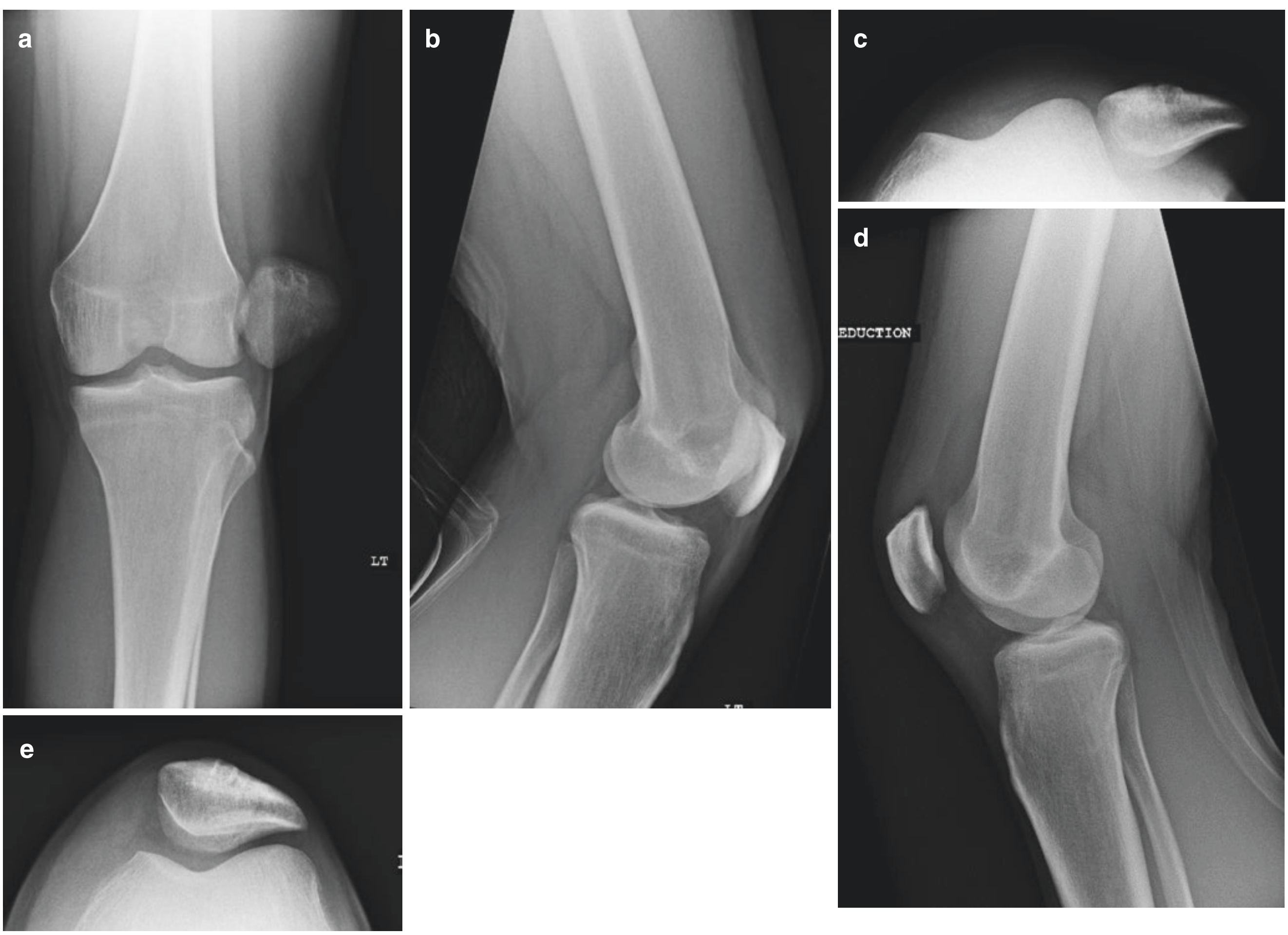


Fig. 24.41 Left patellar dislocation in a 24-year-old male as a result of a ski injury (**a**–**c**). Postreduction images show normal alignment without any obvious fracture (**d**, **e**)

patellofemoral ligament, medial retinaculum, vastus medialis, or adductor mechanism), a laterally subluxed patella in normal alignment in contralateral knee, or failed improvement with appropriate rehabilitation [112, 118, 119]. These indications stress the importance of initial physical exam and appropriate imaging to best direct care [113]. If this is not a first-time dislocation and the athlete is having recurrent patellar instability, this is also an appropriate time to discuss surgical interventions, but there are no clear guidelines on when or how many dislocations warrant transition to surgical treatment [115].

Follow-Up Care

In office follow-up care of a reduced patellar dislocation without surgical indications focuses on protected rest to allow healing for 2–4 weeks followed by early rehabilitation [103, 105, 112, 115, 118]. If an effusion is present, this can be drained for joint decompression, improved examination, and patient comfort as well as indicate need for further imaging if hemarthrosis is present [112, 118, 121]. The majority of studies favor a short period of immobilization in knee extension with progression of flexion to at least 90° over 2–4 weeks [103, 105]. Early mobilization and functional rehabilitation should begin when tolerable with a focus on quadriceps strengthening [112, 115]. A patellar stabilizing brace should be considered and is typically associated with higher patient satisfaction [115].

If patellar subluxation or recurrent subluxation is diagnosed, the treatment regimen is similar. For comfort, a patellar stabilizing brace can be offered, but the long-term treatment is physical therapy with a home program focusing on quadriceps, abductor, adductor, and hip flexor strengthening as well as gastrocnemius stretching. If conservative therapy is adhered to, success is appreciated in approximately 80% of patients [107].

Return to Sports

With conservative care, return to sport can be considered when there is no effusion and full passive range of motion and quadriceps strength is 80% compared to the uninjured leg [103]. A study by Atkins et al. showed that almost half of athletes returned to play by 12 weeks and nearly 70% by 24 weeks. They did note a decrease in athletic performance for about 6 months after injury [103]. Another study, looking at military recruits, showed the majority returned to normal training by 51 days [105]. If surgical patellar stabilization is performed, return to play is higher between 84% and 100%; however, return to previous performance and time to return to play are much more variable between 33 and 77% and 3 and 12 months, respectively [124].

Complications

The most common and concerning complication from patellar dislocation is re-dislocation, which occurs in approximately 30% of first-time dislocations [109, 113, 115]. Sports increase the risk of reoccurrence up to 70% [111]. About 17–38% of first-time dislocations will develop chronic patellar instability, and approximately half of those will require instability surgery [104, 109, 111]. If a re-dislocation is to occur, there is a 6% chance it will happen in the contralateral knee, 60% chance it happens within 1 year, and 90% chance it happens within 3 years [110, 111]. There is a lower re-dislocation rate with initial surgical correction; however, surgery increases the risk of complications such as infections, blood clots, and nerve injuries and doubles the risk of osteoarthritis [112–114, 125, 126]. Despite the lower re-dislocation rate with surgery, long-term outcomes favor conservative care [113, 114, 126].

Pediatric Consideration

The incidence of patellar dislocation is highest in the adolescent population ages 14–18 years old at 148 per 100,000 person-years [109, 127]. The highest overall rate for initial dislocations is females ages 10–14 years old [110, 111, 127]. The highest rate of re-dislocation is for those less than 13 years old at the time of initial dislocation [109, 127]. The lowest success rates of conservative or surgical treatment arise in young athletes with immature physes and trochlear dysplasia [111].

Proximal Tibiofibular Dislocation

Mechanism of Injury

Proximal tibiofibular dislocations happen as a result of direct trauma or during athletic activities [128–132]. Due to the biomechanics of the proximal tibiofibular joint, which mainly dissipates torsional force at the ankle, motion at ankle and knee must align [130, 133]. In sports, the dislocation occurs when the ankle is inverted or plantarflexed with rotational force of the lower leg on a flexed knee [128, 130, 134–137].

Epidemiology

Dislocations at the proximal tibiofibular joint are a rare injury that results from high-velocity trauma or sporting events [128, 130–132]. They can be associated with fractures [132]. Due to the rarity of the injury, there are mostly case studies and no epidemiological data to suggest incidence. The majority occur between 13 and 27 years of age and are associated with sports [132]. The most common sports appear to be soccer, football, rugby, basketball, hockey, horseback riding, roller-skating, snowboarding, and long jump [28, 129–131, 133, 134, 136–145]. The most

common direction of dislocation is movement of the fibular head anterolateral, accounting for up to 85% of dislocations [131–133, 136].

Classification

There are four types of proximal tibiofibular dislocations, described by fibular head movement, as designated by Ogden [132]. Type I is a subluxation of the joint and is typically self-limiting. Type II is an anterolateral dislocation, which is the most common type. Type III is a posteromedial dislocation, which typically results from trauma. Lastly, Type IV is a superior fibular head dislocation and is associated with tibial shaft fractures [132].

Clinical Presentation

Clinical suspicion must be considered for proximal tibiofibular dislocation when someone presents with lateral knee pain. The patient will have lateral knee tenderness with a prominence of the fibular head, pain with weight bearing, and limited knee range of motion especially above 110° of flexion [131, 133, 142]. Pain is exacerbated with fibular head manipulation as well as ankle movements, specifically dorsiflexion and eversion [129, 132, 135, 143, 146]. Due to the proximity of the common peroneal nerve to the fibular head, there may also be common peroneal nerve neuropraxia [132, 142].

Diagnosis

The diagnosis of a proximal tibiofibular dislocation can be easily missed in up to one third of cases [130, 132]. The diagnoses should be considered with all lateral sided knee pain; however, it can often be confused with lateral meniscus tears [131, 138]. Proximal tibiofibular dislocations are a clinical diagnosis; however, comparative x-rays can help clarify an uncertain diagnosis [130, 131, 133, 139]. Comparative x-rays should be done as the diagnosis can still be easily missed on individual x-rays [131]. Radiography should be performed with weight-bearing AP and lateral with about 20° of flexion [138, 139, 147]. In the AP view, the fibular head will be more lateral and not pointing directly toward the lateral femoral epicondyle and in the lateral view; the fibular head will be more anteriorly and not pointing toward the posterior half of Blumensaat line [138, 139, 147]. In addition to standard views, an oblique view is also recommended. Following these criteria is more sensitive and specific than the fibular head overlap with the tibia, which can vary with degree of knee flexion [139, 147]. If still uncertain, CT or MRI can help confirm the diagnosis [129, 133, 134, 148]. MRI is a valuable modality in evaluating concurrent ligament injuries.

Initial Management

If a proximal tibiofibular dislocation is diagnosed and has not spontaneously reduced due to the posterior forces of biceps femoris and LCL, initial reduction should be attempted [128, 134, 136, 137]. To reduce, the knee should be flexed 90°–110° to put ligaments in laxity, and then the foot should be everted, while an anterior to posterior force is applied to the fibular head [130–132, 145]. There should be a "clunk" with successful reduction and a similar amount of fibular head translation as compared to the uninjured side [132, 145]. Closed reduction is successful in about three fourths of the cases [131]. After reduction, ankle dorsiflexion as well as knee stability should be examined [132, 145]. Pre- and post-reduction examinations should also assess the common peroneal nerve integrity with a Tinel's test, resisted dorsiflexion, and sensation of the dorsal foot [135, 144, 149, 150]. If the reduction is successful, the neurovascular status is intact, and there is a debate if the patient needs immobilization or not [144]. Limited data is available; however, there is no reported complication of cases without immobilization [28]. Therefore, it is reasonable to immobilize for comfort and allow weight-bearing as tolerated with short-term outpatient follow-up.

Indications for Orthopedic Referral

Referral to orthopedic surgery is indicated if the reduction attempts fail and in cases with concomitant displaced fractures and those with significant neurovascular compromise [130, 133, 135, 141, 143, 145, 146]. Orthopedics should also be consulted if closed reduction is successful, but instability, subluxation, or pain persists despite 6–12 weeks of conservative care [135, 137].

Follow-Up Care

There is no standard postreduction care of a proximal tibiofibular dislocation [131, 133]. Initial immobilization is debated, but no immobilization or early immobilization appears safe [28, 131, 133, 144]. Variable treatments range from no immobilization to immobilization for up to 6 weeks [146]. Generally, a brace or support bandage is provided or offered for up to 6 weeks with progression to full weight-bearing as tolerated with early rehabilitation of a knee-strengthening program [131, 132, 134, 140, 144–146]. This management is similar if subluxation occurs [134, 136].

Return to Sports

There is no agreement on standard return to play measures after proximal tibiofibular joint dislocation. In general, athletes should be allowed to start a return-to-play protocol after their symptoms are resolved and strength is back to the baseline. Case studies have shown successful return to play in 6–16 weeks, which include at the professional level [130, 131, 134, 141]. Earlier return to play, at 6 weeks, showed no complications at 6-month follow-up with conservative care [131]. After surgery, a 3-month return to play appears safe [135, 137].

Complications

The majority of complications with proximal tibiofibular dislocations are with associated fractures [132, 141, 149, 150]. In isolated dislocations, the most common complication is prolonged pain with activity [131]. The most concerning complication is the common peroneal nerve injury. This is a rare complication and most likely occurs with Type II dislocations [132, 143, 145]. The majority of nerve injuries are transient, but it is important to assess ankle dorsiflexion strength and numbness to the dorsal foot after injury and reduction [132, 133, 144, 145]. Other than nerve injury, other complications are chronic joint instability and risk of osteoarthritis [128, 131, 143].

Pediatric Consideration

Proximal tibiofibular dislocations are rare in the pediatric population and are usually associated with fracture [151]. Therefore, careful evaluation of adjacent structures, including the ankle and hip, is important. Adolescents appear more likely to suffer this injury as the majority of sports-related dislocations occur in individuals between the ages of 13 and 27 years [132]. If atypical dislocation occurs in the pediatric population, hypermobility disorders should also be considered [146].

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25 Tibia and Fibula

William Denq

Key Points

- Up to 30% of sports-related fractures involve the tibia and fibula.
- High-risk sports such as soccer, rugby, and skiing are responsible for a large proportion of these fractures.
- Although a majority of these fractures can be managed conservatively, surgical management can be warranted.
- Open fractures, neurovascular compromise, and suspicion for compartment syndrome may require emergent surgical intervention.
- Tibial diaphyseal fractures are the most common reason for developing acute compartment syndrome.
- Fibular fractures rarely result in lateral compartment syndrome.
- Malunion, nonunion, and refracture rates are low, but inappropriate management and early return to sports may result in these complications.

Introduction

Lower extremity injuries are common in sports. Most injuries are minor and athletes may not seek care. Sports-related fractures predominantly occur in the upper extremity, but up to 30% involve the tibia and fibula [1]. High-risk sports such as soccer, rugby, cycling, and skiing are responsible for a large proportion of these fractures [2, 3]. Management of these injures can often be performed conservatively; however, improper care can result in known complications. Additionally, certain fractures merit emergent operative intervention. This chapter will focus on extra-articular tibial and fibular fractures as they relate to sports trauma.

Anatomy

The tibia has three ossification centers, one specifically for the diaphysis and two for the epiphyses [4]. The centers begin to close earlier in females (15–16 years of age) than males (17–18 years of age) [4]. Proximally, the tibia is a component of the knee joint and, distally, it is a component of the ankle joint. It is medial and connected to the fibula via the interosseous membrane to form the syndesmotic joint [5]. The tibial diaphysis, in cross section, is triangular with three surfaces and borders [6]. The lateral surface is in contact with the anterior compartment, the posterior surface is with the posterior compartment, and the medial surface is subcutaneous [4].

The fibula also has three ossification centers, one for the diaphysis and two for the epiphyses [5]. These centers begin to close around the second decade of life [5]. The fibula is significantly thinner than the tibia and starts behind the tibial head at the proximal tibiofibular articulation (superior tibiofibular joint), running down to form the ankle joint with the tibia [5]. The fibular head is connected to the lateral condyle of the tibia via joint capsule, anterior and posterior proximal tibiofibular ligaments (Fig. 25.1). Distal to the fibular head is the fibular neck where the bone narrows. These comprise the proximal fibula. Distal to the proximal fibula is the fibular diaphysis. The diaphysis, in cross section, is initially triangular with three surfaces and becomes more irregularly contoured as it moves distally secondary to muscle attachments [5].

Nutrient vessels from the posterior tibial artery and periosteal vessels from the anterior tibial artery supply blood to the tibia [7]. The anterior tibial artery can be damaged as it moves through a hiatus in the interosseous membrane. A large nutrient vessel and periosteal vessels from the fibular (peroneal) artery supply the fibular diaphysis and periosteum, respectively [8]. The tibial and fibular

W. Denq (✉)
Departments of Emergency Medicine and Sports Medicine, University of Arizona University of Arizona, Tucson, AZ, USA
e-mail: denq@email.arizona.edu

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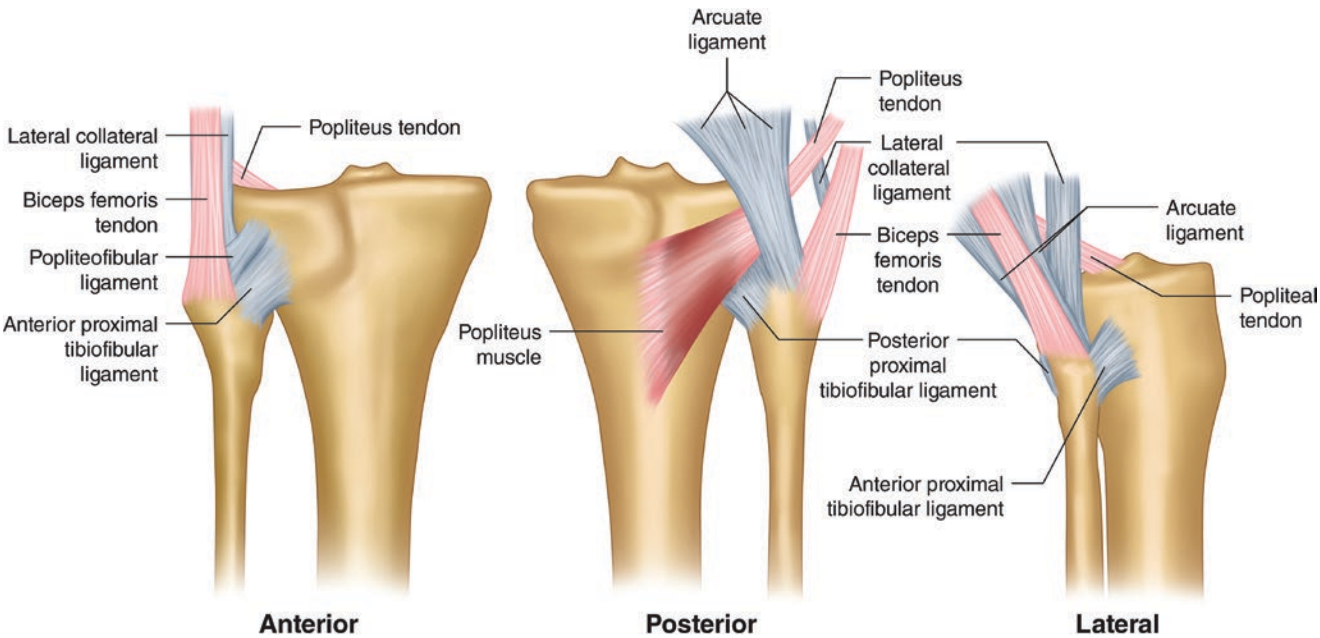

Fig. 25.1 Proximal tibiofibular joint anatomy

nerve that arise from the sciatic nerve are responsible for the motor function of the leg and foot and a majority of the sensation. The tibial nerve passes through the popliteal fossa deep to the soleus and runs distal in a posteromedial fashion until it branches deep to the flexor retinaculum at the origin of the abductor hallucis. The common fibular (peroneal) nerve passes from the superolateral angle of the popliteal fossa, wraps around the neck of fibula piercing the fibularis longus, and divides into the superficial and deep fibular nerves. This nerve is at high risk for damage with injuries to the proximal fibula and fibular diaphysis given its close proximity to the skin [9].

The tibia carries a majority of the whole body weight while the fibula carries only 6–15% of it [10]. The tibia serves as the origin and insertion point for 11 muscles, and the fibula helps stabilize the ankle joint [4, 5]. Due to proximity to major neurovascular bundle, significant injuries such as fractures can compromise blood supply to distal leg and damage neurovascular structures.

Soft Tissue Injuries

A direct blow to the leg can not only result in injury to skin, muscle, tendon, and ligament but also severe conditions such as acute compartment syndrome [11]. When the pressure within the closed compartments of the lower extremity rise to a certain level secondary to bleeding or swelling, muscle ischemia and necrosis can occur resulting in significant morbidity [11, 12]. The incidence of acute compartment syndrome is estimated to be 7.3/100,000 in males and 0.7/100,000 in females [12]. Of this, about 20% are due to sports [12]. Although tibial diaphyseal fractures are the most common cause, soft tissue injury without fracture is the second most common cause [12].

The four compartments of the lower leg are anterior, lateral, superficial posterior, and deep posterior [13]. Diagnosis, while controversial and difficult, relies on clinical suspicion, examination, and measurement of compartment pressures [14]. Emergent fasciotomy is the definitive treatment to decompress the involved compartments [11, 12, 14]. Typically the surgical wound is left open to secondary closure with vacuum assistance or an attempted closure is made 3–5 days after the initial operation [11, 13, 15]. In the general population, over 25% of patients with a fasciotomy had functional disability [16]. Long-term muscle damage secondary to fasciotomy has also been documented [17]. Depending on the individual athlete, the level of recovery and recovery period will vary. Please see Chap. 39 for more on acute compartment syndrome. Other soft tissue injuries are discussed elsewhere in the book.

Fractures

Diaphyseal (Shaft) Fracture of the Tibia

In this section, we will discuss proximal and distal extra-articular and diaphyseal tibial fractures.

Mechanism of Injury in Sports

An isolated fracture of the tibial diaphysis without underlying bone pathology typically requires a high kinetic direct impact. A large majority of these tibial diaphyseal fractures are caused by motor vehicle accidents and sports [18, 19]. Over 80% of sports-related tibial diaphyseal fractures are

due to soccer [18]. There are several proposed mechanisms in soccer that can result in this type of fracture. The most common mechanism is a slide tackle to a planted leg [20]. A smaller, but still prominent, percentage is secondary to skiing. The most common mechanism while skiing is a fall [21]. The knee is flexed during the fall, but the foot is fixed. The fall forward results in an anterior-posterior displacement or torsional strain [21]. The "skier's boot top fracture" results from the hard fixed boot and forward kinetic energy of the superior tibia, while the spiral fracture is secondary to torsional strain [21, 22]. Expanding to the general athletic population, this injury mechanism is either a higher kinetic force or rotational torque exerted on the tibia [23].

Epidemiology

There is a unimodal distribution of young males that sustain high-energy trauma and older females involved in low-energy trauma [18, 19]. The incidence of tibial diaphyseal fractures is cited as anywhere from 1 in 2000 to about 17 in 100,000 per year [19, 24]. They comprise about 2% of all fractures sustained in a 61:39 ratio of males to females [3]. Males, with a peak incidence age 10–20 years, tend to be injured secondary to sports activities, while females who are older than 30 years are injured secondary to walking and indoor activity [19]. Of soccer players who sustained a lower extremity fracture, 8.5% were isolated tibial diaphyseal fractures [25]. The incidence of tibial diaphyseal fractures has noticeably decreased more recently especially in soccer players which may be secondary to change in regulations and improvement in shin guard technology [26, 27].

Classification

The AO Foundation and Orthopaedic Trauma Association (AO/OTA) classification for diaphyseal fractures is widely accepted (Fig. 25.2) [28]. Fracture localization for the tibial

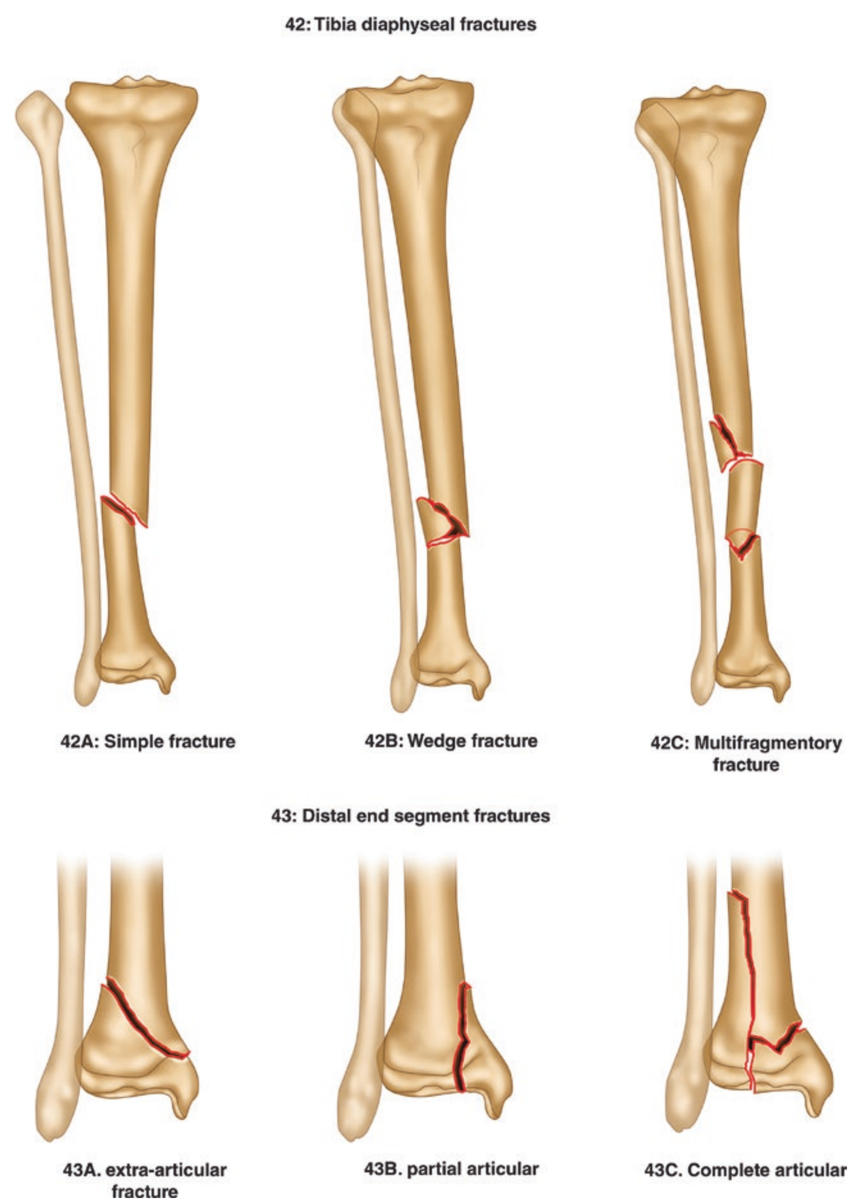

Fig. 25.2 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for diaphyseal and distal tibial fractures [28]

diaphysis is 4 (tibia) and 2 (diaphysis). Localization is qualified by which one third of the diaphyseal segment the fracture is located (proximal, middle, distal). The morphology of diaphyseal fractures is described as simple (Type A), wedge (Type B), and multi-fragmentary (Type C). Type A and Type B fractures are further qualified by the location of the fracture**.** The most common fracture pattern seen in soccer players is the transverse tibial diaphyseal fracture or a 42A3 [27]. In the general population and skiers, the most common fracture pattern seen is a spiral tibial diaphyseal fracture or a 42A1 [19].

Clinical Presentation

The athlete will present to the sideline unable to bear weight on the affected side. Pain, ecchymosis (Fig. 25.3a), swelling, and deformity may be present at the site of injury. Occasionally, they may report weakness and/or paresthesia associated with nerve injury. The skin should be closely examined for tense compartments and to determine if the fracture is open (Fig. 25.4). A neurovascular examination evaluating the dorsalis pedis (DP) and posterior tibialis (PT) pulses and lower extremity strength and sensation should be performed and documented.

Fig. 25.3 Right medial tibial ecchymosis (**a**) in a 40-year-old male as a result of a collision into a tree stump while snowboarding. Plain radiography shows a mildly displaced mid-diaphyseal tibial fracture (**b**, **c**). Initial immobilization with a long-leg posterior splint (**d**)

Diagnosis

Diagnosis of a tibial diaphyseal fracture is confirmed by plain radiography (Figs. 25.3, 25.4, 25.5, 25.6, 25.7, and 25.8). AP and lateral views are recommended along with radiograph series of the knee and ankle. These images will help determine classification of fracture and management. The amount of displacement, angulation, and rotation are key factors that influence operative versus nonoperative treatment [29].

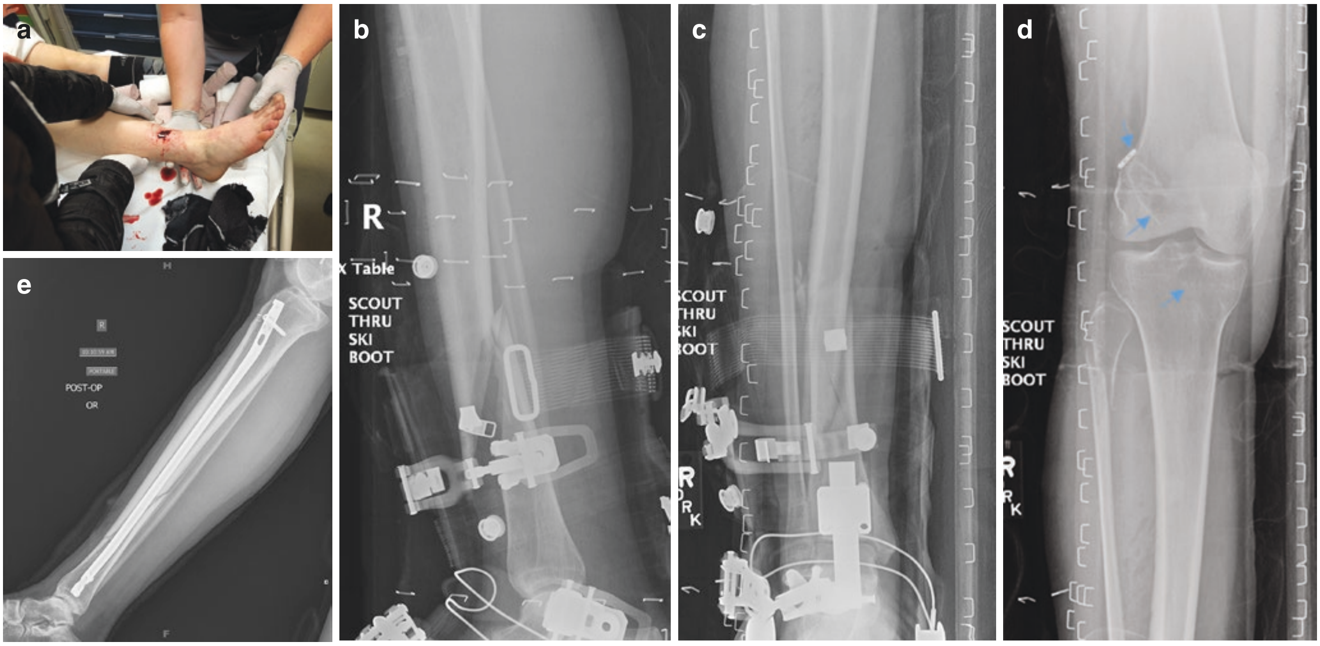

Fig. 25.4 Open distal tibia (**a**) and proximal fibula (Maisonneuve) fractures in a 38-year-old female skier (**b–d**). Tibia fracture was treated with an intramedullary rod (**e**). Previously reconstructed ACL surgery (arrows) is evident in AP view (**d**)

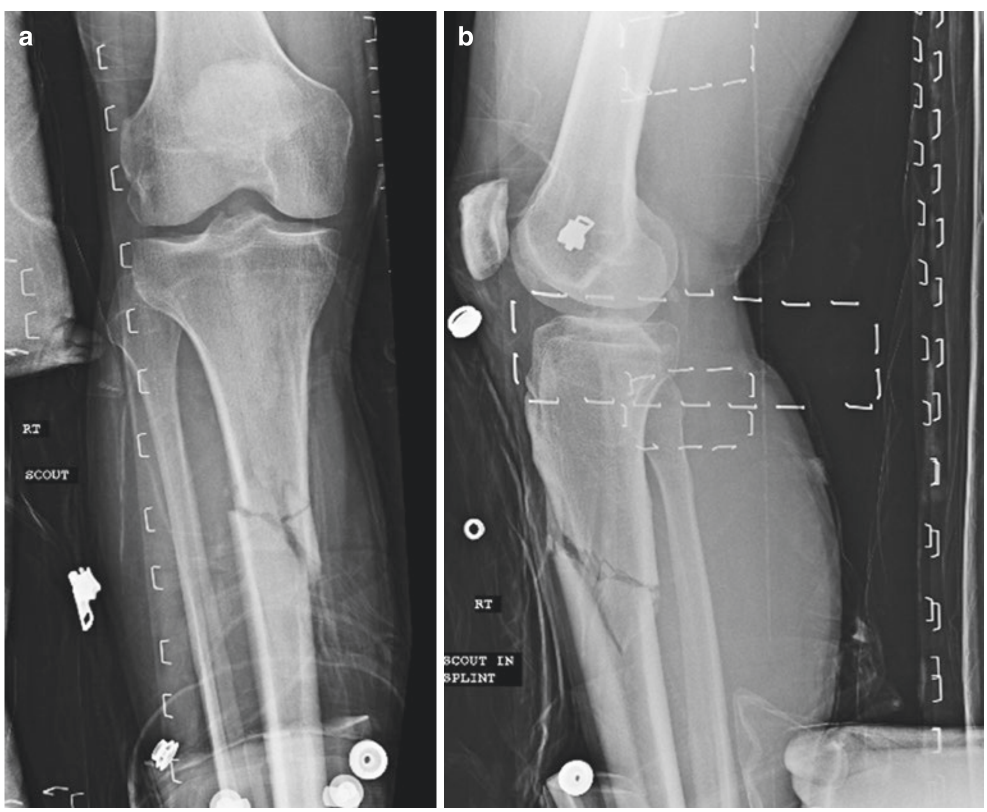

Fig. 25.5 A comminuted and mildly displaced fracture of the proximal right tibia in a 44-year-old male as a result of a ski injury (**a**, **b**)

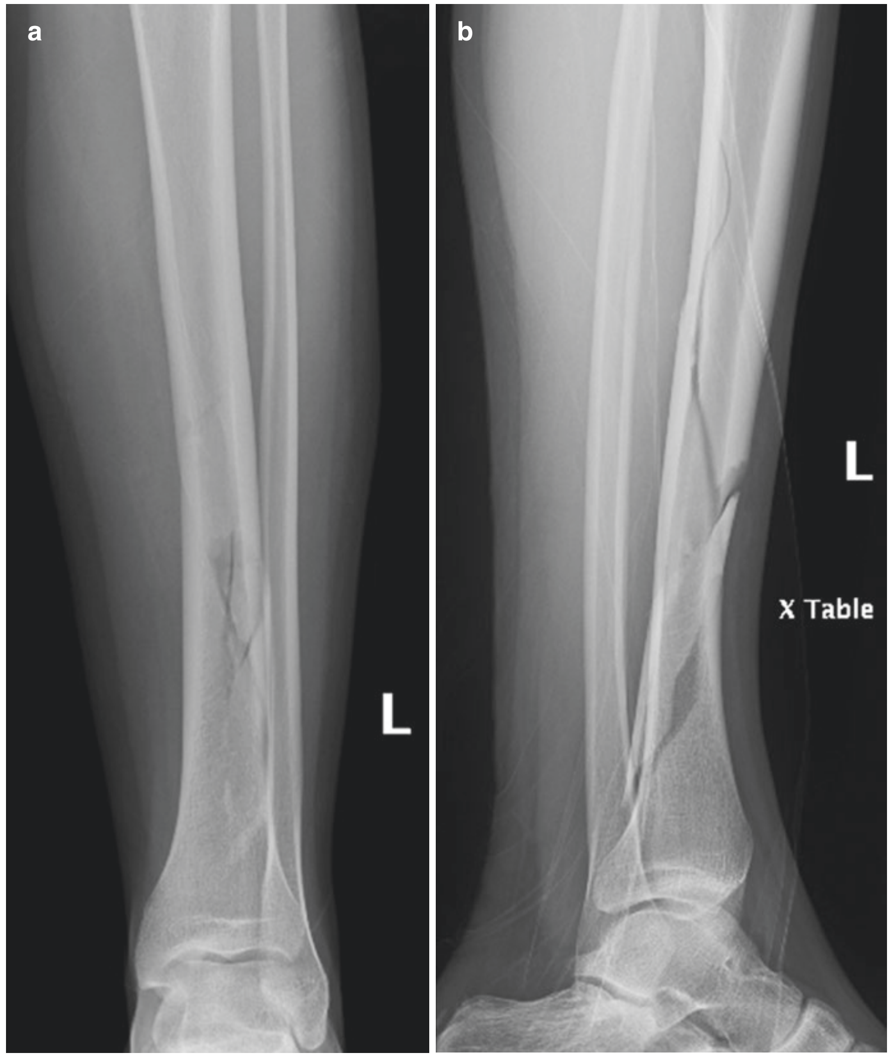

Fig. 25.6 Mildly displaces and comminuted tibia fractures in a 23-year-old male skier (**a**, **b**)

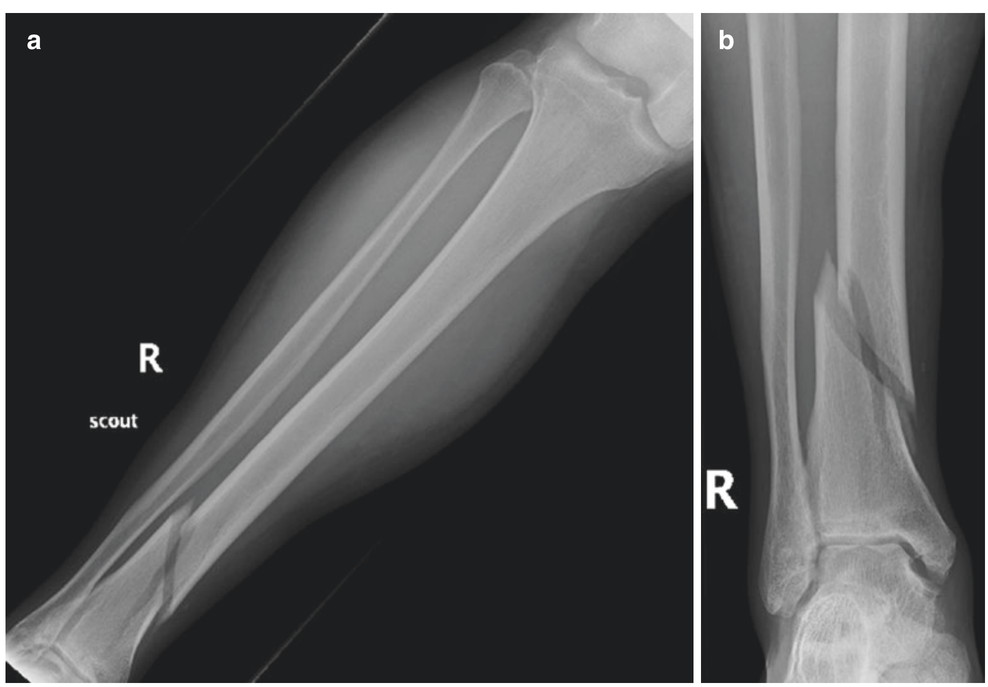

Fig. 25.7 Displaced spiral fracture of the distal right tibia in a 45-year-old male as a result of a ski injury (**a**, **b**)

Fig. 25.8 Mildly displaced, extra-articular, distal tibia fracture in a 21-year-old female skier

Initial Management

The fracture should be immobilized in a well-padded long-leg posterior splint (Fig. 25.3) [29]. The splint, being non-circumferential compared to a cast, allows for swelling that typically occurs after this injury. Tibial diaphyseal fractures are at increased risk for developing compartment syndrome, and casting is not warranted until swelling has stopped or reduced. Elevation and icing of the affected extremity is important to reduce swelling. If pain cannot be controlled with oral medications, consider admitting the patient for monitoring and parenteral administration of pain medication. The patient should be non-weight-bearing and provided an alternative method for mobility (e.g., crutches). Counseling should be provided regarding risk for compartment syndrome.

Indications for Orthopedic Referral

Emergent orthopedic referral is warranted for open fracture, suspicion for compartment syndrome, or neurovascular compromise [29]. Urgent operative indications are unstable, comminuted, and/or segmental fractures as disability, malunion, and nonunion complication rates increase (Figs. 25.4, 25.5, 25.6, and 25.7) [30]. Specifically, displacement >5 mm in AP and lateral planes, >10° angulation in AP, >5° angulation in lateral, and >10° of rotation are general indications [29, 31]. If there is malunion or nonunion that occurs during conservative management, a referral for operative management is indicated [29].

However, the management of closed tibial diaphyseal fractures remains a controversial topic. Traditionally, long-leg casting has been very successful with only 0.7% nonunion rate in isolated, closed stable tibia fractures [32]. However, there may be improved functionality or return to functionality in those who were treated with locked intramedullary nailing (IMN) compared to functional bracing (Fig. 25.4) [27, 30, 33]. Operative fixation may be a reasonable consideration especially in professional athletes who would like to get back to play as soon as possible.

Overall, the evidence is still unclear, and reviews so far do not demonstrate superiority of operative compared to nonoperative management for closed and mildly displaced tibial diaphyseal fractures [34, 35].

Follow-Up Care

Stable tibial diaphyseal fractures can be managed by the experienced primary care physician (e.g., primary care sports medicine physicians). Follow-up should be arranged in 2 days to allow for care establishment and overall fracture assessment. This includes a neurovascular evaluation and general counseling for the patient. A long-leg cast should be placed after swelling has halted or diminished. This typically occurs 1 week after the initial injury. However, an appropriate assessment of swelling should be performed prior to cast application. The cast allows for 5–15° of knee flexion and 90° ankle in neutral [30]. It functions to prevent inappropriate rotation of the knee and ankle to protect the tibia.

Repeat radiographs are recommended weekly for the 2 weeks following the initial injury to ensure appropriate alignment on both AP and lateral views [30]. Once the patient is able to bear weight in the long-leg cast, they should be transitioned to a below-knee or patellar tendon-bearing cast. Over the 6 months following injury, if there is >5° of varus/valgus in any plane, shortening greater than 1.2 cm, and evidence of nonunion, referral to orthopedics is indicated.

Return to Sports

There is some evidence that patients who underwent surgery were quicker to return to activity compared to those who were managed nonoperatively [27, 30, 33]. The proposed reason is earlier mobilization in the operative group allows for preservation of muscle mass and reduced joint stiffness compared to prolonged immobilization in a cast [27]. However, there is also evidence that athletes who undergo an operation actually have an increased time of return to play secondary to residual disabilities associated with the surgery [25].

Choosing between operative or nonoperative management is a decision that should be made on a case-by-case

basis. On an all-injury basis, athletes with tibial diaphyseal fractures have one of the slowest rates of return to sports (mean of 38 weeks) [25]. However, the majority of athletes return to play and a large portion of those athletes return to their pre-injury level of play [1, 23].

Complications

The tibial diaphyseal fracture is one of the most common causes of compartment syndrome [12]. The average annual incidence for men is 6.9 per 100,000 and 0.2 per 100,000 for women [12]. About half of these patients were injured while playing soccer. Fat embolism is reported in small reports to occur in 4–19% of athletes who sustained a tibial diaphyseal fracture [23, 36]. The incidence of nonunion is reported as 2% [23].

Pediatric Consideration

Tibial diaphyseal fractures in adolescents are reported to make up about 5% of all sports-related fractures and occur more frequently in males than females [2, 37]. In skiing, children typically do not have an associated fibular fracture (Figs. 25.9, 25.10, 25.11, and 25.12 [21]. Closed reduction and casting are the mainstay of treatment, but operative management is considered on an individualized basis [37]. Minimally displaced tibial diaphyseal fractures can develop varus angulation as a result of posterior compartment muscular forces [37]. Union typically occurs in 10 weeks with nonunion occurring in less than 2% of cases [37]. Compartment syndrome is less likely in the pediatric population compared to the adult population [37].

Proximal and Diaphyseal (Shaft) Fractures of the Fibula

Mechanism of Injury in Sports

The isolated acute fibular diaphyseal fracture is rather uncommon but is usually secondary to a direct blow to the lateral aspect of the leg [38–40]. Rarely, a forceful ankle dorsiflexion can cause isolated fibular diaphyseal fractures. The suggested mechanism is a forced contracture of the soleus origin with fibular loading that results in significant deformation and fracture of the fibula [39].

There are three reported reasons for an acute isolated proximal fibular fracture. Direct impact to the lateral leg, pronation-external rotation on a fixed leg, and a varus stress [9, 41–44]. Pronation-external rotation on a fixed leg describes the classic mechanism for the Maisonneuve fracture (Fig. 25.4) [42]. The modern description for this fracture is a proximal fibular fracture, a tibial-sided bony or ligamentous injury, and an unstable syndesmosis [44]. This fracture will be discussed separately throughout this section.

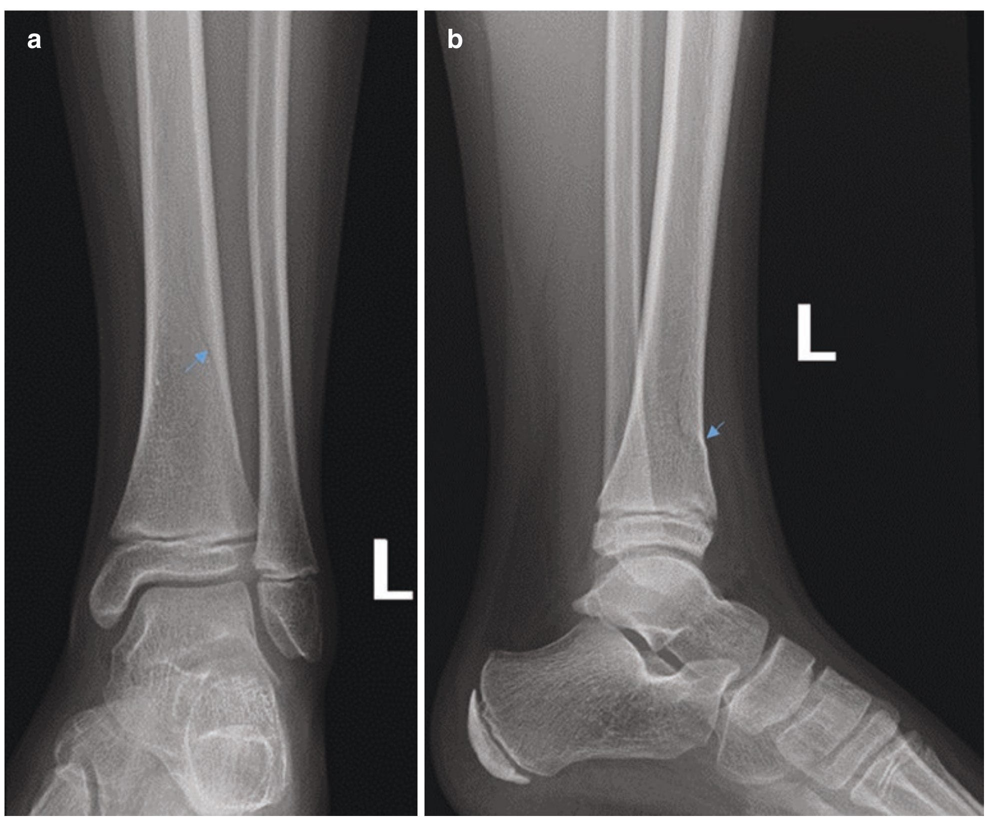

Fig. 25.9 Distal tibia torus fracture (arrows) in an 8-year-old female skier (**a**, **b**)

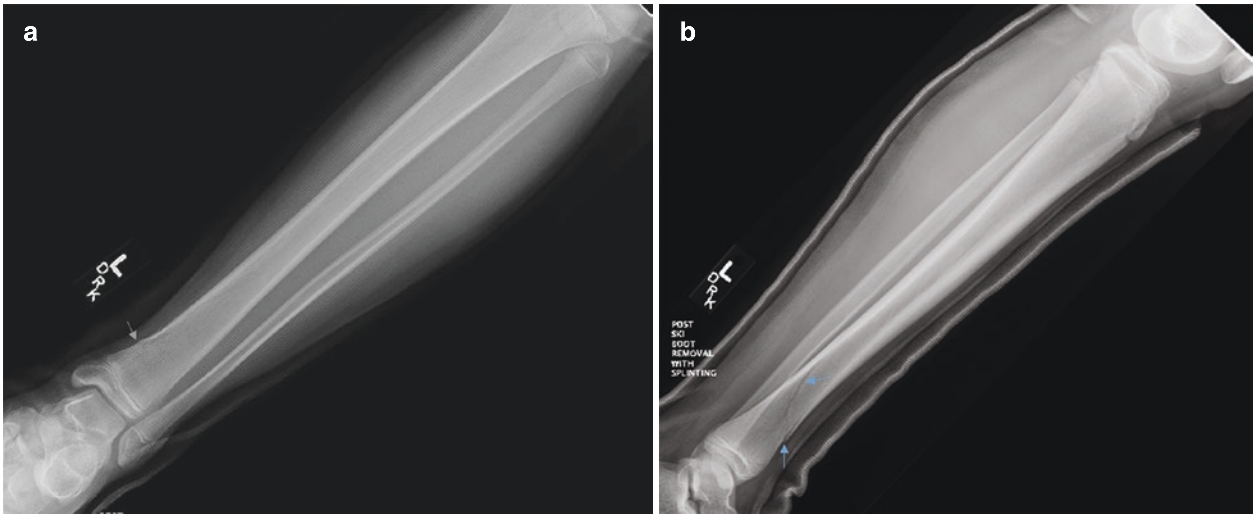

Fig. 25.10 A mildly displaced distal tibia fracture (arrows) in a 15-year-old male skier (**a**, **b**)

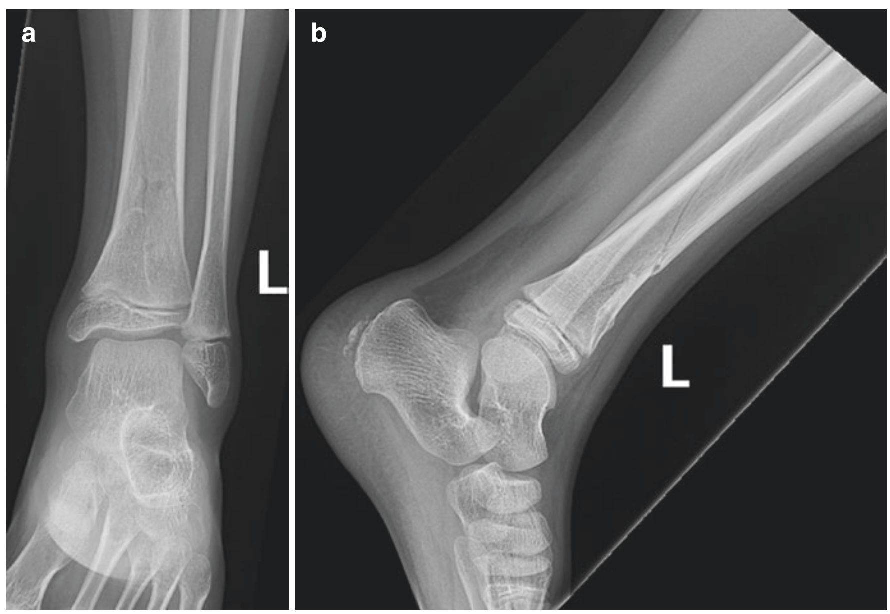

Fig. 25.11 A comminuted, nondisplaced, distal tibia fracture in an 8-year-old male skier (**a**, **b**)

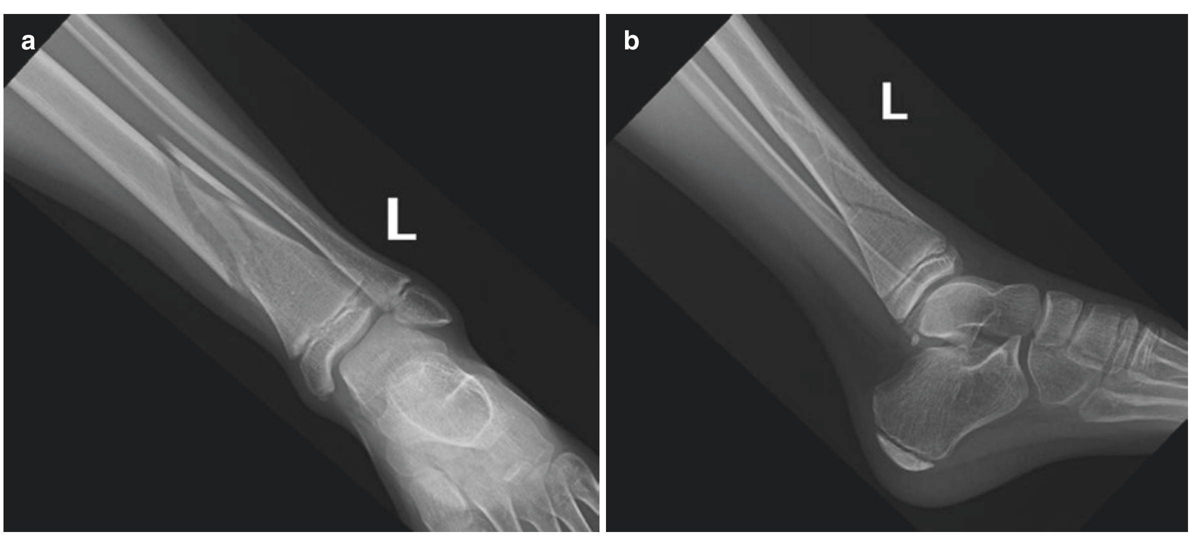

Fig. 25.12 A comminuted, spiral, and displaced distal tibia fracture in a 9-year-old female snowboarder (**a**, **b**)

Epidemiology

Isolated proximal and diaphyseal fibula fractures are only mentioned in case reports and make up a very minor portion of fractures in athletes (Figs. 25.13, 25.14, and 25.15) [9, 40, 41]. Single-center epidemiology studies rarely report these fractures in the general population [3, 45]. Military literature has reported that the upper third isolated fibular fracture or dislocation is known as the classic parachute injury and that is highly uncommon as well [46, 47]. The Maisonneuve fracture is one of the more reported and well-described proximal fibular fractures and is associated with approximately 10% of all ankle fractures [44].

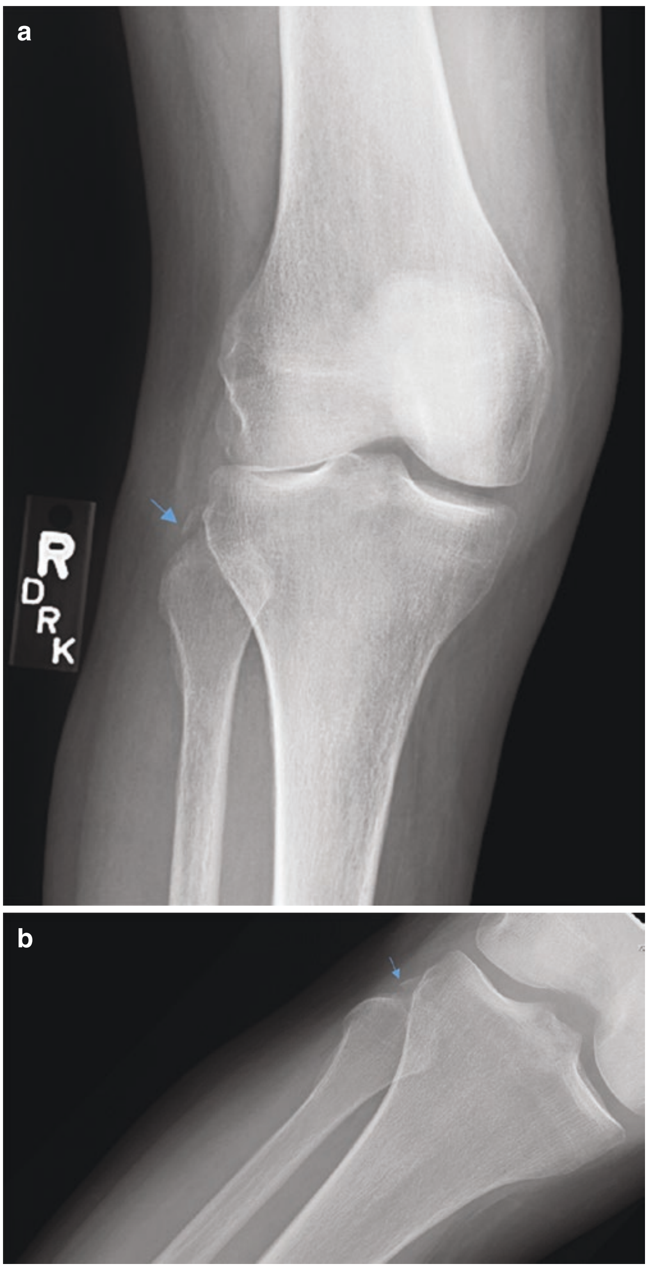

Fig. 25.13 Fracture of the head of the right fibula (arrows) in a 69-year-old female as a result of a ski injury (**a, b**)

Classification

The AO Foundation and Orthopaedic Trauma Association (AO/OTA) classification for fibula fractures is widely accepted [48]. Fracture localization for the fibular diaphysis is 4F (fibula) and 2 (diaphysis). Localization is qualified by which one third of the diaphyseal segment of the fracture is located (proximal, middle, distal). The morphology of fibular fractures is described as simple (Type A) or wedge/multi-fragmentary (Type B). Fracture localization for the proximal fibula is 4F (fibula) and 1 (proximal end). Qualifying the localization is whether or not the fracture is intra-articular (o) or extra-articular (n). The morphology of fibular fractures is described as simple (Type A) or wedge/multi-fragmentary (Type B).

Clinical Presentation

The athlete will present to the sideline with focal tenderness near the fracture site but may be able to bear weight and/or return to play quickly [40]. There may not be swelling or a palpable deformity. The fracture can be misdiagnosed as a soft tissue injury given how benign it may initially present [38, 49]. They may complain of increasing pain as they bear weight that is not resolving. With careful history taking, they may reveal a repetitive mechanism with subacute/chronic pain that became acutely worse. They may present with evidence of foot drop or decreased sensation along the upper lateral leg if the fibular nerve is affected [9].

Regarding the Maisonneuve fracture, the athlete will typically present with difficulty with weight-bearing and distracting ankle pain, but not proximal fibular pain [50].

There should be focal tenderness at the fracture site. Tenderness and range of motion should additionally be evaluated at the ankle. Stability of the knee ligaments, especially the lateral collateral ligament, should be evaluated for any associated injury [51]. A neurovascular examination evaluating the dorsalis pedis (DP) and posterior tibialis (PT) pulses and the fibular nerve should be documented. Test the fibular nerve by asking the patient to ankle plantar- and dorsiflex and assess sensation of the upper two third of the lateral leg. The skin should be closely examined to determine for open fracture or significant tension.

Diagnosis

Diagnosis is often made on plain radiograph of the tibia and fibula in both AP and lateral views (Fig. 25.13 and Chap. 24; Fig. 24.37). A fibular avulsion fracture, also known radiographically as the "arcuate sign," is indicative of injury to the posterolateral corner (Fig. 25.16) and will likely require more advanced imaging such as an MRI [51].

If there is no radiographic evidence and a high degree of clinical suspicion, conservative measures can be taken with follow-up radiographs to look for evidence of remodeling [49]. Diagnostic ultrasound has been used to visualize a fibular diaphyseal fracture, but both the imaging modality and pathology have been too uncommon to determine a sensitivity and specificity [49].

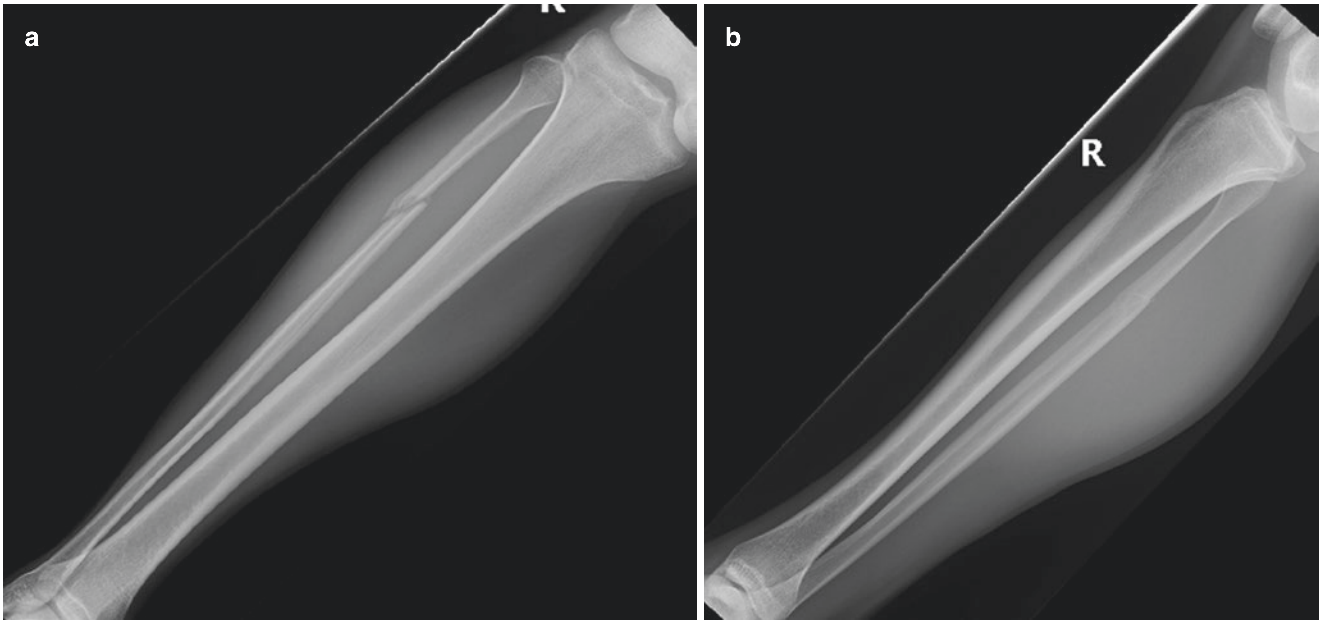

Fig. 25.14 An isolated fibula fracture in a 23-year-old male skier (**a**, **b**)

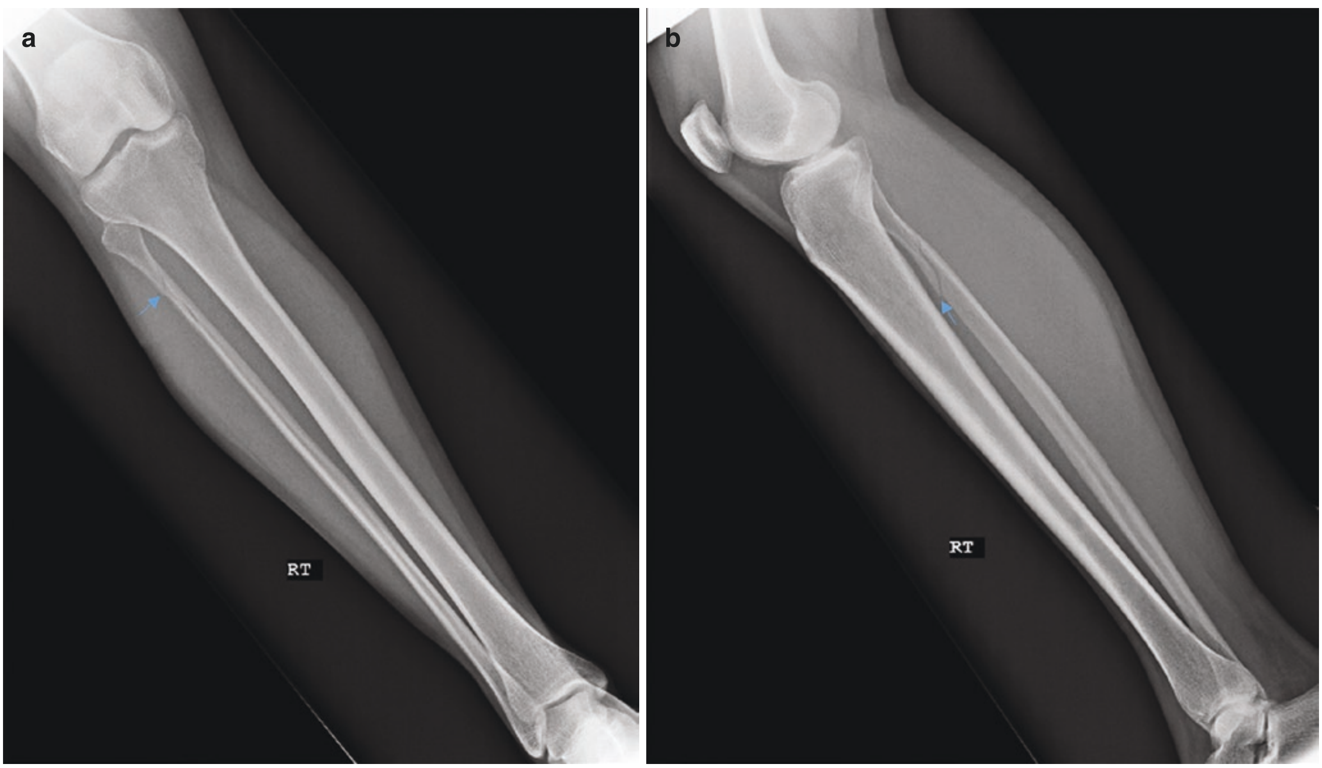

Fig. 25.15 An isolated proximal fibula fracture in a 44-year-old female skier (**a**, **b**)

When assessing a patient who had an ankle injury, especially with a rotational injury, it is prudent to assess the full length of the fibula. Patients with a Maisonneuve fracture may not initially have proximal fibular pain but will typically be tender on palpation [50]. Stress radiography and MRI may be necessary to determine the level of associated syndesmotic injury.

Initial Management

The athlete can be initially immobilized in a stirrup splint with the ankle at 90°. There is very little swelling typically associated with this injury, but the splint should be well-padded to protect the fibular nerve as it runs around the neck of the fibula. Elevation and icing of the affected extremity are important to reduce swelling. The patient can be weight-

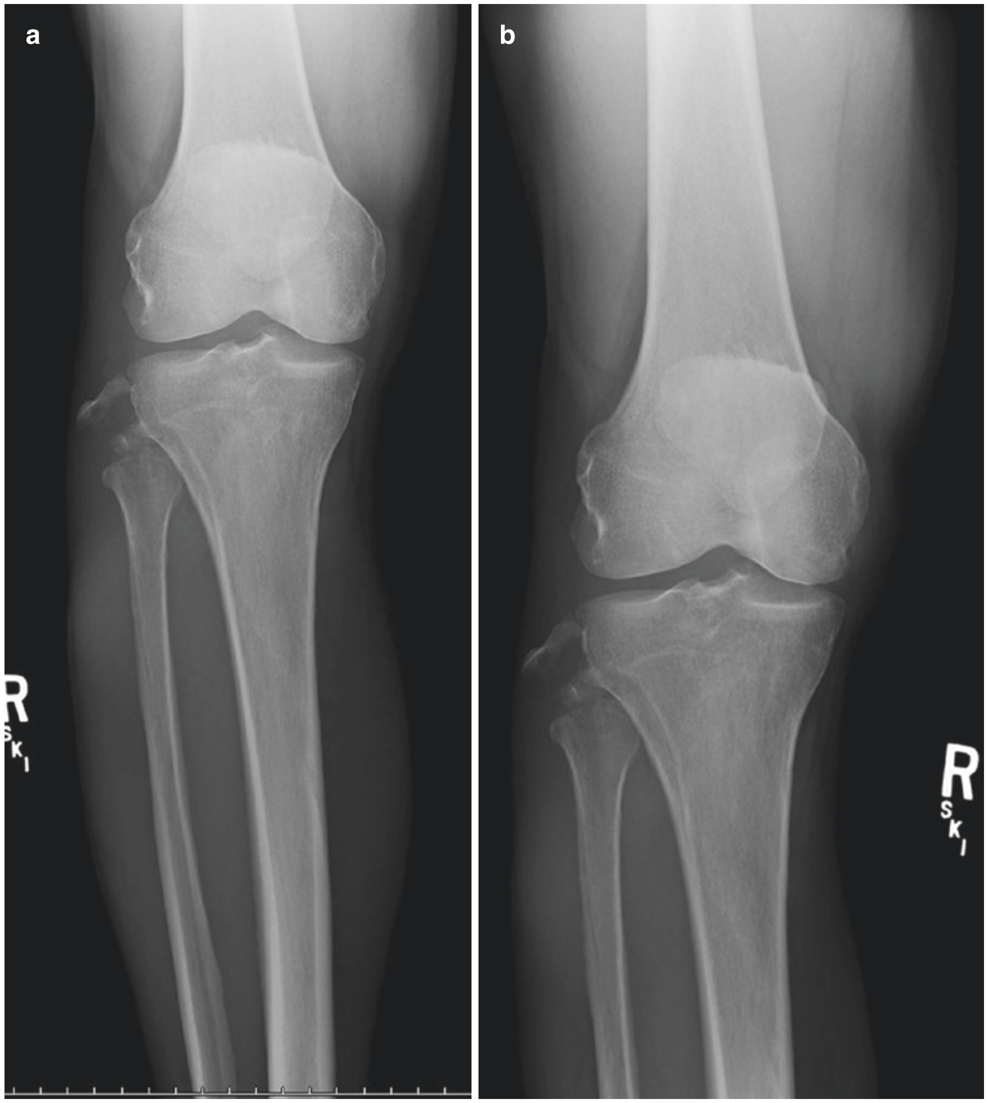

Fig. 25.16 A fibular avulsion fracture (arcuate sign) in a 45-year-old male skier (**a**, **b**) as a result of a tree strike (Courtesy of Dr. Kenneth Libre)

bearing as tolerated, but it is better to encourage non-weight-bearing and provide an alternative method for mobility (e.g., crutches), particularly during the first week after the injury. Counseling should be provided regarding risk for lateral compartment syndrome.

Indications for Orthopedic Referral

Emergent orthopedic referral is warranted for open fracture, suspicion for compartment syndrome, or neurovascular compromise [29]. Urgent operative indications are comminuted, segmental, or significantly displaced fractures with possible neurovascular complications. If there is nonunion, referral to orthopedic surgery is indicated.

Follow-Up Care

Stable, closed fibular fractures can be followed up in 3–7 days by a primary care physician. At that juncture, the athlete should be reevaluated for pain and neurovascular compromise. Provided that pain is controlled and there is no evidence of neurovascular deficits, the athlete should weight-bear as tolerated with ambulatory assistance. Follow up in 3–4 weeks for repeat plain radiographs. In athletes participating in contact sports, some recommend a physical regimen of weight-bearing as tolerated in a fracture brace or functional splint [40]. In about 3–4 weeks post-injury, if the athlete's pain is minimal, they should participate in gradual and controlled weight-bearing exercises for a total of 8–10 weeks [40]. In about 8 weeks post-injury, a final repeat radiograph should be taken to confirm appropriate union.

Most Maisonneuve fractures do not need specific management as long as the concurrent distal tibial fracture is fixed (Fig. 25.4). Stable Maisonneuve fractures that have partial syndesmotic diastasis have been reported to have successful outcomes with closed reduction via internal rotation followed by long-leg casting [43]. Casting should be for

6–12 weeks as long as there is no medial fracture. Repeat radiographs should be obtained every 3–4 weeks to ensure appropriate union.

Return to Sports

Return to play is individualized and based off their progression and any special health considerations they may have. Generally, a radiograph demonstrating appropriate union and tolerance of graduating physical activity without pain are promising indicators. The athlete may need training to understand how to better protect the fracture or avoid a re-fracture. Cases have been reported of refracture secondary to minor trauma in the later stages of healing [40]. This would result in a significant delay in return to play for the athlete.

Complications

Malunion/nonunion is a complication that may require operative fixation and bone grafting [29]. Refracture and lateral compartment syndrome are exceedingly rare, limited to only case reports [40, 52]. Common peroneal neuropathy is an uncommon complication as a result of a fibular head fracture [53].

Pediatric Consideration

A direct blow to the lateral leg is also the most common mechanism in children. Children tend to recover faster from isolated fibular fractures and typically require shorter time to heal [29]. If a proximal fibular fracture is diagnosed, a careful evaluation of the distal tibial epiphysis should be done as the two are associated in an adult Maisonneuve analogue [29]. A high school gymnast was reported to have sustained an isolated Salter-Harris Type III fracture through the proximal fibular epiphysis from a varus injury during a fall from the balance beam [41]. She recovered after casting and a rehabilitation program over the course of 7 weeks. Children tend to recover faster from isolated fibular fractures and typically require 2 less weeks to heal [29].

Diaphyseal Fractures of Both Tibia and Fibula

Mechanism of Injury in Sports

Diaphyseal fractures of both tibia and fibula occur secondary to similar mechanisms in isolated tibial diaphyseal fracture. A direct impact such as a soccer slide tackle to a planted leg is known to cause this fracture [20]. A smaller, but still prominent, percentage is secondary to sports such as skiing (Figs. 25.17, 25.18, and 25.19). In skiing, the mechanism dictates the type of fracture. During a fall forward, there is an anterior-posterior displacement or torsional strain on the fixed foot [21]. The "skier's boot top fracture (Fig. 25.19)" results from the hard boot and forward kinetic energy of the superior tibia, while the spiral fracture is secondary to torsional strain [21, 22]. Expanding to the general athletic population, this injury mechanism is either a direct impact or rotational torque exerted on the tibia that can be seen in both contact (e.g., basketball) and noncontact sports [23].

Fig. 25.17 Diaphyseal and comminuted tibia and fibula fractures in a 41-year-old male skier. Soft tissue swelling is concerning for developing acute compartment syndrome

Epidemiology

There is a unimodal distribution of young males that sustain high-energy trauma and older females involved in low-energy trauma [18, 19]. The incidence of tibial diaphyseal fractures with concomitant fibular diaphyseal fracture is about 7% of all fractures sustained by soccer players in an 18:0 ratio of males to females [25]. Males, with a peak incidence age 10–20 years, tend to be injured secondary to sports activities, while females who are older than 30 years are injured secondary to walking and indoor activity [19].

Classification

The AO Foundation and Orthopaedic Trauma Association (AO/OTA) classification for diaphyseal fractures is widely accepted [28, 48]. Classification for a concomitant tibia and fibula diaphyseal fracture must be described independently.

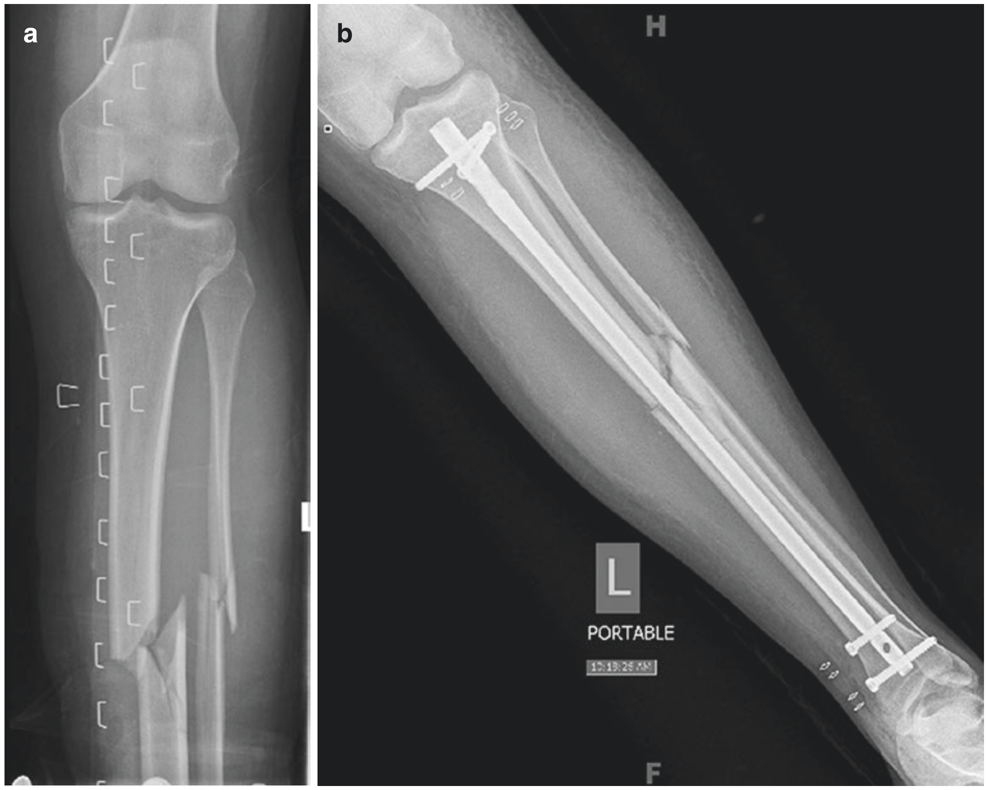

Fig. 25.18 Diaphyseal and comminuted tibia and fibula fractures in a 22-year-old female skier (**a**). Tibia fracture was treated with an intramedullary rod (**b**)

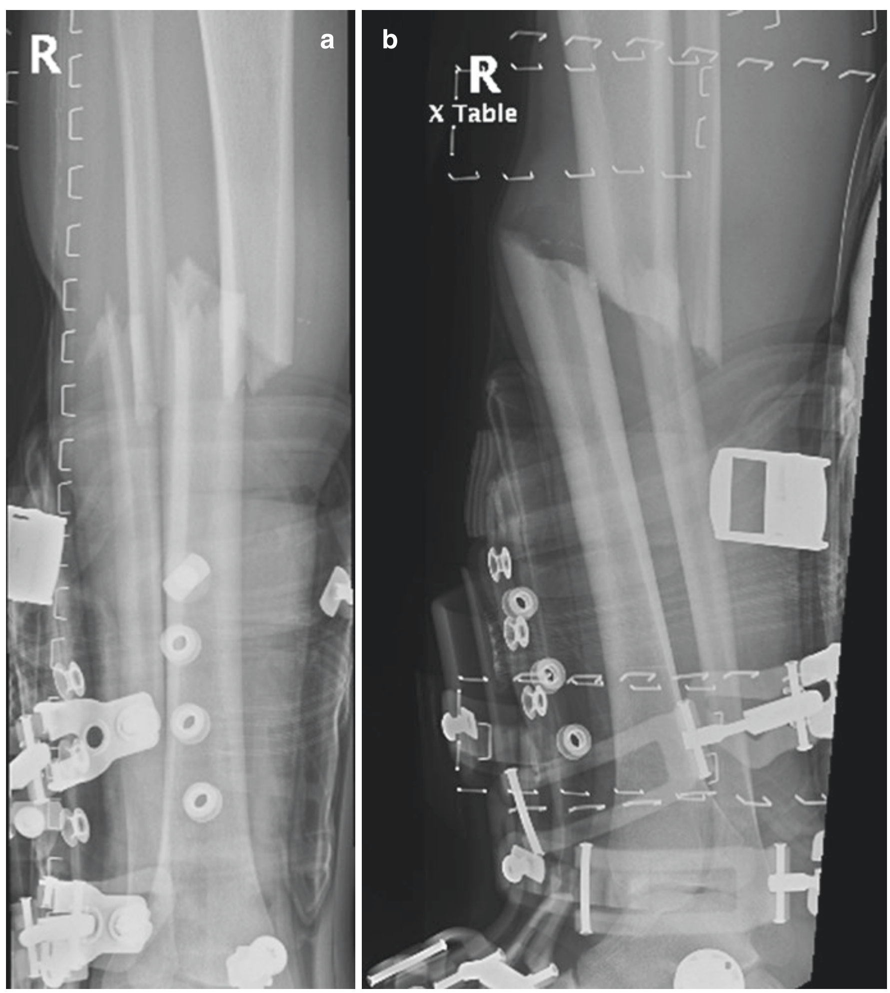

Fig. 25.19 Open and displaced diaphyseal tibia and fibula (boot top) fractures in a 15-year-old male skier (**a**, **b**)

Clinical Presentation

The athlete will present to the sideline unable to bear weight on the affected side. Given the significant force normally required to fracture both the tibia and fibula, there is often visible deformity or break in the skin. At times, there is significant bleeding associated with the injury. At the least, the athlete should have pain and/or swelling at the site of injury. Occasionally, they may report weakness and/or paresthesia associated with nerve injury. Exposure of the affected extremity is crucial. With skiers, great care should be taken to remove the boot as it is likely at the site of fracture. The skin should be closely examined for tense compartments and to determine if the fracture is open. The affected limb should have point tenderness and/or swelling associated at the site of fracture. A neurovascular examination evaluating the dorsalis pedis (DP) and posterior tibialis (PT) pulses and lower extremity strength and sensation should be documented.

Diagnosis

Diagnosis of a tibia-fibula diaphyseal fracture is confirmed by radiography (Fig. 25.18). AP and lateral views are recommended along with radiograph series of the knee and ankle. These images will help determine classification of fracture and management. The amount of displacement, angulation, and rotation are key factors that influence operative versus nonoperative treatment [29].

Initial Management

The fracture should be immobilized in a well-padded long-leg posterior splint. The splint, being non-circumferential compared to a cast, allows for swelling that typically occurs after this injury. Due to the force required and the extent of the injury, the athlete is at increased risk for developing compartment syndrome. Elevation and icing of the affected extremity are important to reduce swelling. If pain cannot be controlled with oral medications, consider admitting the patient for monitoring and parenteral administration of pain medication. All athletes with both tibia and fibula fractures, particularly with unstable fractures, should be stabilized and transported to a facility with access to orthopedic surgery care.

Indications for Orthopedic Referral

Emergent orthopedic referral is warranted for almost all patients with both tibia and fibula fractures, unless the fractures are nondisplaced and stable and no comminution is present. Urgent operative indications are unstable, comminuted, and/or segmental fractures as disability, malunion, and nonunion complication rates increase (Figs. 25.17, 25.18, and 25.19) [30]. In general, the management of tibia-fibula diaphyseal fractures is dependent on the state of the tibia. Fixation of the fibula with an intact interosseous membrane does not seem to contribute to the stability of a tibial diaphyseal fracture [54]. However, a concomitant fibula fracture or significant displacement does increase the risk of failed closed treatment [55]. Please refer to the section on tibial diaphyseal fractures regarding the potential benefits and consequences of conservative management. Although reviews so far do not demonstrate superiority of operative compared to nonoperative management for closed tibial shaft fractures, a tibia-fibula fracture warrants orthopedic surgical evaluation [34, 35].

Follow-Up Care

Even the fact that some experienced primary care physicians may be able to manage stable, closed, and nondisplaced fracture of the tibia-fibula, it is appropriate to refer all patients with both tibia and fibula diaphyseal fractures to orthopedic surgery. Treatment protocols will vary across institutions and are athlete dependent [20]. In general, the follow-up protocol is the same as in patients with isolated tibial diaphyseal fracture.

Return to Sports

Return to sports should follow the same rule as in patients with isolated tibial diaphyseal fractures. However, due to the increased risk of malunion, nonunion, and instability, it seems that the return to sports is longer among athletes with both tibia-fibula fractures compared to the athletes with isolated tibial diaphyseal fractures.

Complications

Tibia-fibula diaphyseal fractures have one of the higher rates of complications when compared to isolated tibia or fibula diaphyseal fractures [20]. They can become displaced after immobilization attempts, develop compartment syndrome, and have delayed union or refracture [20]. Please refer to the section on tibial diaphyseal fractures for more on epidemiology of these conditions.

Pediatric Consideration

In adolescents, the concomitant tibia-fibula diaphyseal fracture is cited as 4% in an 88:12 ratio of males to females [2]. In skiing, children typically do not have an associated fibular fracture [21]. Closed reduction and casting is the mainstay of treatment, but operative management is considered on an individualized basis [37]. Valgus deformity can occur in children with both tibia-fibula fractures as a result of the anterior and lateral muscular traction.

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Ankle Joint

26

Morteza Khodaee, Matthew Gammons, and Kenneth J. Hunt

Key Points

- Ankle injuries including fractures and sprains are common in sports.
- Ottawa ankle rules can be used to determine need for ankle radiographs in the acute setting.
- Many ankle fractures can be treated conservatively.
- Assessing for ankle joint stability is important.
- Fractures with evidence of instability should be considered for surgical fixation.
- Ankle joint dislocations are rare in sports and are usually associated with concurrent fractures.

Introduction

Ankle injuries are some of the most common injuries seen during sporting activity. Sport makes up a large percentage of reported ankle injuries in the general population [1, 2]. While soft tissues injuries such as lateral ligament sprains make up most injuries, ankle fractures are seen relatively commonly in many sports [3, 4]. Many of these injuries have similar mechanism to ankle sprains, but higher-energy traumas can occur creating joint instability. Lateral malleolus fractures are the most commonly reported fracture pattern [5]. Fractures that maintain normal joint alignment can be treated with protected weight-bearing as tolerated and early range of motion [5–7]. Fractures that are displaced or have evidence of joint instability should be considered for surgical repair to restore normal joint alignment and biomechanics [7]. Restoration of ankle range of motion and strength are the goals of early rehabilitation, and return to play will vary based on the type of injury and individual athlete.

Anatomy

Stability and function of the ankle joint is provided by both boney and ligamentous stabilizing structures (Figs. 26.1 and 26.2). The tibiotalar joint, also known as the talocrural joint, is made up of the medial and lateral malleoli, the talus, and the distal tibial weight-bearing surface, otherwise known as the tibial plafond. The alignment of the ankle is commonly referred to as the mortise, which describes the anatomic association of the boney structures (Figs. 26.1 and 26.2). The talus articulates with the malleoli and the plafond throughout range of motion (Chap. 28; Fig. 28.1). Because the talus has a trapezoidal shape with a wider anterior aspect and more narrow posterior aspect, ankle movement affects position of the other boney structures (Chap. 27; Fig. 27.12). In plantar flexion, the talus internally rotates, and in dorsiflexion, talus creates both external rotation and lateral translation of the fibula to accommodate the wider anterior portion of the talus. The ankle is most stable in dorsiflexed position due to this anatomic relationship [5].

The ankle is also supported statically by three main groups of ligaments (Fig. 26.2). The medial ankle is supported by the deltoid ligament which has both superficial and deep layers. The superficial fibers originate from the medial malleolus and inserts on the navicular, the calcaneus, and the talus with a small attachment of the spring ligament. The deep fibers originate on the medial malleolus and insert on the talus. The deltoid ligament is the primary static

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

M. Gammons
Concussion Clinic, Vermont Orthopaedic Clinic, Killington Medical Clinic, Rutland Regional Medical Center, Rutland, VT, USA
e-mail: mgammons@rrmc.org

K. J. Hunt
Department of Orthopedics, Division of Foot and Ankle, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: KENNETH.J.HUNT@CUANSCHUTZ.EDU

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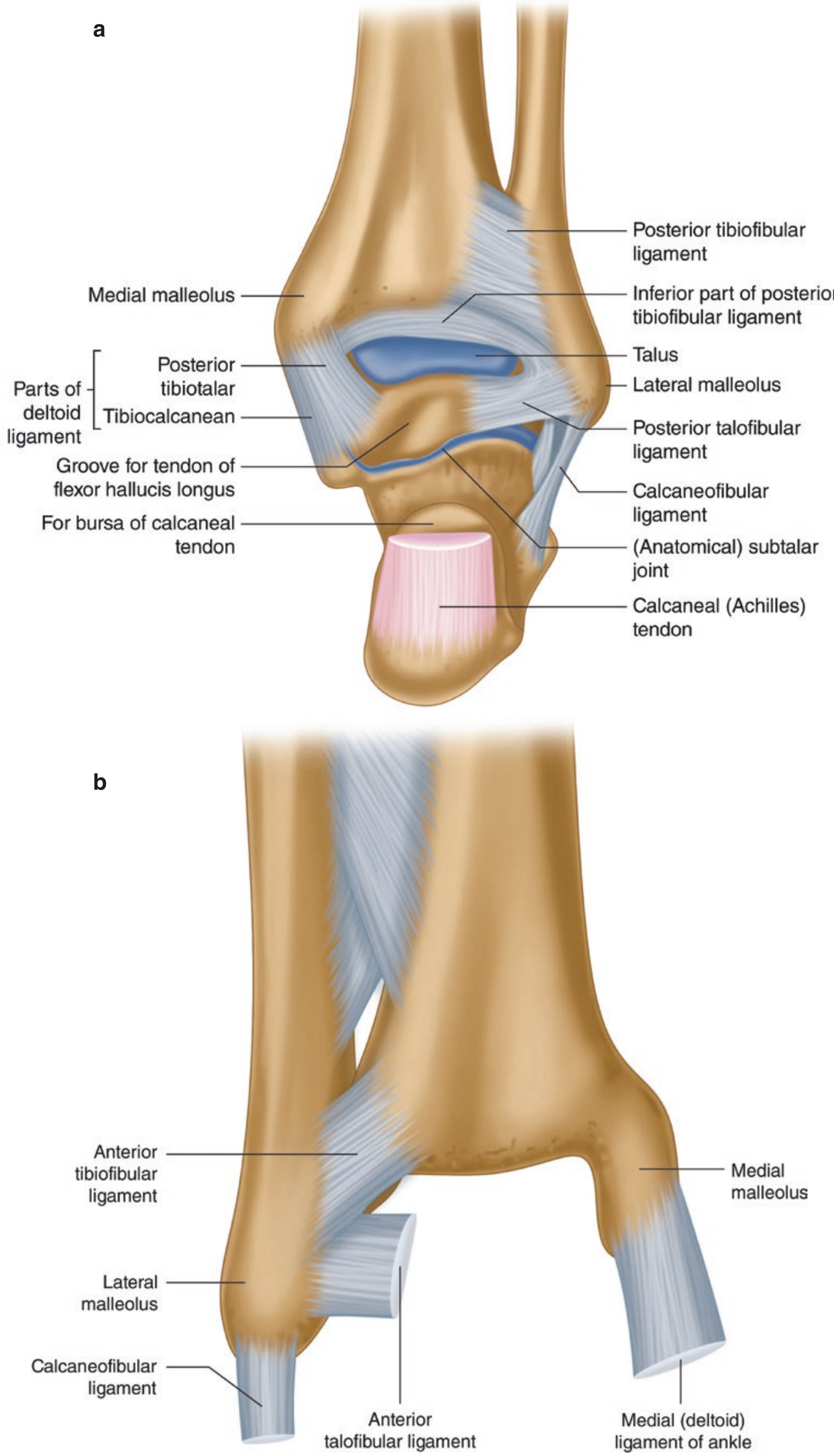
Fig. 26.1 Bony anatomy of the ankle joint. Posterior view demonstrates relationship with ligaments (**a**). Anterior view demonstrates tibiofibular joint and related ligaments (**b**)



stabilizer and prevents external rotation and lateral translation of the talus during weight-bearing [5]. The lateral support consists of three main ligament structures, the anterior talofibular (ATFL), calcaneofibular (CFL), and posterior talofibular ligament (PTFL). These structures are secondary stabilizers that aid in restriction of anterior displacement of the talus (ATFL), excessive supination (CFL), and inversion and internal rotation (PTFL) [8].

Finally, the tibiofibular syndesmosis is an important structure in ankle stability (Figs. 26.1 and 26.2). It consists of the anterior (AITFL) and posterior inferior tibiofibular ligaments (PITFL), the inferior transverse ligament, and the interosseous ligament [5, 9]. There is a small area of hyaline cartilage at distal tibia and fibula contact that is supported by these ligaments [9]. These ligaments resist the medial and lateral translation as well as external rotation forces [9].

There are multiple muscle/tendon complexes that pass around the ankle, but there are not direct boney attachments in the ankle joint (Chap. 28; Fig. 28.2). The subtalar joint is considered by some to be a lesser ankle joint. It if formed by articulation between the talus and the calcaneus and allows for pronation and supination of the foot and ankle [8, 10]. The anterior subtalar joint formed by the head of the talus, the anterior and superior facets, and the sustentaculum tali of the calcaneus (Chap. 27; Fig. 27.1). Anteriorly it articulates with the navicular. The posterior subtalar joint is comprised of the posterior inferior facet of the talus and the superior posterior facet of the calcaneus [8, 10]. The subtalar joint is reinforced by a complex group of ligaments whose anatomy has not been completely defined. These ligaments have been described in several anatomic studies with most of the structures restraining supination and internal rotation [10].

Soft Tissue Injuries

Ankle sprains are some of the most commonly reported injuries in sports [1, 8]. Sprains of the lateral ankle complex are more commonly reported than injuries to the syndesmosis or medial ligaments [1, 8]. These injuries occur with an inversion mechanism, and athletes will usually describe "rolling" their ankle while running or jumping. They sometimes involve stepping on another player's foot or uneven ground or twisting injuries while changing directions. Pain

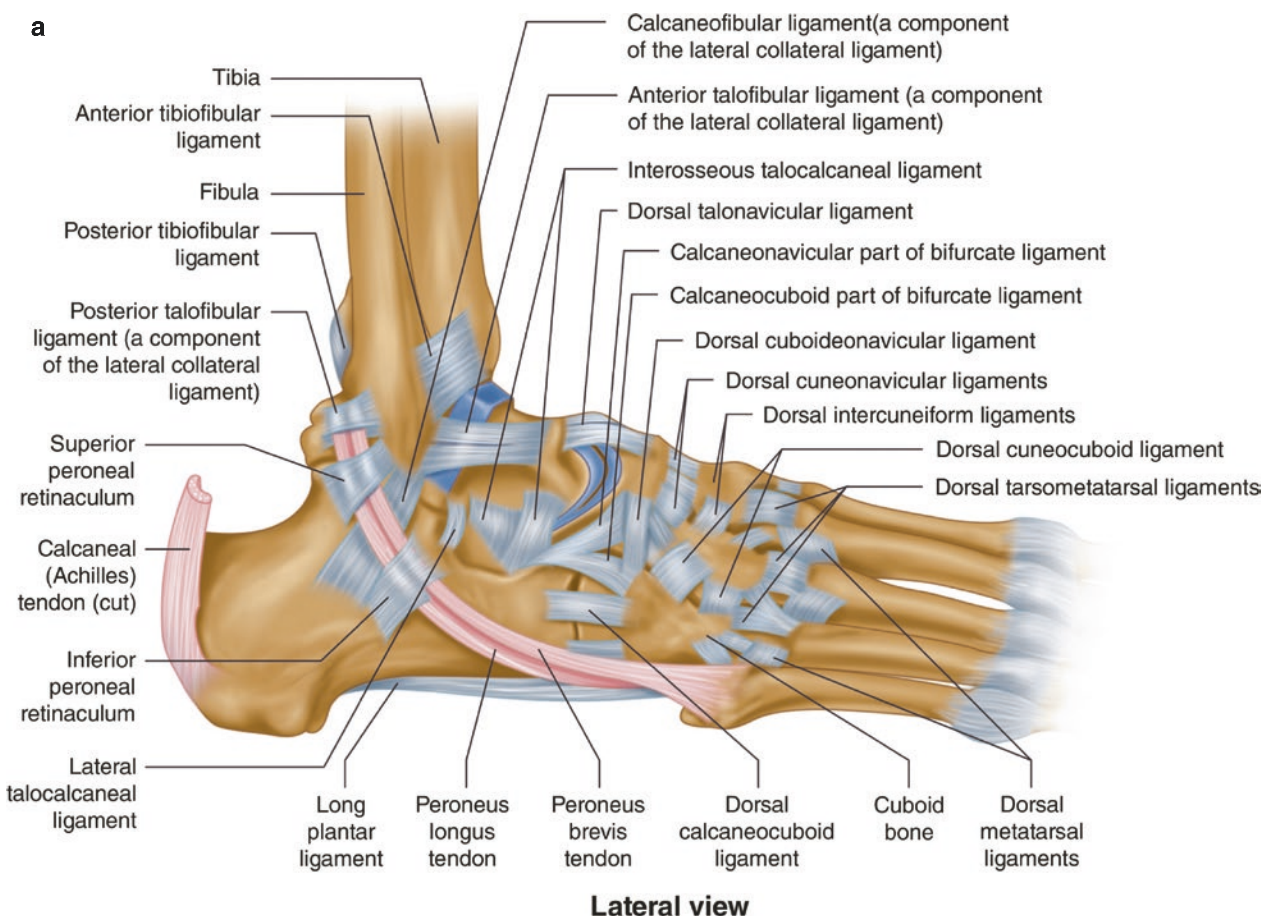


Fig. 26.2 Ankle and foot ligamentous and tendinous anatomy. Lateral (**a**), medial (**b**), and superficial (**c**) and deep (**d**) plantar views

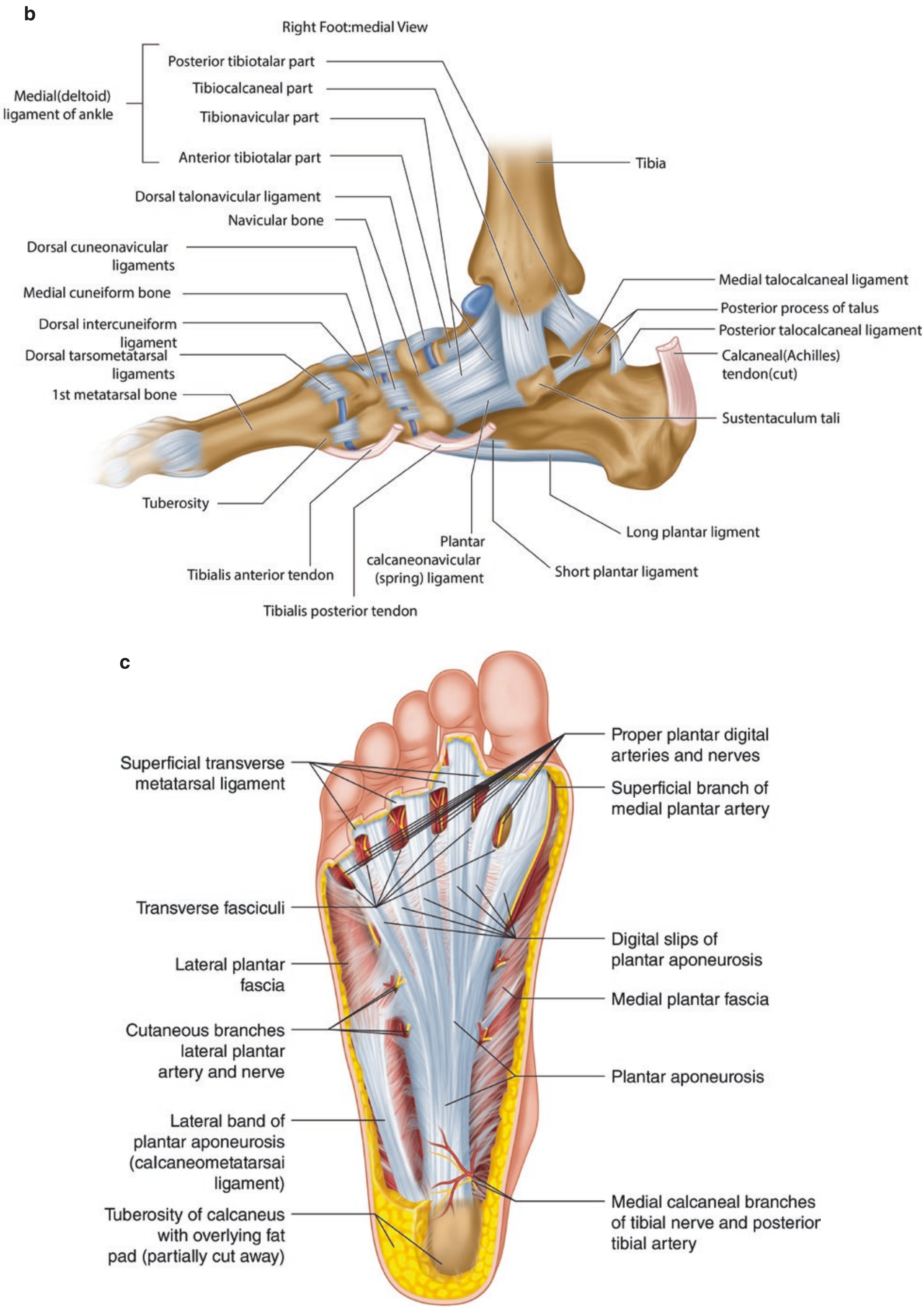


Fig. 26.2 (continued)

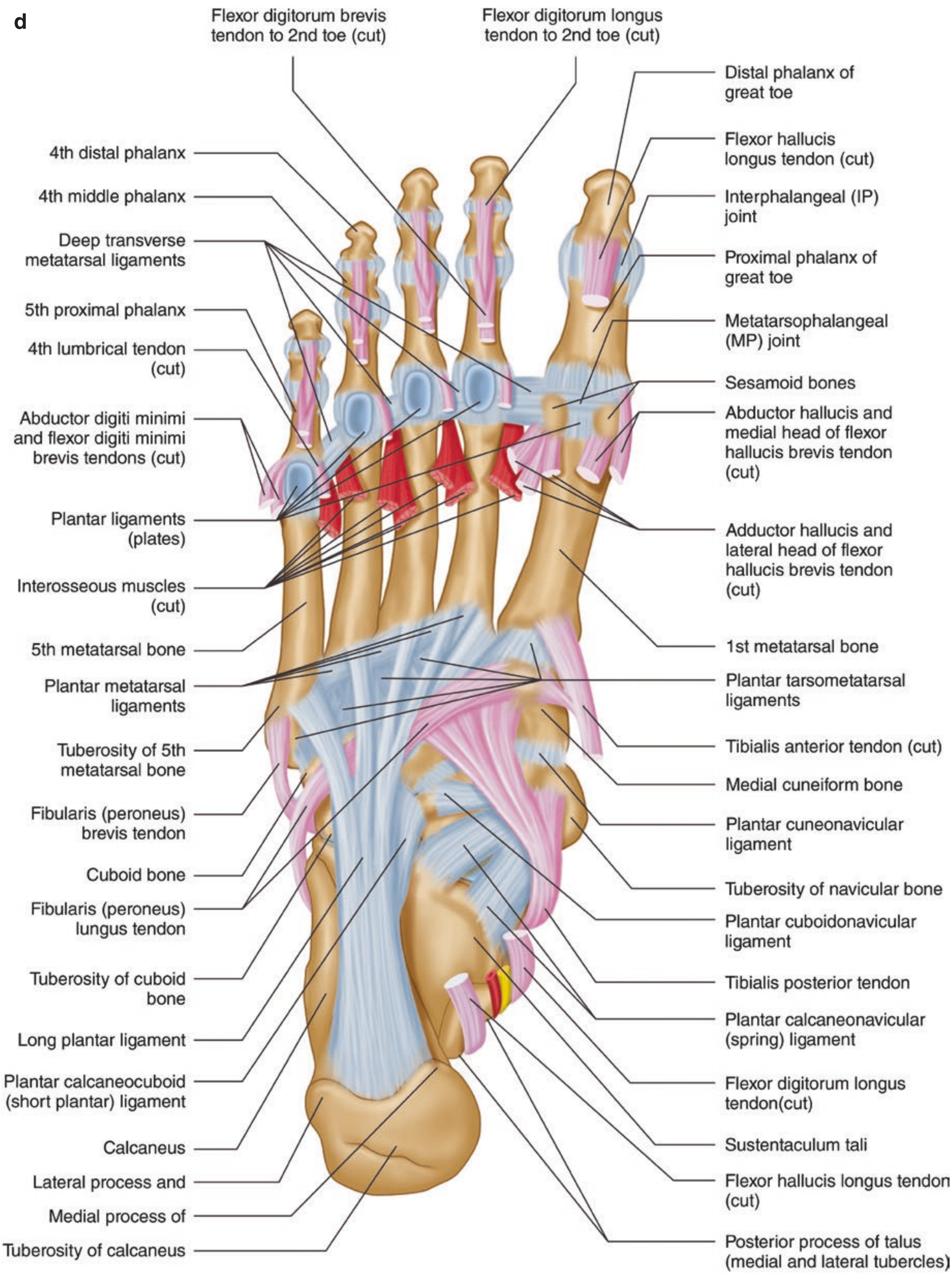


Fig. 26.2 (continued)

and swelling are common and ability to weight-bear is variable. Ecchymosis is common but may be a delayed finding. The Ottawa ankle rules can be used to determine the need for radiographs in acute settings [11]. Treatment consist of protected weight-bearing, boot or brace, and early range of motion exercises have been shown to be effective [1, 8]. Some athletes with higher-grade lateral ankle sprains will have persistent instability requiring further evaluation and treatment [1].

Injuries to the tibia syndesmosis most commonly occur when the ankle is in a dorsiflexed position and the foot is externally rotated. These usually occur through player contact and generally require longer recover than lateral ankle sprains [1, 12]. Players will present with pain, swelling, and occasionally ecchymosis and often have difficulty with weight-bearing. Tenderness over the syndesmosis combined with pain with squeeze or external rotation test is considered diagnostic. Imaging may be indicated based on clinical presentation [11]. Standing AP ankle radiographs can be used to determine joint stability (Fig. 26.3) [1]. Further imaging such as stress radiographs or MRI may be considered but may not add to the assessment of joint stability [1]. Treatment typically consists of protected weight-bearing until pain resolves, but athletes with evidence of joint instability typically require surgical intervention [1].

Deltoid ligament injuries occur commonly with high-grade syndesmotic injuries. Isolated deltoid injuries are less commonly reported than either lateral or syndesmotic injuries and often occur with other soft tissue or boney injury [13, 14]. Forceful eversion usually caused by player contact is the most commonly reported mechanism. These injuries also can occur with landings such as in gymnastics [14]. Medial-sided tenderness with associated swelling and ecchymosis is usually present. Imaging to rule out associated fractures may be needed [11]. Additional imaging requirements vary based on the injury. Treatment is similar to other ankle ligament injuries with protected weight-bearing and progression as symptoms improve. Surgical indications are not clear but may be considered for unstable injuries [13]. More details on these ligamentous injuries are discussed in Chap. 36.

Fractures

Ankle fractures are among the most common injuries in sports [15, 16]. Ankle fracture accounts for an estimated 15% of patients presenting to the emergency departments with ankle injuries [16]. Ankle fractures are divided as isolated malleolus (medial, lateral, and posterior), bimalleolar, trimalleolar, and pilon fractures [15, 16]. Isolated malleolar fractures count for about two-thirds of ankle fractures [17]. Bimalleolar and trimalleolar fractures count for about 25% and 10% of all ankle fractures, respectively [17–19].

Lateral Malleolus

Mechanism of Injury in Sports

Injuries to the lateral malleolus typically occur with an inversion mechanism similar to ankle sprains or with supination and external rotation [20]. The supination and external rotation injuries are thought to occur with a fixed foot such as when an opponent steps on another player's foot; however, other mechanisms may be responsible in some cases [5].

Epidemiology

Isolated fractures of the lateral malleolus are the most commonly reported ankle fractures [5, 21]. While falls are the most commonly reported mechanism, sports-related injuries rank second in several series [17, 21]. Overall distal fibular fractures in sport are much less common, only 4 out of almost 4000 injuries to the foot and ankle in college athletes over a 2-year period [22]. Given the mechanism, these injuries do have the potential to occur in many sporting environments. Soccer, rugby, and American football are commonly reported sports, but fractures have been reported in other sports such as field hockey and wrestling [3, 4, 23, 24].

Classification

There are two commonly used classification systems, the Lauge-Hansen and Danis-Weber (Table 26.1). Both systems attempt to define stable versus unstable fracture patterns but do not alone guide treatment. The Lauge-Hansen classification (Fig. 26.4) is based on the position of the foot at the time of injury, supination or pronation, and describes not only injury to the lateral malleolus. Supination-external rotation (SER) injuries types 2 and 4 describe injuries to the lateral malleolus with and without ligamentous instability [25]. More recent studies have shown that this classification system correlates only 65% of the time with video reviews of injury mechanism [26]. The Danis-Weber classification (Fig. 26.5) considered stable unless there is more than 5 mm of medial space widening. A recent study found this system to be reproducible and comparable to Danis-Weber but did not compare outcomes of patients [27].

Clinical Presentation

Athletes will typically present similar to severe ankle sprains. Swelling and ecchymosis are usually present. The ability to weight-bear is variable, and range of motion may be limited. Athletes who can fully weight-bear after the injury are significantly more likely to have a stable injury [28]. Tenderness over the fibula in the area of the fracture is common. Associated tenderness of the syndesmosis and medial soft tissues may also be present. However, the presence or absence of medial-sided tenderness does not predict ankle stability [29].

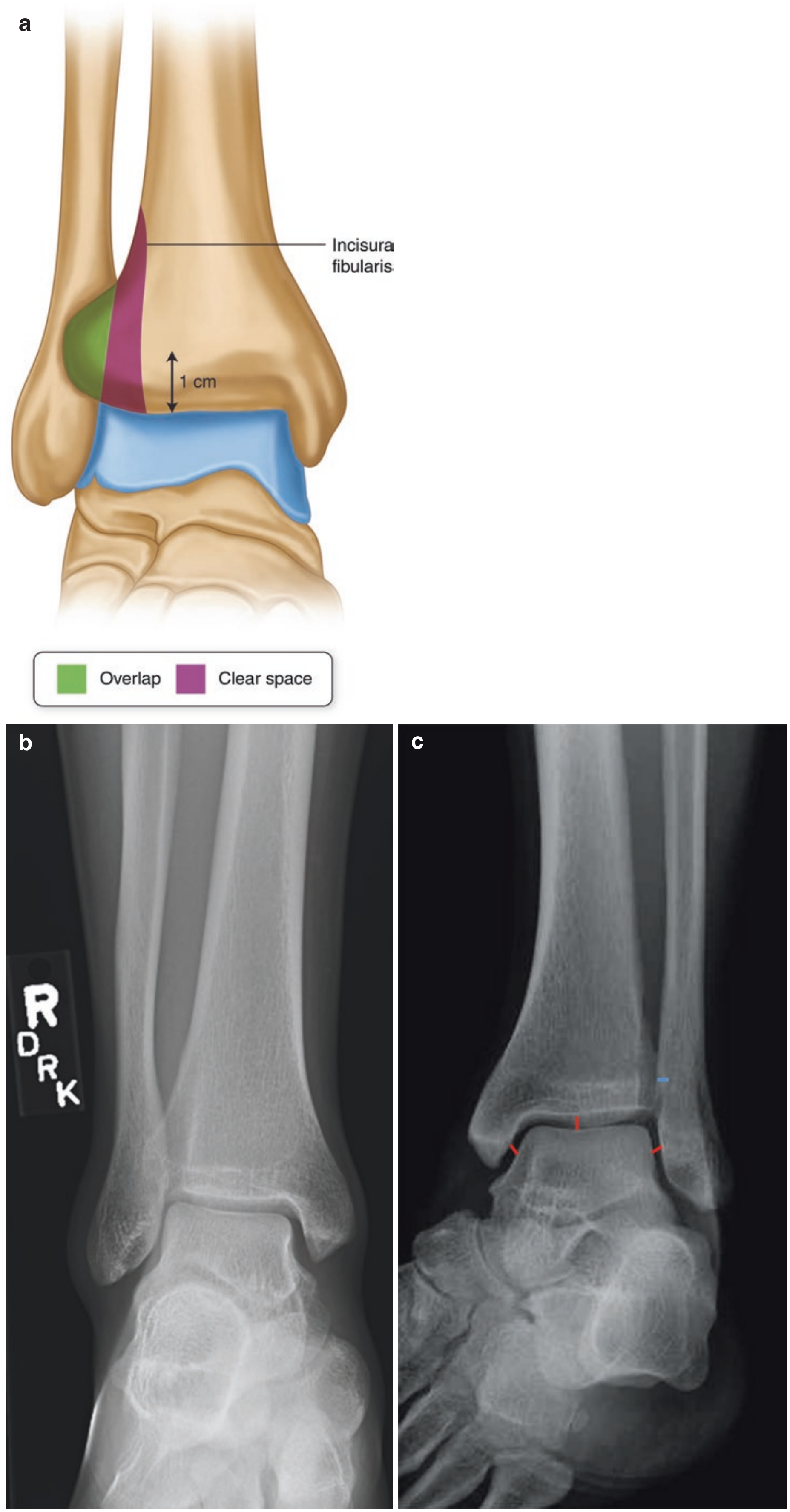


Fig. 26.3 Schematic (**a**) demonstration of the tibia-fibula overlap and its correlation in plain radiography AP (**b**) view. Tibiofibular clear space (between the fibula and the peroneal incisura of the tibia) is normally <4 mm. Tibiofibular overlap <6 mm is considered abnormal. Tibiofibular overlap (blue line) <1 mm in mortise view (**c**) is abnormal. Uniform 3–4 mm space around the talus (red lines) is normal

Table 26.1 Comparison of Lauge-Hansen and Danis-Weber classification for lateral malleolus fractures [5]

Lauge- Hansen (Fig. 26.4)	Danis-Weber (Fig. 26.5)	Description
SER 2	Type A	Below level of syndesmosis
		Stable
SER 4	Type B	At level of syndesmosis
		Stable or unstable
	Type C	Above level of syndesmosis
		Unstable

Diagnosis

In addition to physician examination, radiographs of the ankle can help to determine both the presence of a fracture and stability of the joint (Figs. 26.6, 26.7, 26.8, 26.9, 26.10, and 26.11). The Ottawa ankle rules can be used in the acute setting to determine the need for radiographs [11]. These rules have not been validated on the sideline but are well validated in other acute settings. Inability to weight-bear at the time of injury or examination and tenderness along the posterior edge and tip of the lateral malleolus should prompt radiographs [11]. These rules were designed to detect clinically significant injuries and may miss more stable injuries such as avulsion fractures

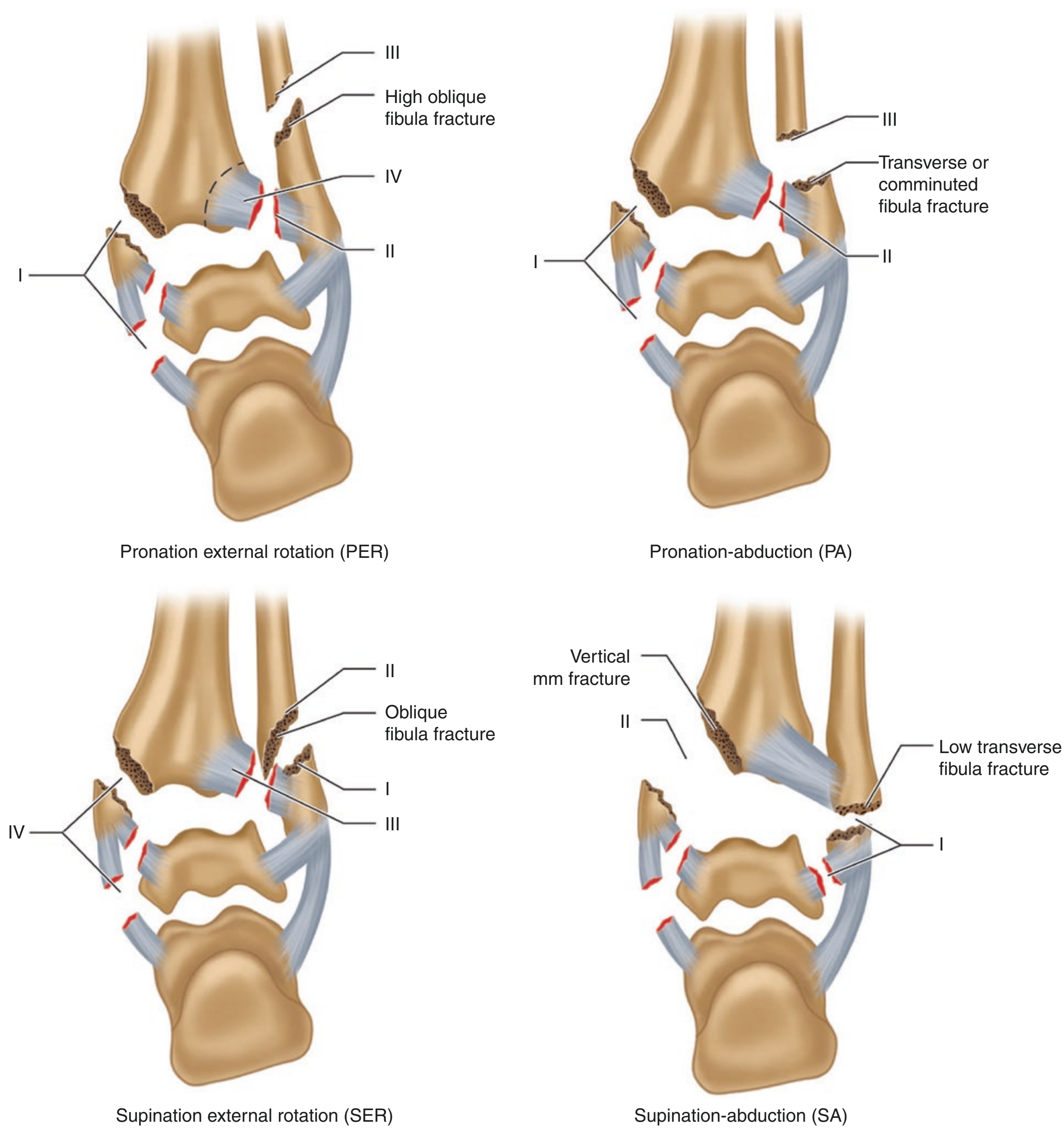


Fig. 26.4 Lauge-Hansen classification of ankle fracture

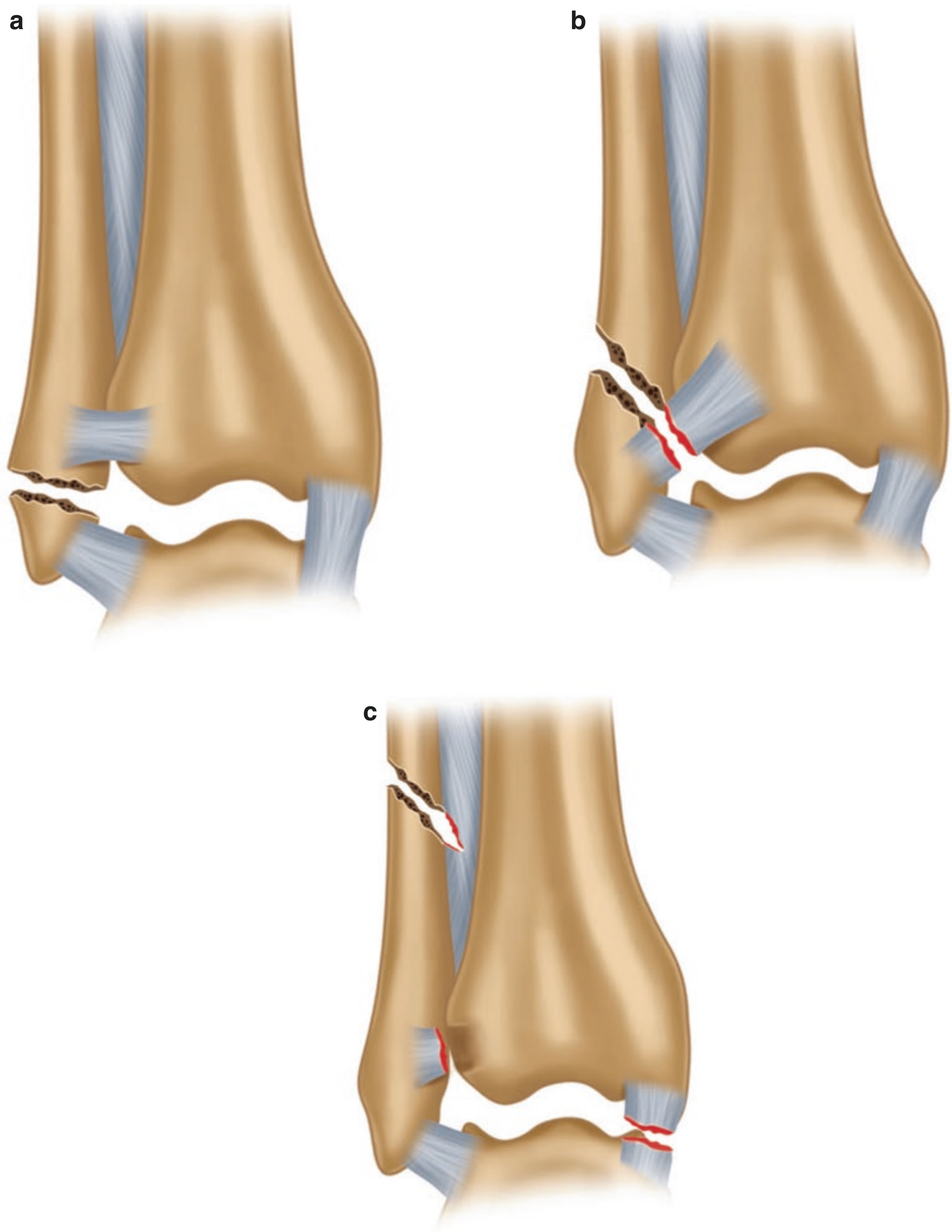


Fig. 26.5 Danis-Weber classification of distal fibula fracture. Type A (**a**), type B (**b**), and type C (**c**)

(e.g., Danis-Weber A). Because in athletes this information may change return-to-sport strategies, individual decisions on radiographs may be made in selected cases. Standard radiographic views include AP, mortise (oblique), and lateral, with weight-bearing to tolerance. Stability is assessed by measuring the medial clear space which is defined as the distance from the superior medial talus to the superior lateral medial malleolus at the level of the talar dome (Fig. 26.3) [5]. Some suggest a space greater than 5 mm is suggestive of instability; however, there are high rates of false positives using this method alone [30]. Because of this, additional radiograph techniques can be used to help determine stability. Manual stress radiographs are performed with the ankle in a neutral position. Using both hands, the tibia is internally rotated 10° while an external rotation force is applied to the foot [5]. This technique has the advantage of applying dynamic forces to the ankle; however, it is technically difficult, may have issues with reproducibility and radiation exposure of the assistant, and may cause pain to the patient [5]. The gravity stress view can also be used to help determine stability. The patient is placed in the lateral decubitus position, medial malleolus up, with approximately half the leg extended over the table. A pillow or rolled towel is placed under the leg as this allows the foot to fall into external rotation [5]. This technique does not require assistance and is generally well tolerated by the patient. Studies have found this to be effective as manual technique [31, 32]. Both stress radiograph techniques may overestimate instability of the deltoid ligament and syndesmosis, so some authors suggest the use of a weight-

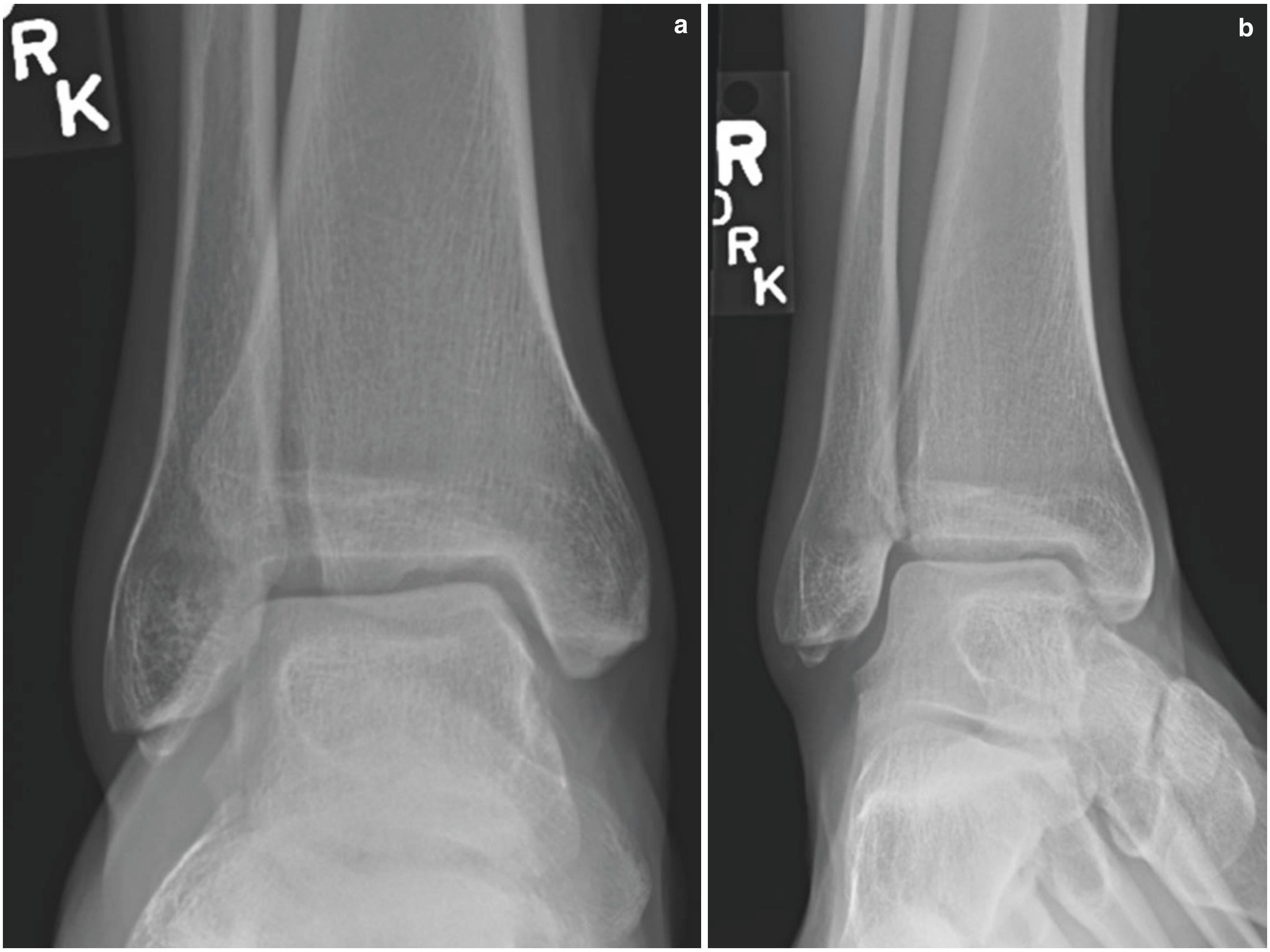


Fig. 26.6 An avulsion fracture of the distal fibula in a 30-year-old male and a result of a ski injury (**a**, **b**)

bearing radiograph [5, 33]. Weight-bearing radiographs allow for evaluation the inherent stability of the ankle joint but are influenced by how much weight the patient is able to place on the affected limb [5]. The decision on which of these techniques to use will depend on provider preference and the individual patient. Ultrasound has been shown to be effective in the evaluation of acute ankle injuries. It has both good sensitivity (87%) and specificity (96%) for lateral malleolus fractures, and when used in conjunction with the Ottawa ankle rules, ultrasound can increase the specificity and aid in ruling out the need for radiographs [34, 35]. However, this technology is not as commonly available in the clinical setting and has not been shown to demonstrate stability. MRI does not generally add to the evaluation of lateral malleolus fractures as findings of injury to the deltoid ligament are common but do not correlate well to instability [36]. An exception is a suspected injury to the syndesmosis that is not clearly demonstrated on radiographs.

Initial Management

Initial immobilization depends on the fracture and individual patient. Stable fractures can be protected in a splint, boot, or brace, and weight-bearing can be allowed as tolerated. Crutches or other assistance can be used for severe pain in a short-time (<7 days) immobilization. A short leg posterior or U-splint can be considered with some authors suggesting transition to a short leg cast for several weeks prior to transition to a boot and weight-bearing [5]. Early range of motion exercises can begin as soon as pain allows. Unstable injuries can be protected similar to stable injuries (e.g., splint or boot), but most recommendations include limited weight-bearing. However, evidence is lacking on the effect of this weight-bearing to tolerance on these injuries. Patients in both cases should be given instructions for swelling management (e.g., elevation and on monitoring for signs for compartment syndrome).

Fig. 26.7 Mildly displaced distal fibula (Weber A) fracture in a 60-year-old male as a result of a ski injury while getting off of the chairlift (**a**–**c**)

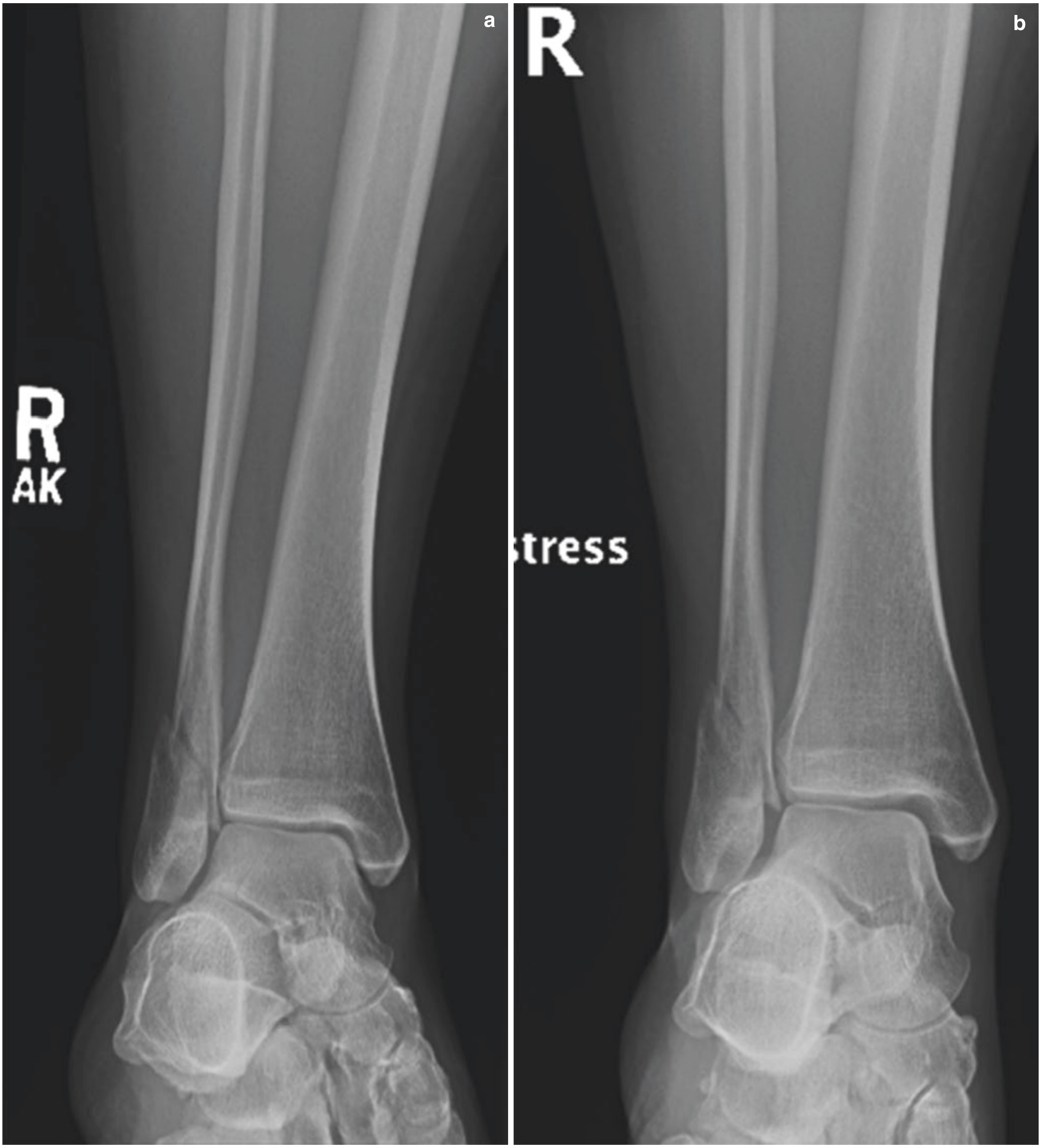


Fig. 26.8 Mildly displaced distal fibula (Weber B) fracture in a 47-year-old female as a result of a ski injury (**a**). Stress view shows mild widening of the talofibular space (**b**)

Indications for Orthopedic Referral

Although rare, athletes with neurovascular deficits and those with open fractures should be immediately transported to the nearest emergency department for evaluations and management. Other indications include unstable injuries and injuries with significant associated peroneal nerve injury. For non-displaced unstable injuries, defined as no widening on normal radiographs but widening on stress or weight-bearing films, both surgical and conservative treatments have been found acceptable in the large reviews. However, these studies

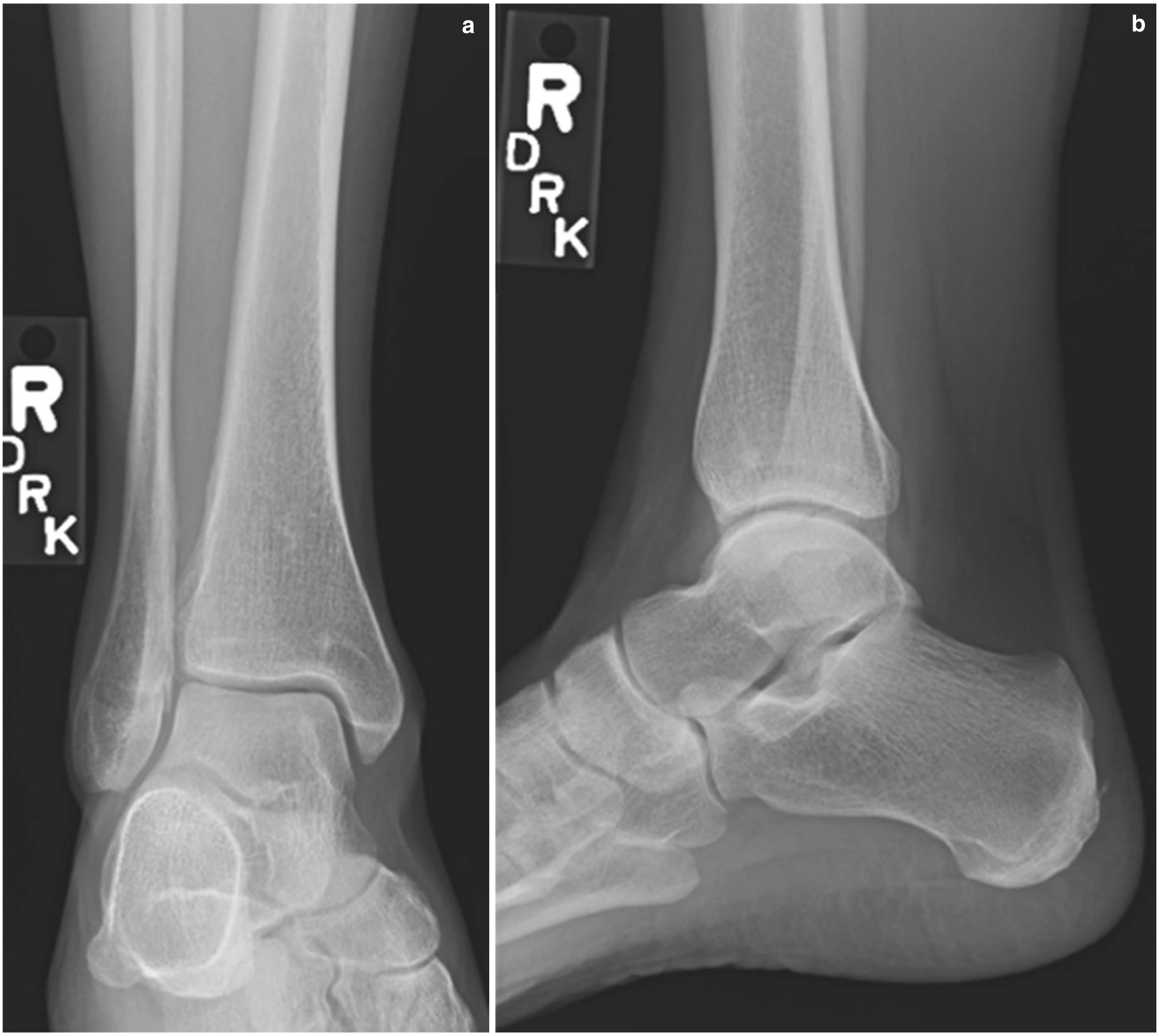


Fig. 26.9 Mildly displaced distal fibula (Weber B) fracture in a 59-year-old female (**a**, **b**)

typically did not include athletes, so it is unclear if results can be translated to higher-demand patients. Surgeon and athlete preference will dictate care in these circumstances [7].

Follow-Up Care

Stable injuries can be followed based on provider preference. A repeat x-ray and evaluation at 6 weeks to asses for fracture healing may be adequate in cases where athletes are able to weight-bear early and have good pain control. Repeat radiographs in 7–10 days can be considered for cases where the initial stability is not clear. For patients with more severe pain, follow-up can be considered at closer intervals to ensure improvement in pain and for re-evaluation of other possible injuries. For patient who have surgical fixation, follow-up intervals will vary by surgeon preference. Use of a function brace or boot, early mobilization, and weight-bearing after surgery have been shown to be safe and effective. Studies have shown improved outcome scores, and for athletes early mobilization allows early progression to rehabilitation and return to play [37–40].

Return to Sports

Several studies, including those with isolated fractures of the lateral malleolus, have found athletes are able to return to activity after these injuries with 83% of patients returning to military duty and 96% of younger patients returning to preinjury levels [41, 42]. There are no specific guidelines for lateral malleolus injuries. Once athletes are fully weight-bearing

Fig. 26.10 Displaced bilateral distal fibula (Weber B) in a 76-year-old male as a result of a ski injury (**a**, **b**)

and have normal strength and balance, they may progress back into higher-level activities. For some sports, this may be accomplished over 3–6 weeks for stable avulsion injuries that can be protected in a functional brace. Other injuries may require more time to regain necessary functional strength and performance, but early mobilization and weight-bearing as tolerated decreases time to return to play [39].

Complications

Post-traumatic arthritis is the most commonly reported complication with the rates increased in cases involving malunion altering joint biomechanics. Some studies, however, have not found increased rates in non-displaced unstable injuries [5]. Pain, peroneal nerve dysfunction, and weakness are additional potential complications in both conservatively and surgically managed fractures.

Pediatric Considerations

Ankle injuries are common in pediatric athletes, and the Ottawa ankle rules have been validated in children over the age of 5 years [43]. Concern for occult injuries of the lateral physis of the distal fibula, Salter-Harris type I, may prompt providers to take a conservative approach to pediatric patients even in the setting of negative radiographs. MRI studies, however, has shown that when suspected Salter-Harris type I injuries are rare and when present, they heal with similar treatment to ankle sprains [44]. Other types of Salter-Harris injuries are rare [45]. An accessory ossification center is a known anatomic variation seen at the distal tip of the fibula and should not be mistaken for an avulsion injury [45].

Medial Malleolus

Mechanism of Injury in Sports

Exact mechanism of isolated medial malleolus fractures in sports have not been well described. Injuries to this area typically occur with pronation and external rotation [46].

Epidemiology

Isolated fractures of the medial malleolus represent 7–10% of fractures in large reviews [6, 21, 47]. They appear to be much less common than lateral malleolus fractures representing between 4% and 15% of fractures in athletes and military personnel [4, 23, 41].

Fig. 26.11 Mildly displaced distal fibula (Weber C) fracture in a 66-year-old female

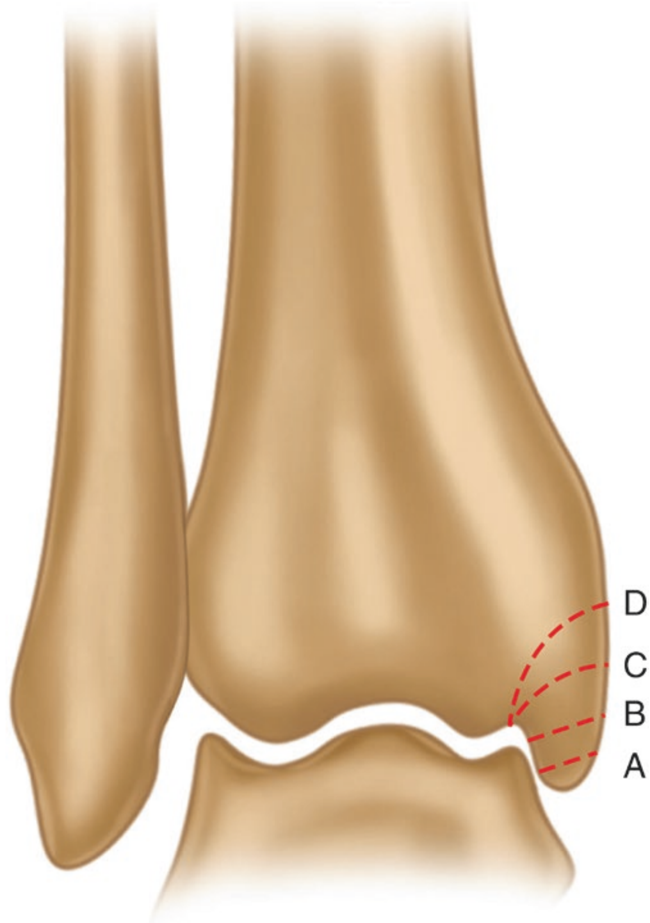


Fig. 26.12 Herscovici classification of medial malleolus fracture

Classification

The most common medial malleolus fracture classification is known as the Herscovici classification (Fig. 26.12) [6, 47, 48]. This classification divides the fractures into four types based on the appearance of AP radiographs. Type A fractures are avulsion-type injuries that occur at the tip. Type B fractures occur between the tip and the plafond, type C fractures are at the level of the plafond, and type D fractures run obliquely from the plafond vertically. This classification has reasonable reproducibility but difficult with classifying type C versus type D injuries [20].

Clinical Presentation

Athletes will typically present similar to severe ankle sprains. Swelling and ecchymosis are usually present. The ability to weight-bear is variable, and range of motion may be limited. Tenderness over the medial malleolus in the area of the fracture is common. Associated tenderness of deltoid ligament may also be present.

Diagnosis

In addition to physical examination, radiographs of the ankle can help both to determine the presence of a fracture and stability of the joint (Figs. 26.13 and 26.14). The Ottawa ankle rules can be used in the acute setting to determine the need for radiographs [11]. The radiographic evaluation of the medial malleolus is similar to the lateral malleolus. Standard views include AP, mortise, and lateral. The AP view is used for fracture classification, and some authors suggest 2 mm of displacement as a cutoff value [6, 47]. The value of views other than lateral and additional imaging such as CT scan or MRI has not been determined in isolated medial malleolus fractures but can be considered on an individual basis [6]. CT scans can be helpful to better visualize displacement and articular step-off since two-dimensional radiographs have inherent limitations. Ultrasound has been shown to be effective in the evaluation of acute ankle injuries. It has good specificity (98%), but less sensitivity (71%), for medial malleolus fractures and when used in conjunction with the

Fig. 26.13 Mildly displaced distal tibia (medial malleolus) fracture in a 62-year-old female

Ottawa ankle rules ultrasound can increase the specificity and aid in ruling out the need for radiographs [34, 35].

Initial Management

Both non-displaced and displaced fractures (>2 mm displacement) can be protected in a boot or splint. Weight-bearing status is unclear and some authors recommend no weight-bearing, but one small study did not find a difference in outcomes with weight-bearing as tolerated [6, 47].

Indications for Orthopedic Referral

Although rare in isolated fractures, open fractures and patients with neurovascular compromised status should be referred immediately for orthopedic evaluation. Athletes with medial malleolus fracture displacement of >2 mm should be referred for further evaluation and possible surgical intervention.

Follow-Up Care

Non-displaced injuries can be followed based on provider preference. A repeat x-ray and evaluation at 7–10 days can be considered to assess stability of the fracture. Radiographs at 6 weeks to asses for fracture healing may be adequate in cases where athletes are able to weight-bear early and have good pain control. For patients with more severe pain, follow-up can be considered at closer intervals to ensure improvement in pain and for re-evaluation of other possible injuries.

Return to Sports

There is limited information on return to play in athletes with isolated medial malleolus fracture. One study of active military that included medial malleolus fractures found 83% could return to active duty but less (64%) were able to return to running [41]. Based on provider preference, athletes can transition to unprotected weight-bearing around 6 weeks. Once athletes are fully weight-bearing and have normal strength and balance, they may progress back into higher-level activities.

Complications

Nonunion, malunion, and post-traumatic arthritis have been reported in both conservative and surgically treated fractures. Injuries to the posterior tibial tendon have been reported in surgically treated fractures [6].

Pediatric Considerations

Ankle injuries are common in pediatric athletes, and the Ottawa ankle rules have been validated in children over the age of 5 [43]. Isolated fractures of the medial malleolus are rare. Salter- Harris type I and type II and minimally displaced (<2 mm) fractures can be treated similar to non-displaced adult fractures. Fractures with more displacement may require reduction and/or surgical intervention [45]. Salter-Harris type III fractures most commonly involve the media malleolus [45]. These injuries are treated similar to adults but non-weight-bearing is recommended for up to 6 weeks, and surgery is indicated for fractures >2 mm of displacement [45].

Posterior Malleolus

Mechanism of Injury in Sports

Exact mechanism of isolated posterior malleolus fractures in general as well as sports has not been well defined. It is believed that small avulsion-type injures are caused by rotational forces while larger fractures are created by the addition of compressive forces [49].

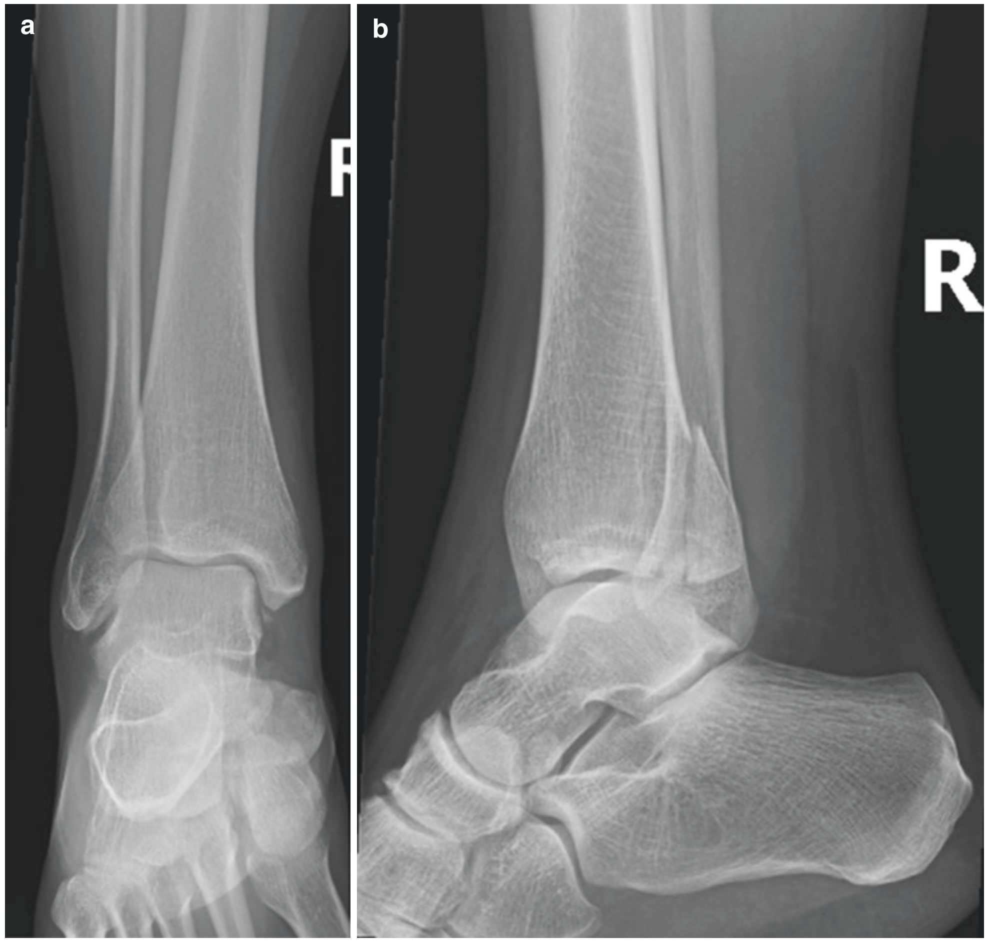


Fig. 26.14 Medial malleolus avulsion (**a**) and posterior malleolus (**b**) fractures in a 32-year-old male as a result of a snowboarding injury

Epidemiology

Isolated fractures of the posterior malleolus are rare (0.5–1% of all ankle fractures) [19]. Because these injuries often occur in conjunction with other injuries, studies often do not separate out isolated injuries (Fig. 26.14). A study on NFL players reported 9 posterior malleolus fractures out of 131 reported fractures but did not specify isolated versus combined fractures [4]. The posterior malleolus was reportedly involved in 26% of ankle fractures in military recruits but again did not specify isolated injuries [41].

Classification

There have been several proposed classification systems for posterior malleolus fractures and nonspecific to isolated injuries. The use of fragment size has fallen out of favor as the relationship between size and treatment is controversial. The Haraguchi classification divides posterior malleolus fracture into three types [50]. Type I is a posterolateral-oblique fracture represents just over two-thirds of fractures. Type II is a medial-extension fracture and type III is a small-shell fragment. A wide variation in fracture patterns are noted on CT imaging, bringing into question the usefulness of this classification. Bartoníček suggested a classification system (Table 26.2) dividing posterior malleolus fractures into five fracture patterns using CT imaging [19, 49].

While some authors recommend treatment based on this classification, it is not clear if either classification system correlates to improved outcomes or should guide treatment options [19, 49].

Table 26.2 Bartoníček classification of posterior malleolus fractures [49]

Type	Description
1	Extra-incisural fragment with an intact fibular notch
2	Posterolateral fragment extending into the fibular notch
3	Posteromedial two-part fragment involving the medial malleolus
4	Large posterolateral triangular fragment
5	Nonclassified, irregular, osteoporotic fragments

Clinical Presentation

Athletes will typically present similar to severe ankle sprain and other ankle fractures. Swelling and ecchymosis are usually present. The ability to weight-bear is variable, and range of motion may be limited. Pain with active and passive dorsiflexion is common. Tenderness over the posterior aspect of the ankle is usually present but may present more as soft tissue pain in the retro-Achilles area rather than focal boney tenderness.

Diagnosis

As with other ankle injuries the Ottawa ankle rules can be used to help determine the need for radiographs based on examination [11]. Standard ankle views include AP, mortise, and lateral. Fractures are generally seen in the lateral view, but radiographic evaluation alone has poor reliability in determining both the presence of a fracture and its morphology [19]. CT scan is the preferred imaging to define the fracture, articular displacement, and classification [19, 49, 51]. It is also important to determine whether there is injury to the syndesmosis since posterior malleolar fractures can accompany these injuries.

Initial Management

Isolated posterior malleolus fracture, both non-displaced and displaced (>2 mm displacement), can be protected in a boot or splint. Weight-bearing status is by provider preference, and some authors recommend non-weight-bearing, but one small study did not find a difference in outcomes with weight-bearing as tolerated [6, 47].

Indications for Orthopedic Referral

Although rare in isolated fractures, open fractures and patients with neurovascular compromised status should be referred immediately for orthopedic evaluation. Athletes with posterior malleolus fracture displacement of >2 mm should be referred for further evaluation and possible surgical intervention [6, 47].

Follow-Up Care

Non-displaced injuries can be followed based on provider preference. A repeat x-ray and evaluation at 7–10 days can be considered to assess stability of the fracture. Radiographs at 6 weeks to asses for fracture healing may be adequate in cases where athletes are able to weight-bear early and have good pain control. For patients with more severe pain, follow-up can be considered at closer intervals to ensure improvement in pain and for re-evaluation of other possible injuries.

Return to Sports

There is limited information on return to play in athletes with isolated posterior malleolus fracture. Therefore, a similar approach may be taken as in athletes with isolated medial malleolus fractures.

Complications

Malunion and post-traumatic arthritis have been reported in both conservative and surgically treated fractures. Injuries to the posterior tibial tendon have been reported in surgically treated fractures [6].

Pediatric Considerations

Ankle injuries are common in pediatric athletes and the Ottawa ankle rules have been validated in children over the age of 5 [43]. There is no separate physis for the posterior malleolus, so typical Salter-Harris injuries do not occur. Preadolescent children may have involvement of the posterior malleolus with other injuries such as triplane fractures. Adolescents can get fracture patterns similar to adults once physeal structures are closed [45].

Bimalleolar and Trimalleolar

Mechanism of Injury in Sports

Both bimalleolar and trimalleolar fractures are believed to occur with a combination of supination and external rotation in most cases. However, the exact mechanisms of these fractures are not well defined in sports. These fractures are thought to be due to higher-energy injuries than isolated malleolus fractures [52, 53].

Epidemiology

Bimalleolar and trimalleolar fractures are less common than isolated malleolar fractures but estimates vary. Bimalleolar fractures account for approximately one-fourth of ankle fractures, while trimalleolar fractures account for about 7% to 14% [19, 53, 54]. This is consistent with data on military recruits that found bimalleolar and trimalleolar fractures to be 19% and 3% of injuries, respectively [41]. However, one large series found the opposite with bimalleolar representing only for 4% of fractures versus 11% for trimalleolar fractures [21]. Information on sports-related bimalleolar and trimalleolar fractures is limited.

Classification

Bimalleolar and trimalleolar fractures fall under two main classification systems, Lauge-Hansen and the AO. The AO systems divides these fractures based on relationship to the syndesmosis, below (infra), at the level (trans), and above (supra) [26]. Lauge-Hansen uses mechanism of injury to define the fracture patterns. The patterns are described as supination-external rotation, supination-adduction, pronation-external rotation, and pronation-abduction. Based on the energy applied, this classification theorizes that struc-

tures will be damaged from lateral to medial, supination injuries or medial to lateral, pronation injuries. The AO system has better correlation to observed injury mechanism than Lauge-Hansen [26]. Because both of these systems can be complex, simple description of the fracture pattern is often used (Figs. 26.15, 26.16, and 26.17) [55].

Clinical Presentation

Because these fractures generally involve higher energies, most athletes will present with pain, swelling, ecchymosis, and inability to bear weight. Tenderness will be diffused around the ankle, and both passive and active ankle range of motion are usually limited. Deformity may be present with displaced fractures (Fig. 26.16).

Diagnosis

Standard radiographs of the ankle should be obtained; however, almost all fractures will require CT scan to determine the extent of the injury and fracture morphology [56, 57]. The value of MRI has not been determined as although it may delineate other soft tissue and ligament injuries. As the MRI may or may not change surgical approach and treatment, its use is not clear [52, 58].

Initial Management

Careful examination of the skin and neurovascular structures is important. If there are no concerns for compartment syndrome or skin breakdown, then a short leg splint with bulky dressing is usually applied. Based on the fracture and individual, patient should include a posterior splint with a U-splint to stabilize the fracture. It is critical in trimalleolar fractures that reduction be confirmed the repeat radiographs after the fracture has been reduced and a splint has been placed. Patients should remain non-weight-bearing and be instructed to monitor for signs and symptoms of compartment syndrome.

Indications for Orthopedic Referral

Almost all bimalleolar and trimalleolar fractures will require surgical intervention and should be referred for orthopedic evaluation. Open fractures and fractures with concern for skin compromise, neurovascular injury, or compartment syndrome should be referred immediately for evaluation. The timing of surgery varies based on provider preference, individual fracture, and swelling, so early referral, within a few days, for other fractures should be considered.

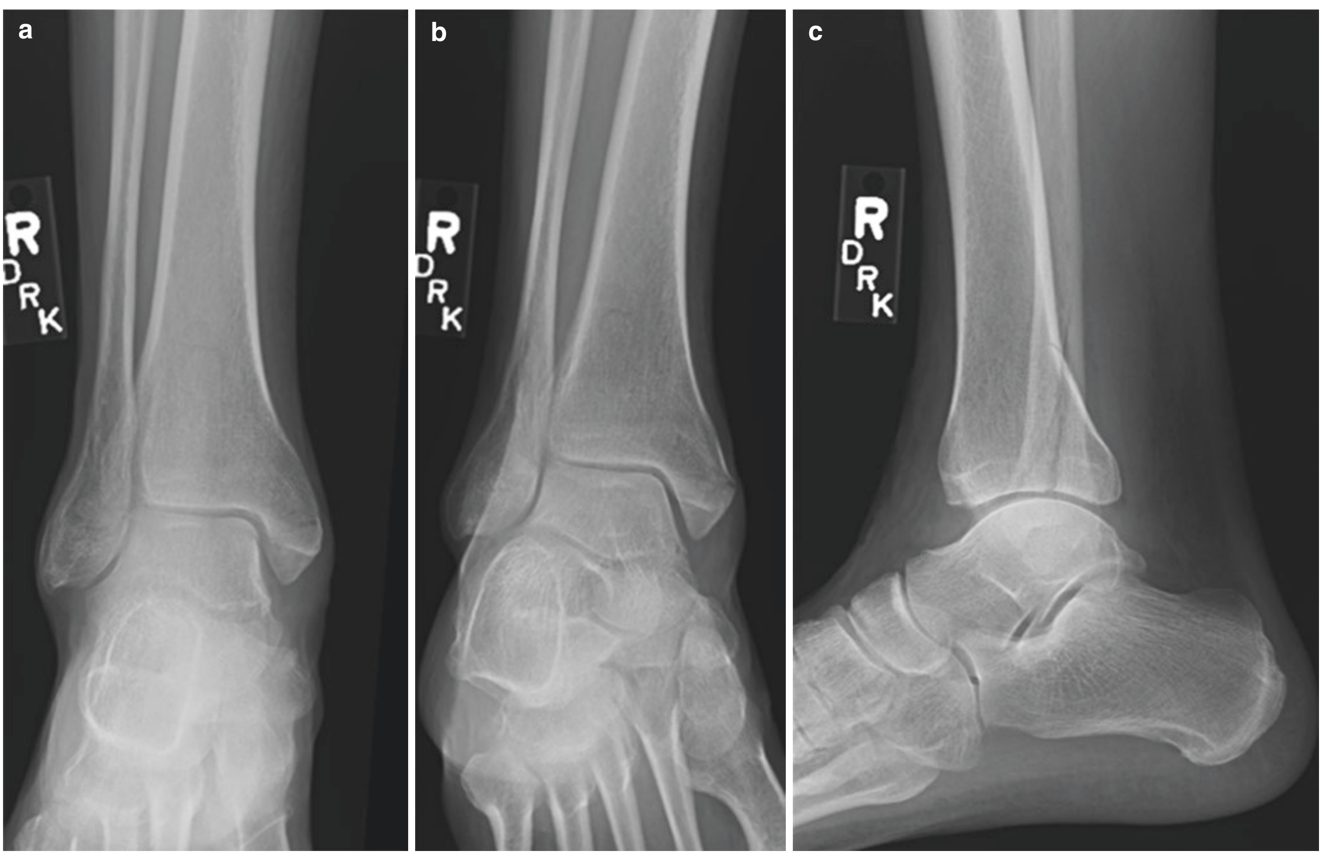


Fig. 26.15 Non-displaced distal tibia and fibula (trimalleolar) fracture in a 66-year-old female (**a**–**c**)

Fig. 26.16 A displaced and comminuted trimalleolar fracture with posterior talus dislocation (**a**–**d**) in a 27-year-old female as a result of a snow tubing injury. Postreduction radiograph reveals a better anatomic alignment (**e**)

Fig. 26.17 A displaced and comminuted trimalleolar fracture with lateral talus dislocation in a 20-year-old female as a result of a mountain biking injury (**a**–**c**). CT scan images demonstrate the extent of the injury (**d**–**f**)

Follow-Up Care

Based on the timing of orthopedic follow-up, consideration for an early recheck (3–5 days) to evaluate the skin and for swelling can be considered. Non-displaced bimalleolar fractures may be considered for conservative treatment. Although no specific guidelines exist, follow-up frequency, weekly, with radiographs should be considered to ensure fracture stability. A short leg non-weight-bearing cast can be applied once swelling resolves. Non-weight-bearing status should be considered for 6 weeks postoperatively with cast changes as needed. No data exists on early mobilization of these injuries, but range of motion and strength with progression to

weight-bearing in a boot can take place in the 6-week range. Progression out of the boot will depend on the orthopedic provider and individual patient.

Return to Sports

Unfortunately, these fractures can create significant morbidity and make it difficult to return to sporting activity. In one small series (n = 12), nine patients were able to return to sporting activity after surgery for trimalleolar fractures with two having moderate to severe difficulty [54]. In a series of 47 patients treated for both bimalleolar and trimalleolar fractures, only 27% reported ability to return to their preinjury sporting level [53]. Similar to other ankle fractures, there are no specific guidelines on the timing of return to play, and the decision will be based on the individual athlete and the return to functional movement and strength.

Complications

Complications are common after these injuries. Stiffness, pain, and swelling are commonly reported in the first 1–2 years after the injury [53, 54]. Post-traumatic osteoarthritis is the most common long-term complication [54].

Pediatric Considerations

Similar to posterior malleolus injuries, these injury patterns are rare in children as distal tibia injury patterns are more common [45].

Distal Intra-Articular Tibia (Pilon)

Distal intra-articular (non-malleolar) tibia fractures are typically called *pilon* (plafond) fractures [59].

Mechanism of Injury in Sports

Low-energy fractures occur with torsional forces and minimum impact on soft tissue and ankle joint [59–61]. Low-energy pilon fractures are intra-articular distal tibia diaphysis fractures. High-energy fractures occur as a result of significant axial forces with significant risk for soft tissue and ankle joint injuries [59–61]. The high-energy fractures (some call them “true” pilon fractures) usually happen in traffic accidents or fall from a height [59–61].

Epidemiology

Pilon factures are rare in general and specifically in sports. Pilon fracture counts for about 5% of all tibia fractures [59–61]. The low-energy fractures can happen in sports such as skiing as a result of twisting. High-energy pilon fractures may happen as a result of landing in sports such as ski jumping and parkour.

Classification

AO/OTA is the most commonly used classification system for tibia pilon fractures (Fig. 26.18) [62]. Type A are extra-articular, type B are partial articular, and type C are complete articular fractures [62]. Type C fractures are usually a result of high-energy compression mechanisms. There have also been multiple CT-based pilon fracture classifications [61, 63–65]. Leonetti and Tigani introduced a new classification based on CT images in 2017 [64]. In their system, they categorized pilon fractures based on the number of number of intra-articular fracture fragments and displacement (Fig. 26.19) [64]. Reproducibility and prognostic values of these systems are yet to be shown in large studies. The degree of associated soft tissue injury including open fractures should be taken in consideration for classifications and eventually management.

Clinical Presentation

Due to the possible high-energy mechanisms, soft tissue injuries and open fractures are relatively common [59–61, 63]. Athlete usually presents with significant pain, inability to bear weight, edema, ecchymosis, and occasionally deformed ankle [59–61, 63].

Diagnosis

Standard ankle plain radiography views should be obtained as soon as possible (Figs. 26.20, 26.21, 26.22, and 26.23). Due to the complexity of most of pilon fractures, CT scan is critical to evaluate the extent of the fracture and surgical planning. MRI may be indicated to evaluate soft tissue injuries, such as Morel-Lavallée lesion, but is rarely indicated. Ultrasound may be helpful to evaluate soft tissue injuries and to detect distal pulses if difficult to identify.

Initial Management

ATLS protocol should be followed as these fractures are often associated with multisystem injuries [59–61, 63]. Early diagnosis and complete neurovascular evaluation are the key for the initial management. Athletes should be stabilized and immobilize as soon as possible [60, 61, 63].

Indications for Orthopedic Referral

Urgent referral and transportation to a facility with a higher level of care are indicated for athletes with open pilon fractures and those with neurovascular and tissue compromised

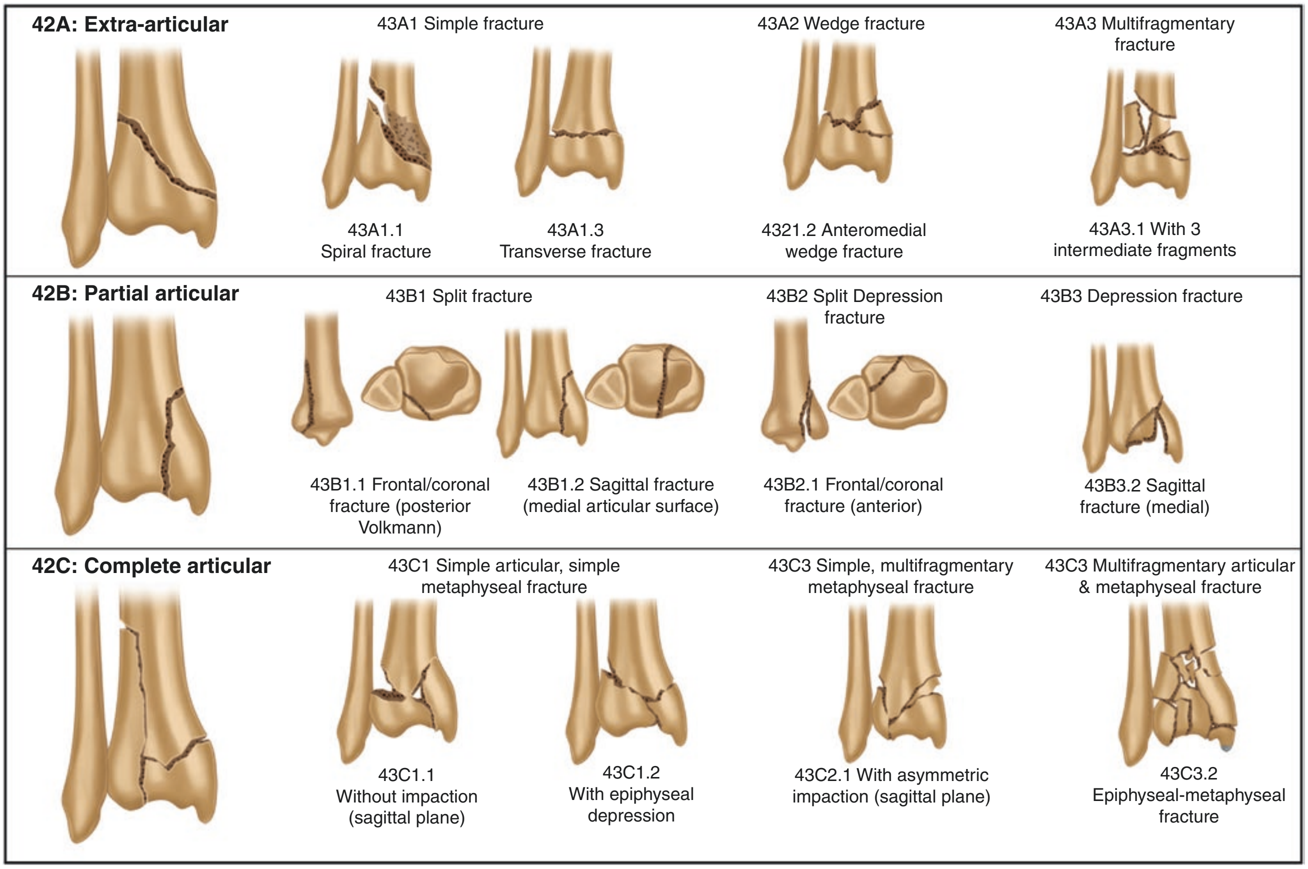


Fig. 26.18 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for distal tibia fractures

[59–61, 63]. Otherwise, athletes with stable pilon fractures should be referred to orthopedic surgery within a couple of days.

Follow-Up Care

Athletes should be followed on a regular basis (e.g., 1 to 2 weeks intervals) postoperatively. Duration of non-weight-bearing and partial weight-bearing depends on the severity of the injury and types of treatment.

Return to Sports

As pilon fractures can be devastating injuries to joint cartilage, most athletes are not able to return to sports at the preinjury level. Typically, it would take many months for the athletes to start the return to practice and game protocol. Athlete should be completely asymptomatic with a good range of motion and strength to start sport-specific activities.

Complications

Immediate complications of pilon fractures include open fractures, associated multiorgan injuries, soft tissue (e.g., contusion and compartment syndrome) and skin (e.g., blister and laceration) injuries, infection, and compartment syndrome [59–61, 63]. Long-term complications include malunion, nonunion, and osteoarthritis [59–61, 63].

Pediatric Considerations

Pediatric intra-articular distal tibia fractures are rare and count for about 20% of all pediatric ankle fractures [45, 66]. Distal tibia physis closes in an 18-month period (at about 14 years of age for girls and 16 for boys). The closure starts in the central part of the physis and then advances to anteromedial and posterolateral parts [45, 66]. The distal tibia is the second most common pediatric epiphysis fracture behind only to distal radius epiphysis fracture [45, 66].

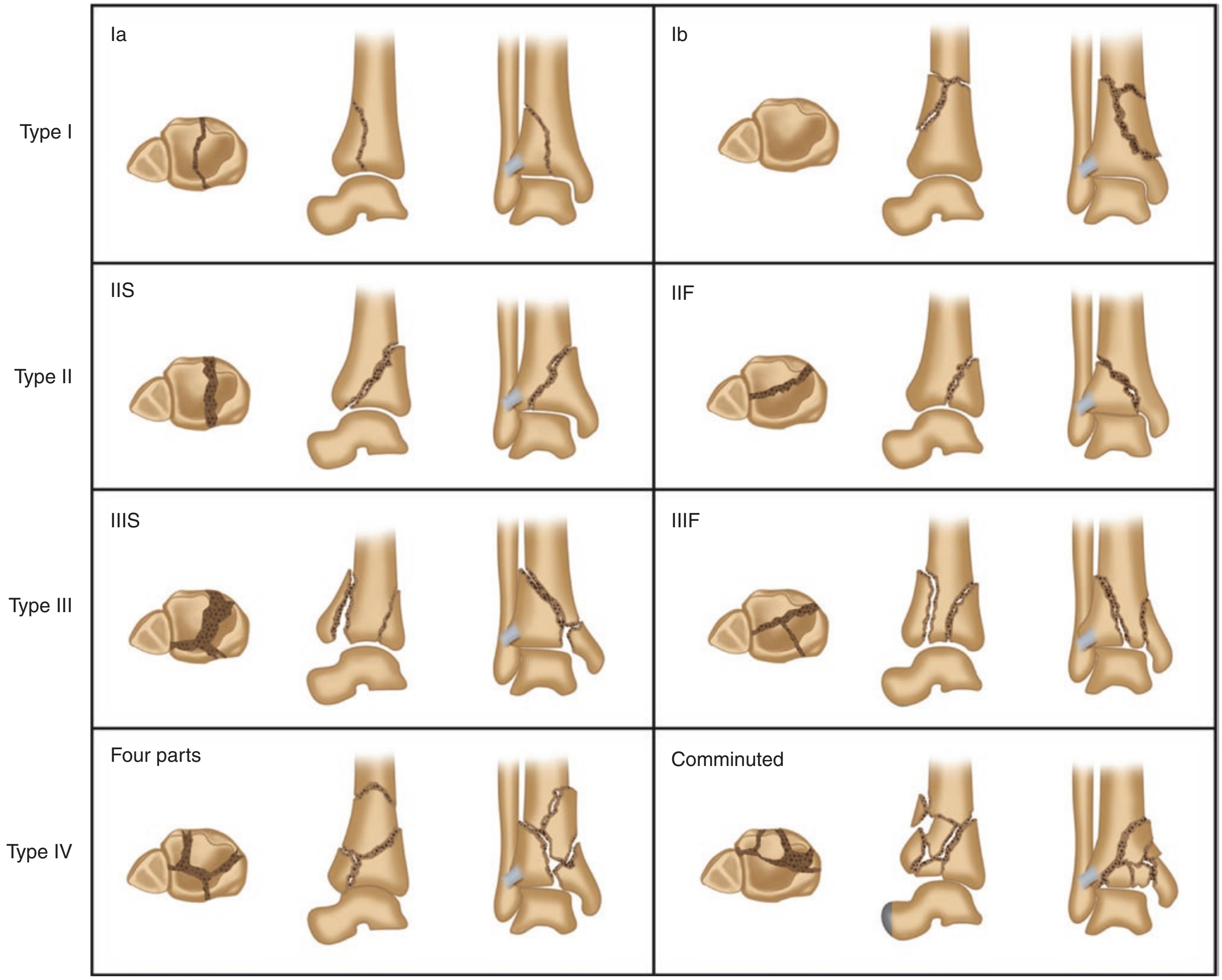


Fig. 26.19 Leonetti and Tigani classification system. Type I fractures are divided into Ia (non-displaced) and Ib (extra-articular) fractures. Type II (displaced two part) fractures are divided into IIs (fracture line is in sagittal plane) and IIf (fracture line is in frontal plane). Type III (displaced three part) fractures are divided into IIs (the main fracture line is in sagittal plane) and IIIf (the main fracture line is in frontal plane). Type IV fractures are divided into four parts and comminuted fractures

Tillaux fracture (Fig. 26.24) is an intra-articular Salter-Harris III fracture with avulsion of the anterolateral epiphysis of the distal tibia [45, 67, 68]. It occurs at the time the medial physis is closed, but the lateral physis is still open [45, 67, 68]. The fracture fragment becomes smaller and more lateral with progression of the closure of the physis. The mechanism of the injury is usually an external rotation with eversion in a plantarly flexed foot [45, 67, 68].

Triplane fracture is a subgroup of the Salter-Harris IV fracture which occurs before the closure of the distal tibia physis [45, 66, 67]. So patients are usually a little younger than the ones with Tillaux fractures. The fracture line is usually through three planes: sagittal (epiphysis), coronal (metaphysis), and transverse (physis) [45, 66, 67]. The injury usually occurs when a supinated foot forces to externally rotate [45, 67, 68].

Diagnosis and nonoperative management of the Tillaux and triplane fractures are similar.

Patients usually present with an “ankle sprain” and inability to bear weight [45, 66–68]. Edema and ecchymosis are

Fig. 26.20 Non-displaced, intra-articular distal tibia fracture in a 27-year-old male as a result of a snowboard injury **a**–**c**). CT scan images demonstrate the extent of the fracture (**d**–**g**). Postoperative image shows near-anatomic alignment (**h**)

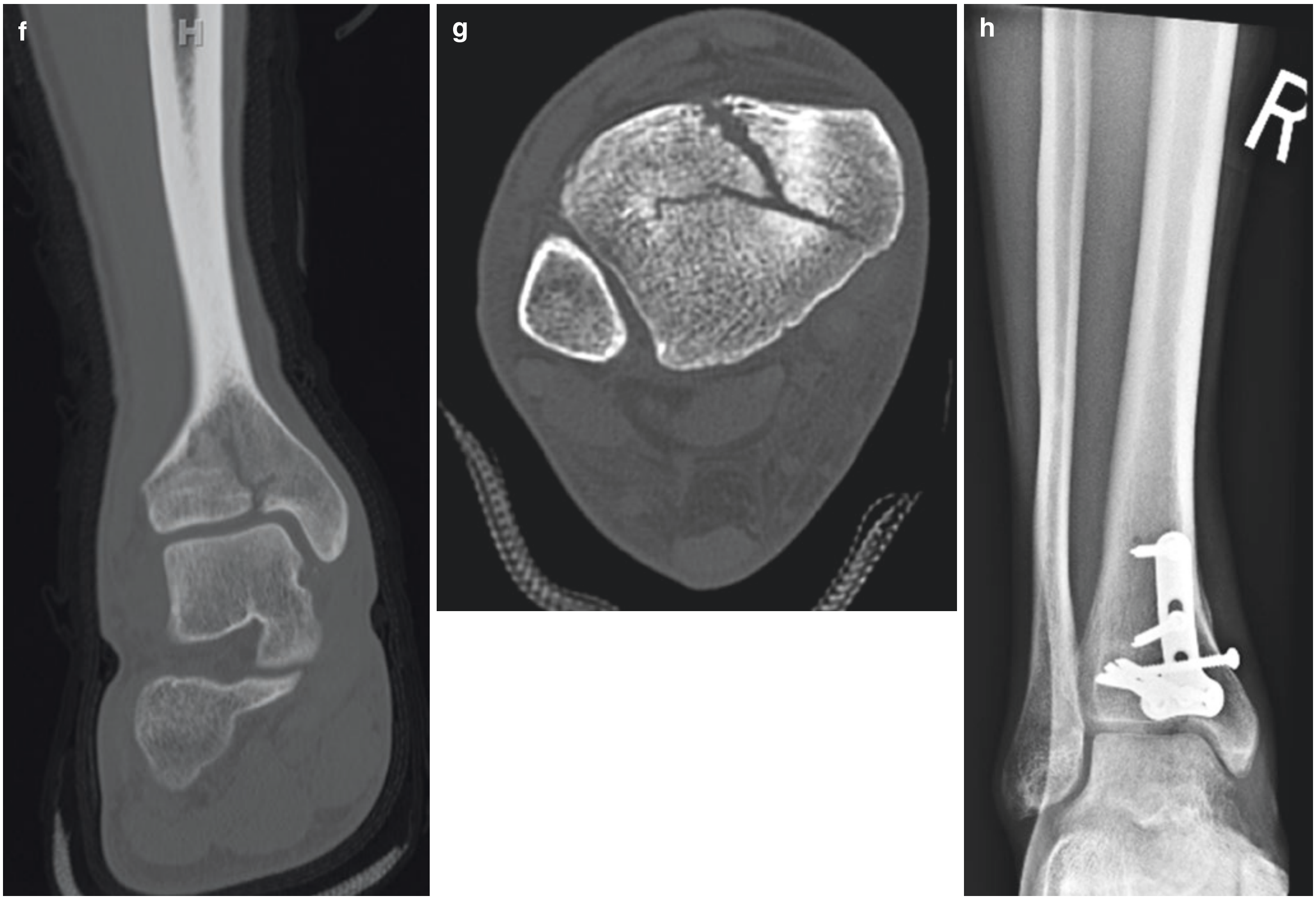


Fig. 26.20 (continued)

usually present. Fracture lines are usually visible on standard plain radiography ankle views. Usually, a CT scan (Fig. 26.24) is indicated to evaluate the extent of the fracture and preoperative planning [45, 66–68]. Typically, fractures with <1 mm displacement can be managed nonoperatively.

Dislocations

True ankle joint (tibiotalar or talocrural) dislocation is uncommon in sports. Direction of the dislocation is based on the displacement of the talus in relation to the tibia. Posterior dislocations are the most common type (Fig. 26.16) [69]. The majority of ankle dislocations are accompanied by distal tibia or fibula fracture (Figs. 26.16, 26.17, and 26.25). However, rare cases of pure ankle dislocations have been reported in the literature [70–72]. Many ankle dislocations present as open dislocations [69]. The mechanism of injury is usually high-speed sports or landing when the foot is dorsally flexed. Therefore, ankle dislocations are more common in sports such as volleyball, basketball, motor sports, and winter sports. Early detection and complete neurovascular evaluation are the key for the initial management. It is highly recommended to obtain plain radiography before reduction. Reduction should be performed, ideally with good anesthetics, as soon as possible. All athletes with ankle dislocations should be transferred to ED after appropriate immobilization. Most athletes would need urgent orthopedic evaluation and most likely surgical intervention. Open injuries should be urgently irrigated and debrided to reduce risk of subsequent infection or foreign bodies. Dirty injuries may require serial debridement. Early, controlled mobilization of these injuries can be helpful to prevent stiffness, which can be an issue following these injuries. Return to sport is largely predicated on injury severity, sport, and position.

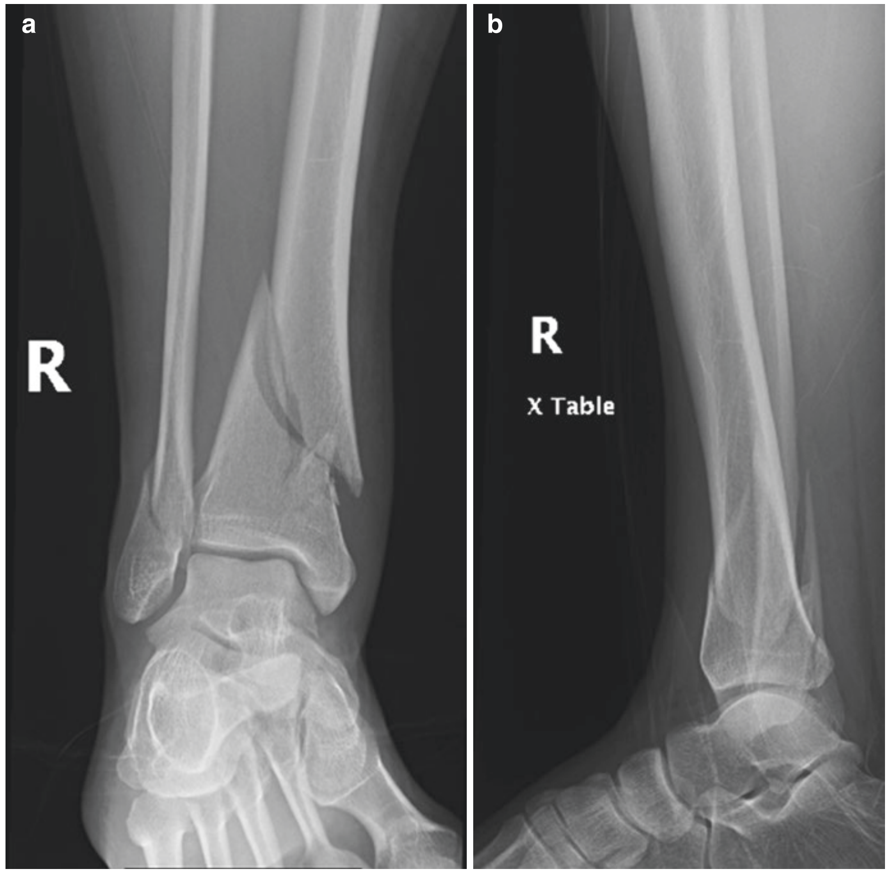


Fig. 26.21 (**a**, **b**) Mildly displaced intra-articular distal tibia and fibula (Weber B) fracture in a 41-year-old female as a result of a ski injury

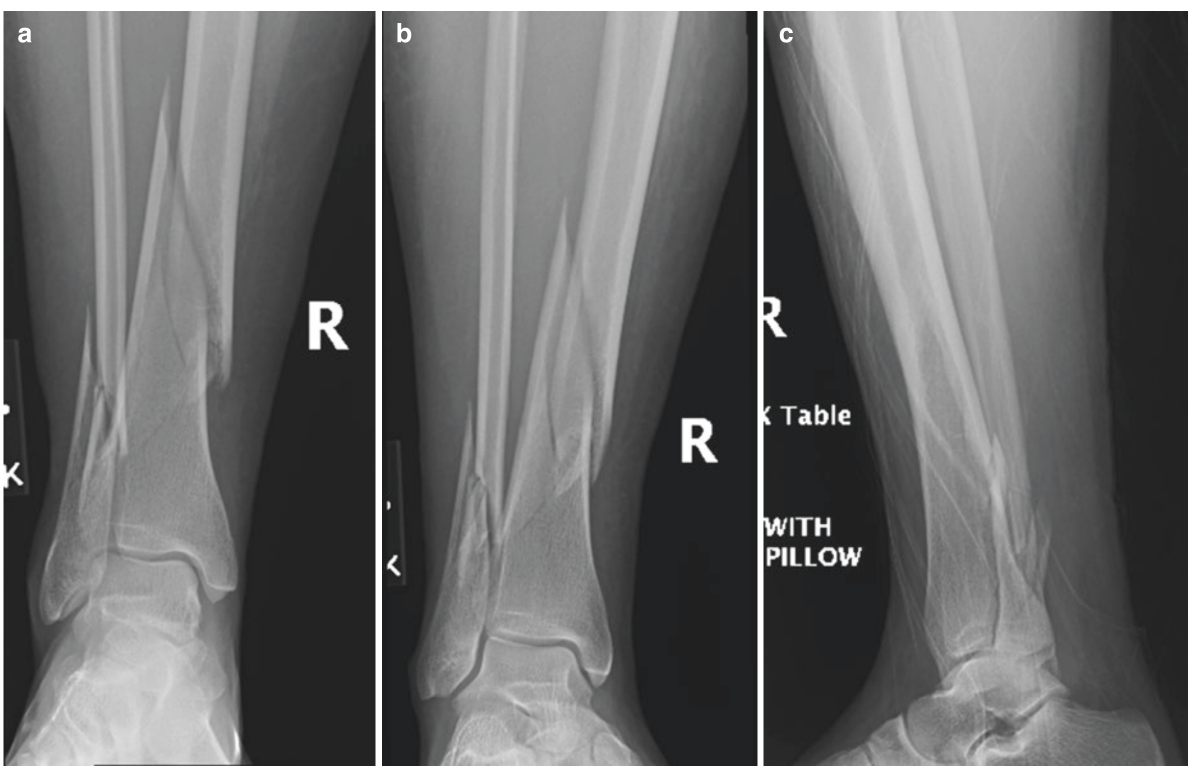


Fig. 26.22 Displaced distal tibia (intra-articular) and fibula fracture in a 60-year-old female as a result of a ski injury (**a**–**c**)

Fig. 26.23 Plafond (pilon) fracture of distal tibia and mildly displaced distal fibula (Weber B) fractures in a 47-year-old female as a result of a ski injury (**a**, **c**). Stress view shows mild widening of the talofibular space (**b**). CT scan images demonstrate the extent of the fracture (**d**–**g**). Postoperative image shows near-anatomic alignment (**h**, **i**)

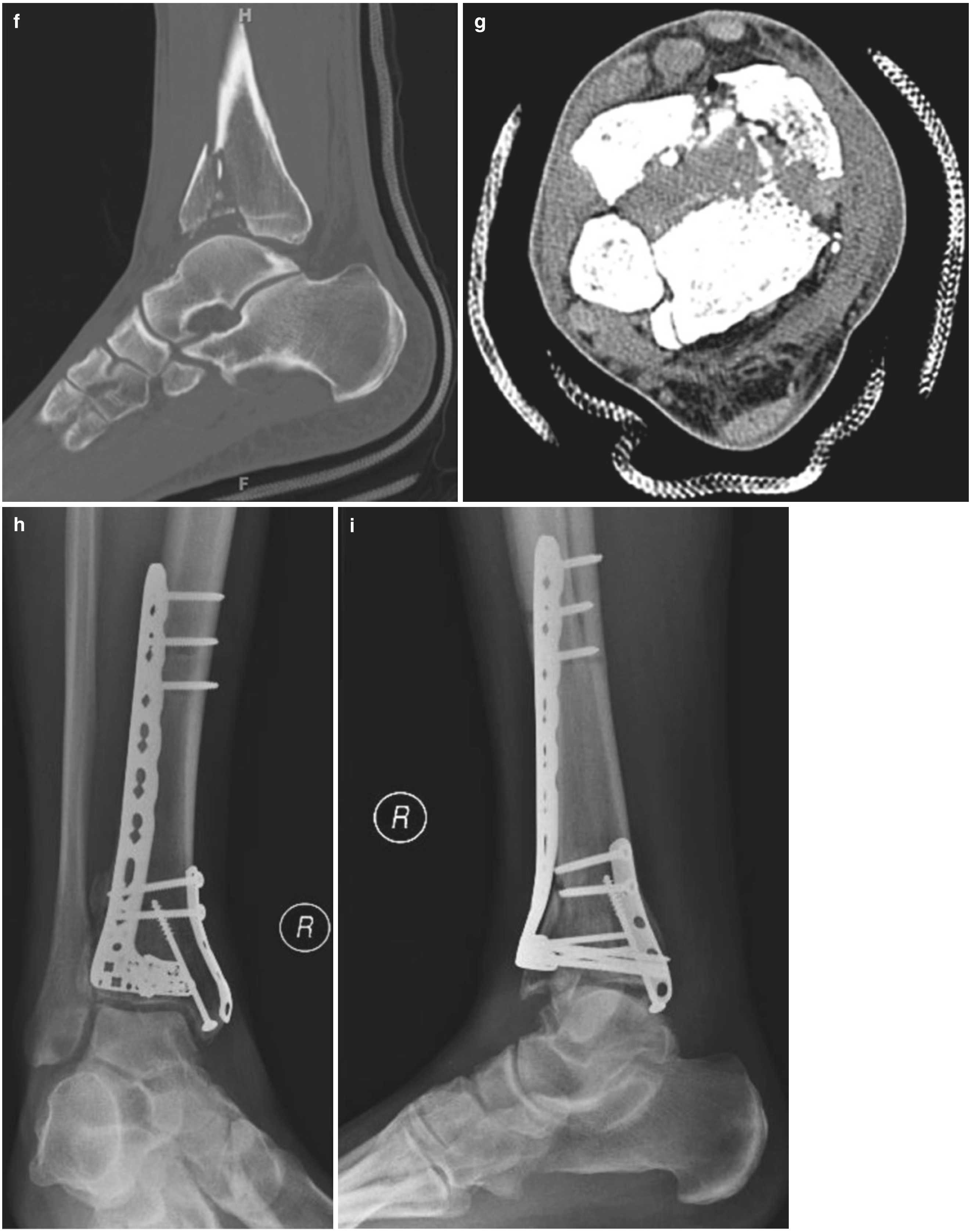


Fig. 26.23 (continued)

Fig. 26.24 A mildly displaces intra-articular Salter-Harris III fracture of the lateral tibial epiphysis (Tillaux fracture) in a 10-year-old girl as a result of twisting her foot while stepping in a hole (**a**, **b**). CT scan images demonstrate the extent of the fracture line (**c**, **d**)

Fig. 26.25 A displaced and comminuted trimalleolar fracture with lateral dislocation of the talus with moderate valgus angulation (**a**–**d**) in a 35-year-old female as a result of twisting her ankle while falling on ice. Postreduction radiographs reveal a better anatomic alignment (**e**, **f**). Postoperative radiographs show near-anatomic alignment (**g**, **h**)

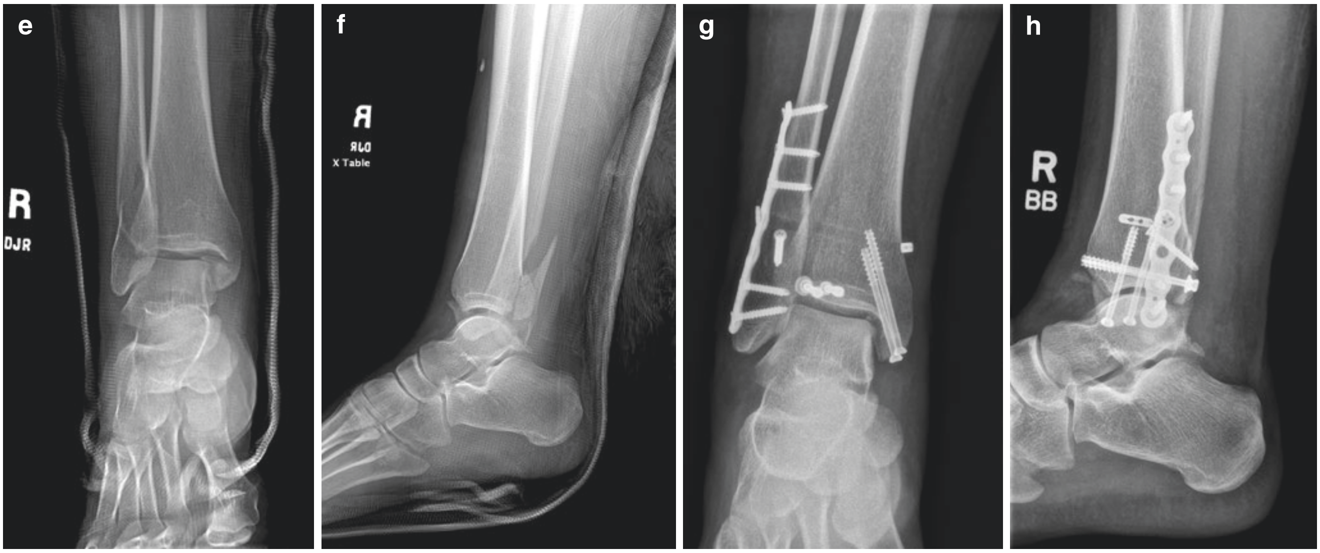


Fig. 26.25 (continued)

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Tarsus

27

Jennifer D. Stromberg

Key Points

- Acute tarsal fractures are rare.
- Calcaneus fractures are the most common tarsal fractures.
- Talus is the second most common tarsal bone presenting with fracture which typically involves the neck.
- Diagnosis often requires CT scan.
- Operative intervention is frequently necessary.

Introduction

Acute fractures of the tarsal (tarsus) bones are uncommon and rarely occur in isolation, with the exception of the calcaneus and talus [1]. Less than 10% of midfoot fractures are due to blunt trauma and most occur from motor vehicle accidents [2]. Incidence of these fractures in the sports setting is not known, but because these fractures often require high energy and occur with falls from heights, they have been reported in sports such as snowboarding [3]. The calcaneus is the largest tarsal bone and accounts for approximately 60% of tarsal fractures [4–6]. It is integral to weight-bearing and gait mechanics and long-term disability with these injuries is high [4–6]. Fractures of the talus are uncommon and usually occur at the neck, and because of blood supply, displaced fractures may result in osteonecrosis [7]. Acute navicular fractures are rare and in most cases fractures are avulsions involving the ligamentous attachments. Body fractures are uncommon and often require surgical intervention [8]. Cuboid fractures are also commonly ligamentous avulsion injuries, but crush fracture of the main body of the cuboid can occur with direct trauma or compression of the lateral foot column [9]. Cuneiform fractures are almost always associated with fracture or fracture-dislocation of the tarsals or tarsometatarsal joint but rarely may occur in isolation [10].

Anatomy

The tarsal bones have multiple articulations (Chap. 28, Fig. 28.1, and Chap. 26, Fig. 26. 2). The calcaneus has three articulations with the talus (anterior, middle, and posterior facets), one with the cuboid (Fig. 27.1). Calcaneus serves as the attachment site for the Achilles tendon and origin for the superomedial spring ligament and the tibiocalcaneal component of deltoid ligament (Chap. 26; Fig. 27.2). The posterior facet articulation with the talus acts as the primary load-bearing surface of the subtalar joint [4]. The sustentaculum tali also supports the talar neck (Fig. 27.1). The talus is the second largest tarsal bone. It has no muscle or tendinous attachments and greater than 50% of the surface is articular cartilage. Blood supply comes from three main arteries (posterior tibial, anterior tibial, and perforating peroneal), with the main supply coming from a branch of the posterior tibial artery which runs through the tarsal canal [11]. This blood supply makes displaced fractures of the neck more susceptible to osteonecrosis. The body of the talus articulates superiorly with the distal tibia, medially and laterally with the medial and lateral malleoli, and inferiorly with the calcaneus [12]. The head of the talus articulates with the navicular distally and the calcaneus plantarly, while the lateral process articulates with the distal fibular and calcaneus, and the posterior process articulates with the distal tibia and calcaneus [12]. The posterior process is separated by a groove containing the flexor hallucis longus [12]. The navicular acts as the keystone of the arch with articulations with the talar head proximally, cuneiforms distally, and cuboid laterally [8, 13]. Because of these articulations, the navicular has a large surface area of articular cartilage. The cuboid bone has six

J. D. Stromberg (✉)
Carolina Family Practice and Sports Medicine, Duke Community and Family Medicine, Cary, NC, USA
e-mail: Jennifer.stromberg@duke.edu

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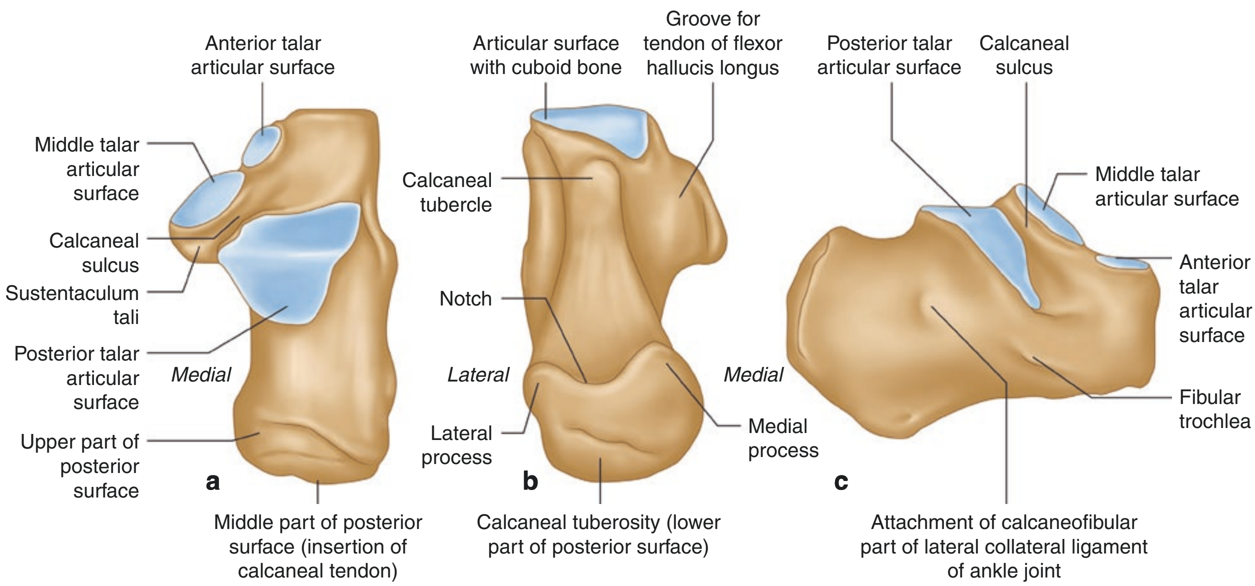

Fig. 27.1 Calcaneus anatomy (**a**) Superior view, (**b**) Inferior view and (**c**) Lateral view

Fig. 27.2 Insertional Achilles tendon rupture with avulsion fracture of the calcaneus (arrow) in a 43-year-old male as a result of a snowboarding injury

surfaces and articulates proximally with the calcaneus, medially with the lateral cuneiform and sometimes navicular, and distally with the fourth and fifth metatarsals [9]. The articulation with the fourth and fifth metatarsals is a primary contributor to dorsiflexion, plantarflexion, pronation, and supination [9]. The peroneal longus tendon also runs within the peroneal sulcus on the plantar surface [9, 14]. The medial cuneiform articulates with the navicular, first and second metatarsals, and the middle cuneiform. Both tibialis anterior and peroneus longus have attachments on the medial cuneiform. The middle cuneiform articulates with the navicular, second metatarsal, and medial and lateral cuneiforms. The lateral cuneiform articulates with the cuboid, navicular, middle cuneiform, and the third metatarsal with tendon attachments of tibialis posterior and flexor hallucis brevis. For all the cuneiforms, the plantar ligamentous attachments are thought to be weaker than the dorsal ones. This combined with the bony anatomy makes dorsal fracture-dislocations more common [15].

Soft Tissue Injuries

Ankle sprains are some of the most common injuries seen in sports [16]. Because the mechanism of injury is similar, tarsal ligament injuries may be confused or occur with lateral sprains. Several studies have found high association between lateral ankle injuries and mid-tarsal joint sprains [17, 18]. The mid-tarsal (Chopart) joint includes the calcaneocuboid, calcaneonavicular, and dorsal talonavicular ligaments. MRI studies following inversion sprains have reported associated injuries of these ligaments in about 20% of cases [17, 18]. Athletes will generally present with pain and swelling in the midfoot region with varying degrees of ability to bear weight.

These injuries generally respond to similar conservative treatment to ankle sprains but can be a cause of persistent pain [16].

Plantar fascia pain is most commonly a middle-age degenerative overuse condition; however acute plantar fascia injuries (including the plantar fascia rupture) have been reported in athletes [19]. These injuries may occur with foot strike in running and are also associated with ankle sprain mechanism. Runners are most commonly affected, but injuries have been reported in other sports such as basketball and tennis [19]. Pain is usually acute in onset and most athletes will have difficulty with weight-bearing. Ecchymosis may occur with plantar fascia rupture. Treatment is almost always conservative with initial immobilization and progression to activity as pain allows [19].

Other soft tissue midfoot injuries such as contusions may occur from direct trauma and can be confused with deeper injuries such as bone contusions [10, 20].

Fractures

Calcaneus

Mechanism of Injury in Sports

Calcaneus fractures are typically high-energy injuries associated with an axial load which drives the lateral talar process into the calcaneus [5, 6]. They are typically seen in motor vehicle accidents or falls from height, or in the case of sports, high-energy landings that involve jumping from heights [4, 21]. Lower-energy injuries may also occur with sudden forced dorsiflexion and eccentric contraction of Achilles leading to an avulsion fracture (Fig. 27.2), as the Achilles tendon is subjected to high loads (up to ten times body weight with running or jumping) [21, 22]. These injuries typically result from a lower-energy fall or sudden push-off from standing [5, 21, 22]. Avulsion fractures can also occur with direct trauma, particularly if there is intrinsic tightness of the gastrocnemius. Extra-articular fractures typically result from twisting forces in the hindfoot [21, 22].

Epidemiology

There is a paucity of information on the incidence of tarsal fractures in sports. Epidemiological reviews have rarely reported sports or recreational activities as injury mechanisms for these fractures [23–25]. Calcaneus fractures are the most common tarsal fracture, accounting for only 2% of all fractures, but 60% of tarsal bone fractures [5, 6]. Displaced intra-articular calcaneus fractures (DIACFs) account for 60–75% [4, 21, 26]. Tuberosity avulsion fractures account for only 1–3% of calcaneus fractures and peak in women in their 70s due to decreases in bone strength [21]. Open fractures carry a 50% chance of concomitant injury [5].

Classification

Calcaneus fractures are typically described as either intra-articular or extra-articular (Figs. 27.3, 27.4, 27.5, 27.6, and 27.7). There are several different classification systems. In the Essex-Lopresti system, fractures are classified by depression or tongue type based on lateral foot radiographs (Fig. 27.8). Both types share a primary fracture line but differ in secondary fracture lines [5, 6]. In a tongue-type fracture, the posterior fragment is displaced posterosuperiorly by forces from Achilles tendon – this can lead to pressure necrosis of the skin and convert to open fracture [5]. The Sanders system uses coronal CT and is based on displacement and location of articular fragments of the posterior facet (Fig. 27.9). Type I has <2 mm of displacement, Type II are displaced two-part fractures, Type III are displaced three-part fractures, and Type 4 are comminuted intra-articular four-part fractures. Subtypes for Types II and III describe the fracture pattern anatomically: A, lateral; B, middle facet; and C, medial through sustentaculum [6]. Overall neither system is predictive of outcomes although Sanders Types III and IV tend to have worse outcomes [6].

In a tuberosity avulsion fracture, there are similar issues as with respect to risk for soft tissue compromise, but these are more often seen in the elderly [17]. Tuberosity avulsion fractures are further broken down into three types. In a Type I or sleeve fracture, a small shell of cortical bone avulses from the tuberosity. In a Type II or beak fracture, an oblique fracture line runs posteriorly from the superior aspect of the posterior facet. In a Type III or infrabursal fracture, the avulsion occurs from the middle of the tuberosity [21, 22].

Clinical Presentation

Clinical presentation of tarsal fractures varies based on the injured bone and type of fracture. Athletes will often have difficulty with weight-bearing but some injuries may masquerade as sprains. Examination of joint stability and range of motion can help to elucidate more significant injuries. Plantar ecchymosis is common in calcaneus fractures (Fig. 27.3). In calcaneus fractures hindfoot skin may be swollen without discernable skin creases, tented or blanched. These can be signs of potential compromised blood supply to overlying skin [5].

Diagnosis

While presentation and physical examination can give clues to injured structures, most tarsal bone injuries require radiographs and advanced imaging. The Ottawa foot and ankle rules can be used to rule out clinically significant fractures; however, these rules only include pain over the navicular and do not include other tarsal bones and may miss other injuries that may affect treatment in athletes [27, 28]. For calcaneus injuries lateral radiographs are key for fracture classification [5]. Calcaneus (axial) view (Figs. 27.3, 27.4, and 27.5) can

Fig. 27.3 An extra-articular calcaneus fracture (**a**, **b**) in a 60-year-old male as a result of jumping in a shallow pool. Plain radiography (**c**, **d**) 2 weeks later demonstrates the fracture line (arrows)

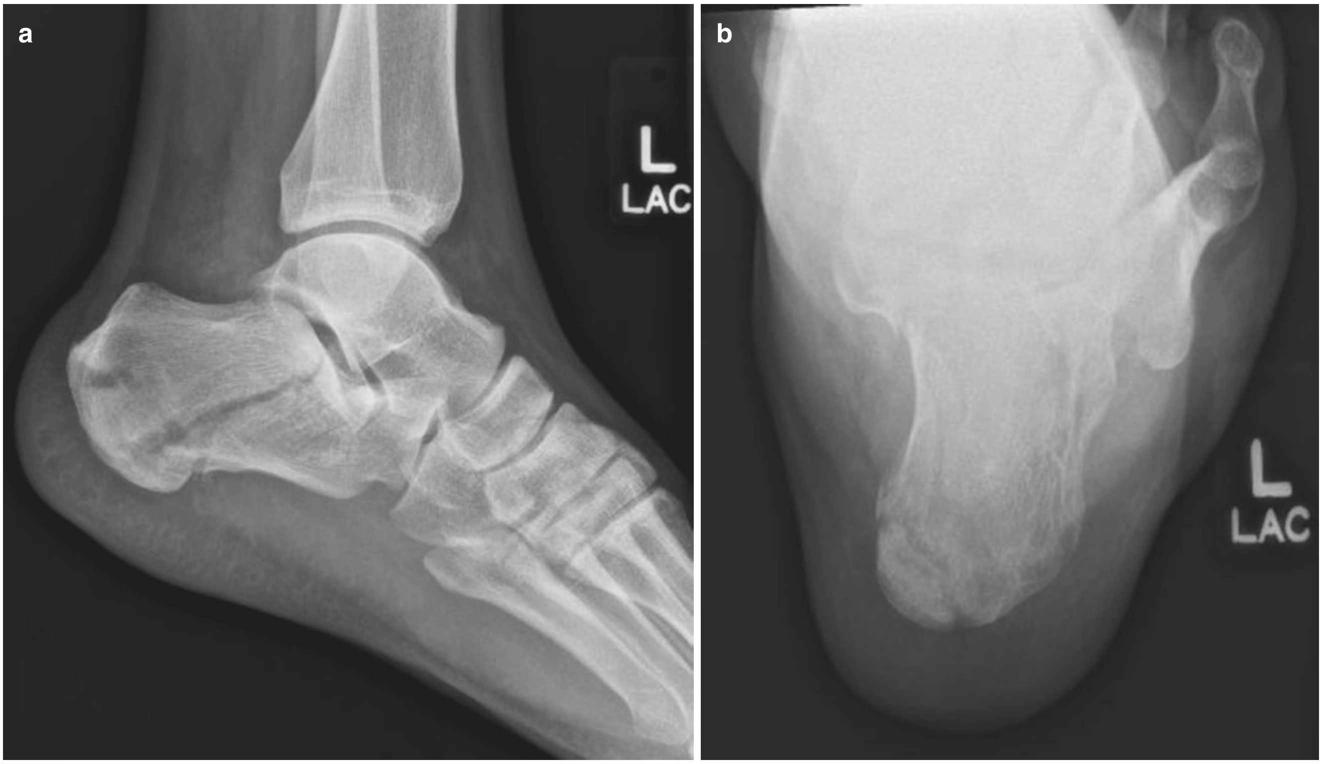

Fig. 27.4 An intra-articular and mildly displaced comminuted calcaneus fracture in a 25-year-old male as a result of a motor vehicle accident (**a**, **b**)

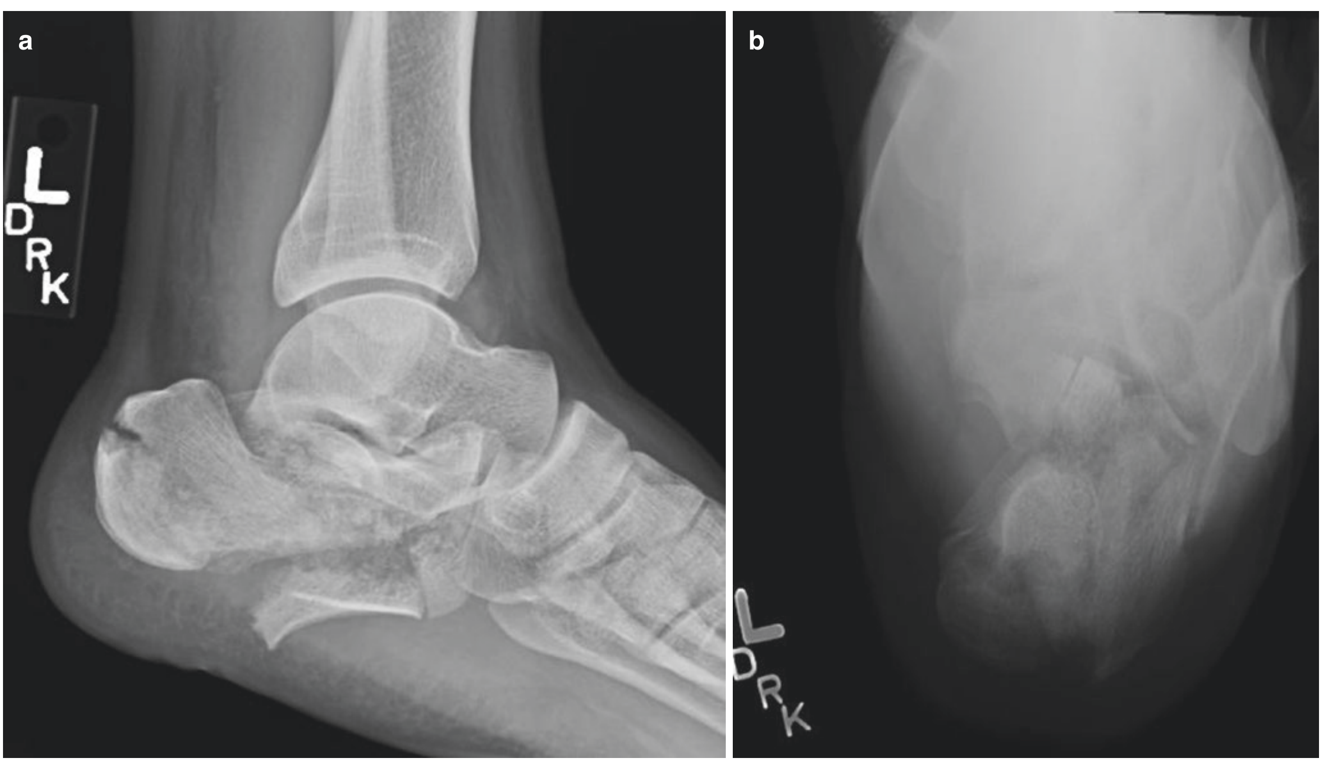

Fig. 27.5 An intra-articular and displaced comminuted calcaneus fracture in a 21-year-old male as a result of tree strike while skiing (**a**, **b**)

Fig. 27.6 An intra-articular and displaced comminuted calcaneus fracture in a 36-year-old male as a result of a fall

help delineate the talocalcaneal joint and plantar aspects of the calcaneus. Specialty views such as axial views of the heel, Harris view, posterior facet views, and Broden's view can also be considered but may be omitted in favor of CT scan [4].

Initial Management

If there is no threat of skin compromise, the ankle can be splinted in neutral position, preferable with a bulky Jones splint to decrease swelling and provide compression [5]. Minimal or moderately displaced tuberosity fractures without evidence of skin compromise can be casted or splinted in 20° of plantarflexion with a cast window or anterior mold to allow monitoring for signs of skin compromise with follow-up in 1–2 days [5, 21]. Posterior splints are also an option, but this makes it difficult to monitor skin integrity. Splinting in plantarflexion helps decrease the deforming force applied by the Achilles and thus risk of further displacement/skin compromise [5]. Keeping the knee in slight flexion can also theoretically decrease this force [5]. Acceptable nonoperative treatment criteria include fractures with articular surface displacement of ≤2 mm; well-preserved calcaneus height, length, and width; and lack of gross varus or valgus alignment of the tuberosity [4]. Minimally displaced (<1 cm) tuberosity fractures without evidence of soft tissue compromise can be treated nonoperatively with casting in plantarflexion as described above [21].

Indications for Orthopedic Referral

Fractures with evidence of skin compromise require emergent treatment and orthopedic referral within a matter of hours [5]. Bohler angle (BA) can also be used to identify calcaneus fractures (Fig. 27.10). This is measured by a line connecting the highest point of the anterior process to the highest point of the posterior facet and a line tangential to the superior edge of the tuberosity. Normal BA is 20–40°. Contralateral views may be helpful in borderline cases. BA <20° are concerning for displacement. BA of <0° are seen in more severe injuries which may later require subtalar fusion, especially if the angle is still decreased after healing [4]. The critical angle of Gissane (Fig. 27.10) extends through the posterior talar articular surface and angles at the point of the tarsal sinus over toward the anterior surface for the cuboid bone [4]. This angle should be between 130 and 145°. Angles >145° are concerning for posterior facet injuries [6]. Abnormalities in both angles suggest fracture displacement and indication for referral. There are conflicting data on the benefits of operative intervention. Several studies demonstrated no improvement in outcome, but others showed functional improvements for operative treatment of displaced fractures [6, 26]. One study showed a sixfold increased rate of subtalar fusion for failed outcomes in patients with displaced intra-articular fractures treated nonoperatively [26]. There is an also increased risk of malunion and posttraumatic subtalar arthrosis in this group [26]. The calcaneus maintains the length of the lateral column and protects the posteromedial arch contents [4]. Therefore, loss of height, heel widening, subfibular and calcaneocuboid joint impingement, and varus heel alignment are seen with nonoperative treatment [26]. Tongue-type calcaneus or displaced tuberosity fractures with skin blanching or breakdown require emergent referral, whereas minimally displaced tuberosity fractures should be referred urgently or monitored every 1–2 days for skin compromise until orthopedic evaluation can be facilitated [21]. Patients with a Bohler angle of 0–14°, a light workload, Sanders Type II fractures, female gender, and age less than 30 years do seem to benefit from operative intervention compared to nonoperatively treated counterparts [4]. Operative treatment of displaced intra-articular calcaneus fractures, DIACFs, has also been associated with decreased direct and indirect costs at the 4-year mark [4]. Sanders Type III and IV fractures have poorer results with either nonoperative or operative treatment, but recent literature seems to suggest a benefit for operative intervention [4, 6]. A 2016 meta-analysis demonstrated decreased chronic pain, subtalar osteoarthritis, and subtalar arthrodesis with operative treatment of DIACFs, but an increase in complications [29]. There was not a significant difference in functional outcomes or problems with wearing shoes between the operative and nonoperative groups.

Follow-Up Care

Conservatively treated fractures not involving the tuberosity can be followed in 1 week for re-evaluation. A posterior splint or CAM walker boot can be used to allow ankle and

Fig. 27.7 A calcaneal (sustentaculum tali) fracture in a 23-year-old female as a result of motor vehicle accident (**a**, **b**). CT scan images demonstrate the extend of the fracture (**c**, **d**)

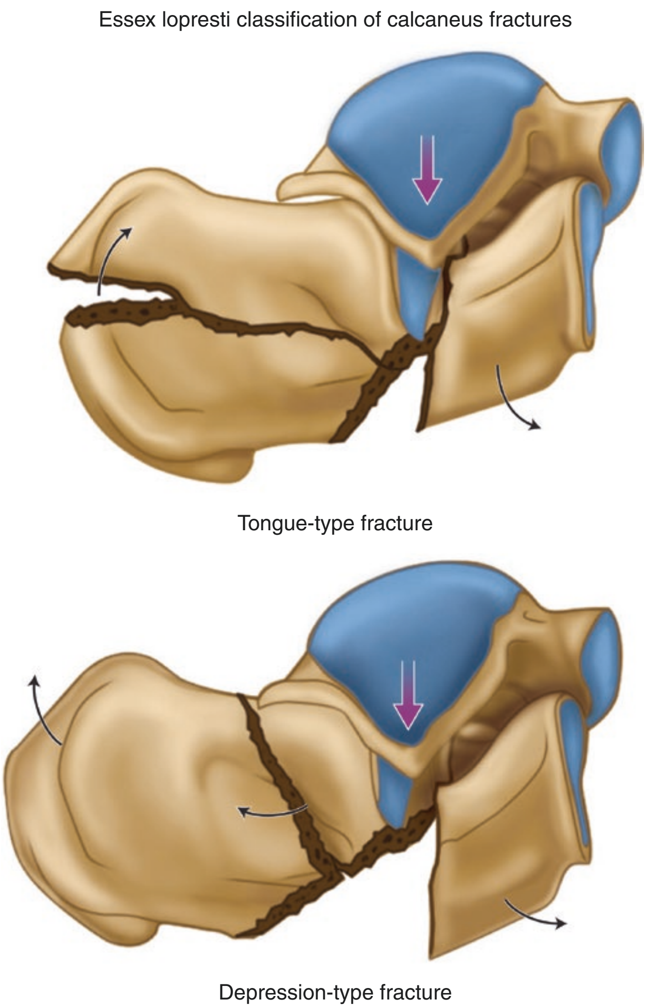

Fig. 27.8 Essex-Lopresti classification of calcaneus fractures

subtalar range of motion, but athletes should be non-weight-bearing for 4–6 weeks and then transition from partial to full protected weight-bearing over 3–6 weeks [6]. Tuberosity fractures which are treated nonoperatively should be re-evaluated in 1 week to ensure no further displacement or soft tissue compromise [21]. At this point the patient may be placed into a short-leg cast in mild plantarflexion. Weekly cast changes or skin checks are recommended throughout the course of immobilization, typically 6–8 weeks [21]. CT scan may be used to confirm fracture union if needed [21].

Return to Sports

Return to sports varies and requires athletes to have regained strength and mobility. Conservatively treated injuries, except Sanders III and IV, can progress back into activity once full weight-bearing is allowed. Both surgically and conservatively treated DIACFs have relatively poor short- and long-term outcomes with many patients having significant disability for 3–5 years and never returning to previous activity levels [4]. These athletes must gradually increase to weight-bearing as tolerated following immobilization and need to regain motion, strength, and function to determine readiness for return to sport [4, 21].

Complications

Unfortunately, many calcaneus fractures will have persistent pain and disability even with appropriate management. Malunions may present with lateral hindfoot pain, due to subfibular impingement, trouble with shoe wear, peroneal stenosis, tendinosis, and possible dislocation [4, 26]. Anterior ankle pain may occur due to loss of height resulting in a more horizontal talus, anterior tibiotalar impingement, and decreased ankle dorsiflexion, and/or arthrosis involving the subtalar and/or calcaneocuboid joints, as well as overall hindfoot varus and lateral column overload [4, 26]. Some of these issues can be corrected surgically, though the recommended procedures may be technically difficult [26]. Partial- or full-thickness skin breakdown in tongue-type fractures which are not treated emergently are more likely to require secondary surgeries for soft tissue deficits [5]. Calcaneocuboid joint involvement and peroneal tendon dislocation are often overlooked at the time of initial injury and can create persistent lateral ankle and foot pain [4]. Tuberosity fractures can also lead to weakness in plantarflexion and difficulty climbing stairs, as well as a bony protuberance [21]. Nonoperative treatment of displaced fractures of the posterior facet can lead to altered gait patterns, particularly on uneven ground [26].

Pediatric Considerations

Calcaneus avulsion fractures involving the apophysis (Fig. 27.11) are rare but do occur and can often be misdiagnosed as a muscle strain or Sever's disease [30]. Sever's disease can also lead to an acute stress fracture with superior displacement of a fragment of the calcaneus apophysis. Limited data is available on these types of injuries, and they are best treated on a case-by-case basis based on symptoms and amount of displacement [30].

Talus

Mechanism of Injury in Sports

Talus fractures typically occur from forced dorsiflexion or a fall from a height [11, 12]. Talar head injuries are thought to be due to a sudden dorsiflexion of a fully plantarflexed foot that compresses the talar head or by hyperdorsiflexion that compresses the talar head against the anterior tibial edge [11]. Impaction talar head fractures may result from subtalar dislocations [11]. Talar neck fractures are believed to occur from forced dorsiflexion [11, 12]. Body fractures including lateral process fractures are thought to be created by com-

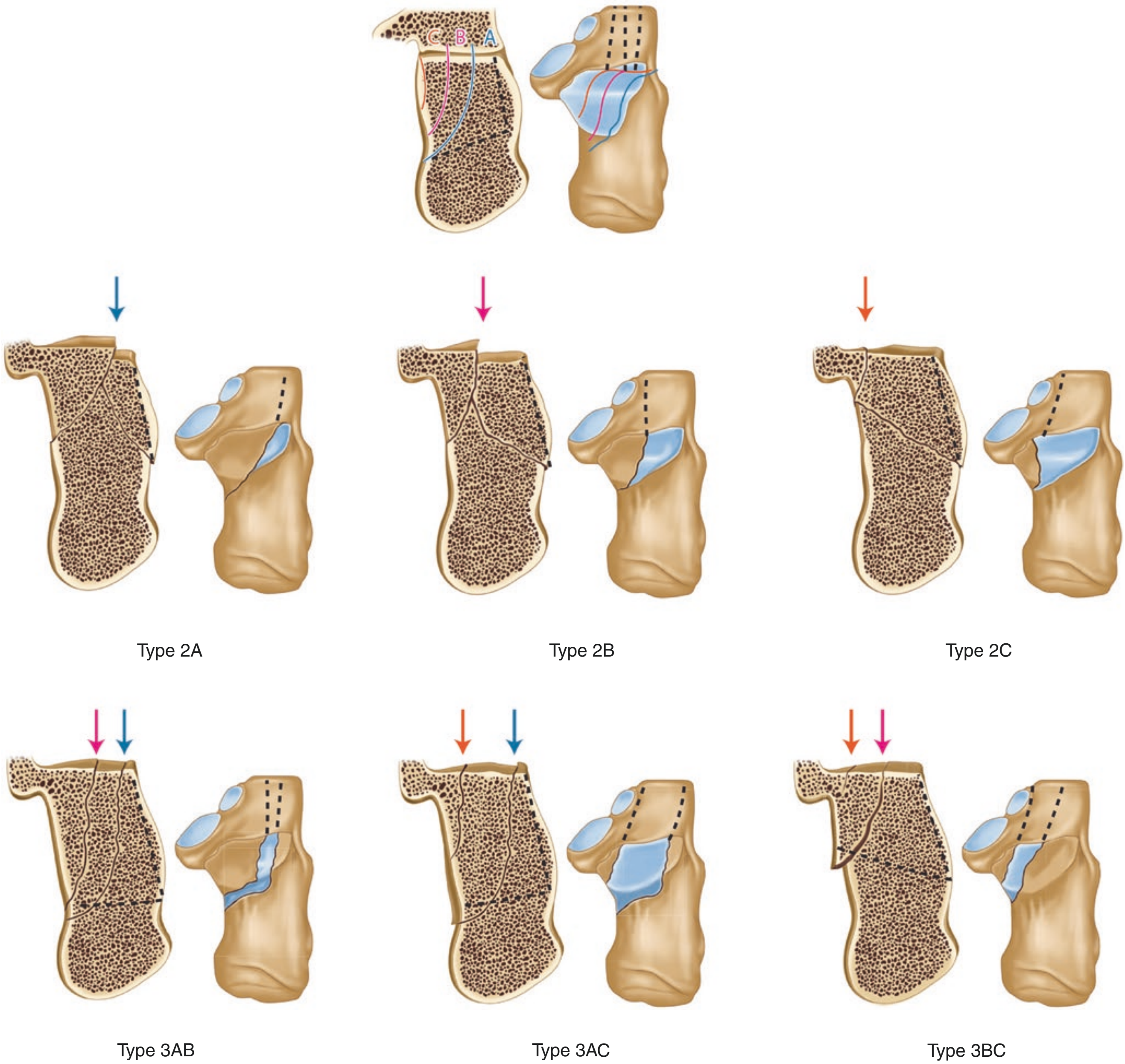

Fig. 27.9 Sanders classification of intra-articular calcaneus fractures in coronal and axial CT views. Type 1 (not demonstrated) are non-displaced fractures. Lines A, B, and C are the primary fracture lines which demonstrate the position of the fracture in regard to the posterior facet of the subtalar joint. Type 2 fractures are that two fragments and Type 3 are fractures with three fracture fragments. Type IV (not demonstrated) are comminuted and displaced fractures

pressive forces created from axial loading while the foot is dorsiflexed. Lateral process injuries occur when this is combined with external rotation or eversion [3, 11].

Epidemiology

Talus fractures are the second most common tarsal fracture but are relatively rare, incidence 0.1–0.85% of all fractures [11]. Individual case reports or series on lateral process talar fractures and cuneiform fractures have been cited in sports such as ice hockey and snowboarding [3, 10].

Classification

Talus fractures are broken down by anatomic location (Fig. 27.12), with neck (Figs. 27.13 and 27.14), body (Fig. 27.15), and lateral process (Fig. 27.16) fractures having individual classifications. Dorsal avulsion fractures are rare

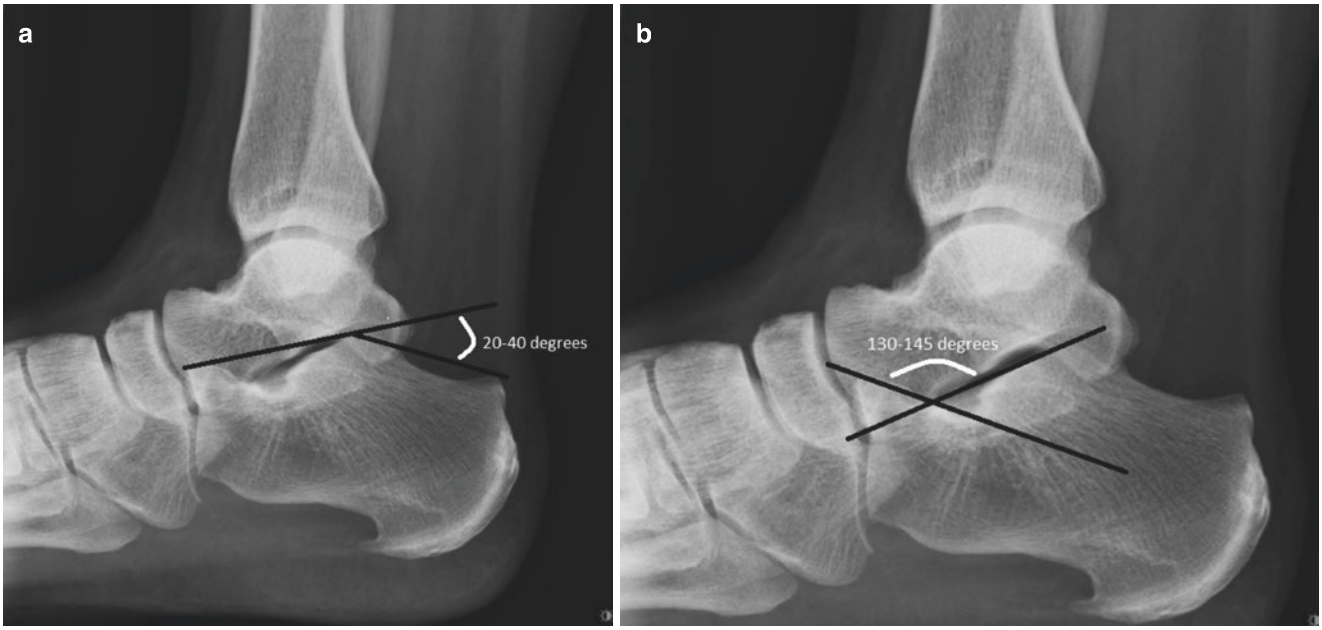

Fig. 27.10 Bohler angle (**a**) and angle of Gissane (**b**) on lateral view

in sports (Fig. 27.17). Talar head injuries do not have a separate classification and would be described on their appearance on imaging (e.g., displacement, comminution). Talar neck fractures, inferior fracture line propagates in front of lateral process, account for 50% of talus fractures and may be associated with fracture of medial malleolus [7, 11]. Talar neck fractures are classified based on displacement (Table 27.1).

Talar body fractures can be hard to differentiate from talar neck and are defined by an inferior fracture line which propagates behind the lateral process involving the posterior facet of the subtalar joint [11]. Body fractures may be classified based on anatomic location (Type A, transchondral or osteochondral; Type B, coronal shear; Type C, sagittal shear; Type D, posterior tubercle; Type E, lateral process; and Type F, crush) or by groups (Group I, proper or cleavage [horizontal, sagittal, shear, or coronal]; Group II, talar process or tubercle; and Group III, compression of impaction) [11].

Lateral process fractures (Fig. 27.16) were originally described by three types [31]. Type I fractures extend from the talofibular articular surface to the posterior talocalcaneal articular surface of the subtalar joint. Type II fractures are comminuted fractures involving both the articular surfaces and the entire lateral process. Type III fractures are chip fractures that involve the anterior and inferior portion of the posterior articular process and do not extend into the talofibular articulation. Others have proposed a more descriptive classification based on bone, chondral, and ligamentous injury, which includes injury to the posterior process (Table 27.2) [32].

Clinical Presentation

Athletes with talus fractures present with pain, swelling, and ecchymosis at the ankle [12]. Peripheral fractures (lateral process, posteromedial talar body, talar head) can be often mistaken for ankle sprain as pain is lateral or medial and complete weight-bearing may not be affected [12, 34]. Athletes with talar head fractures typically present with a history of fall or forced dorsiflexion associated with pain, tenderness, swelling, and ecchymosis in the talonavicular region [11].

Diagnosis

In athletes with suspected talus fractures, plain radiography should include AP, oblique and lateral views of the foot, as well as consideration for standard views of the ankle [4]. For suspected talus injuries, AP and mortise views are used to visualize lateral process and talar dome. Lateral foot and ankle views are used to evaluate for talar neck, tibiotalar, subtalar, and talar navicular articulations. Canale view (Fig. 27.13) is usually used for suspected talar neck fracture in the coronal plane [12]. CT is recommended for further evaluation of suspected injuries, as initial radiographs may not reveal fracture. CT scan can show displacement of talar head fracture, extent of articular injury in body injuries, and angulation and shortening in neck fractures [11, 12].

Initial Management

If there is no concern for skin compromise, a posterior short-leg splint can be used for immobilization. Patients should be non-weight-bearing and have follow-up in

Fig. 27.11 Apophyseal calcaneus fracture (**a**, **b**) in a 10-year-old male as a result of jumping off of a moving truck. Plain radiography 5 weeks later demonstrates callus formation (**c**, **d**)

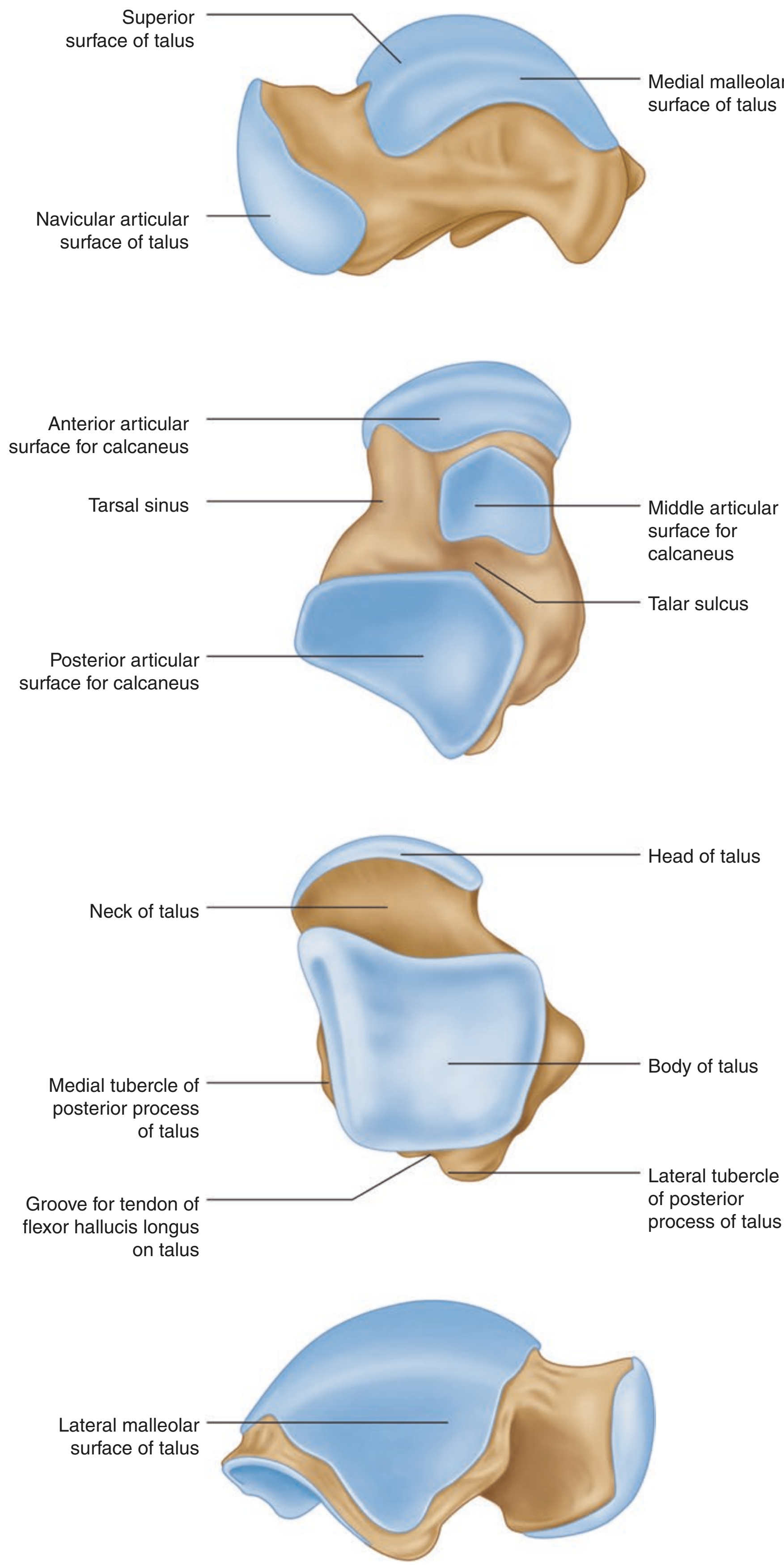

Fig. 27.12 Talus anatomy

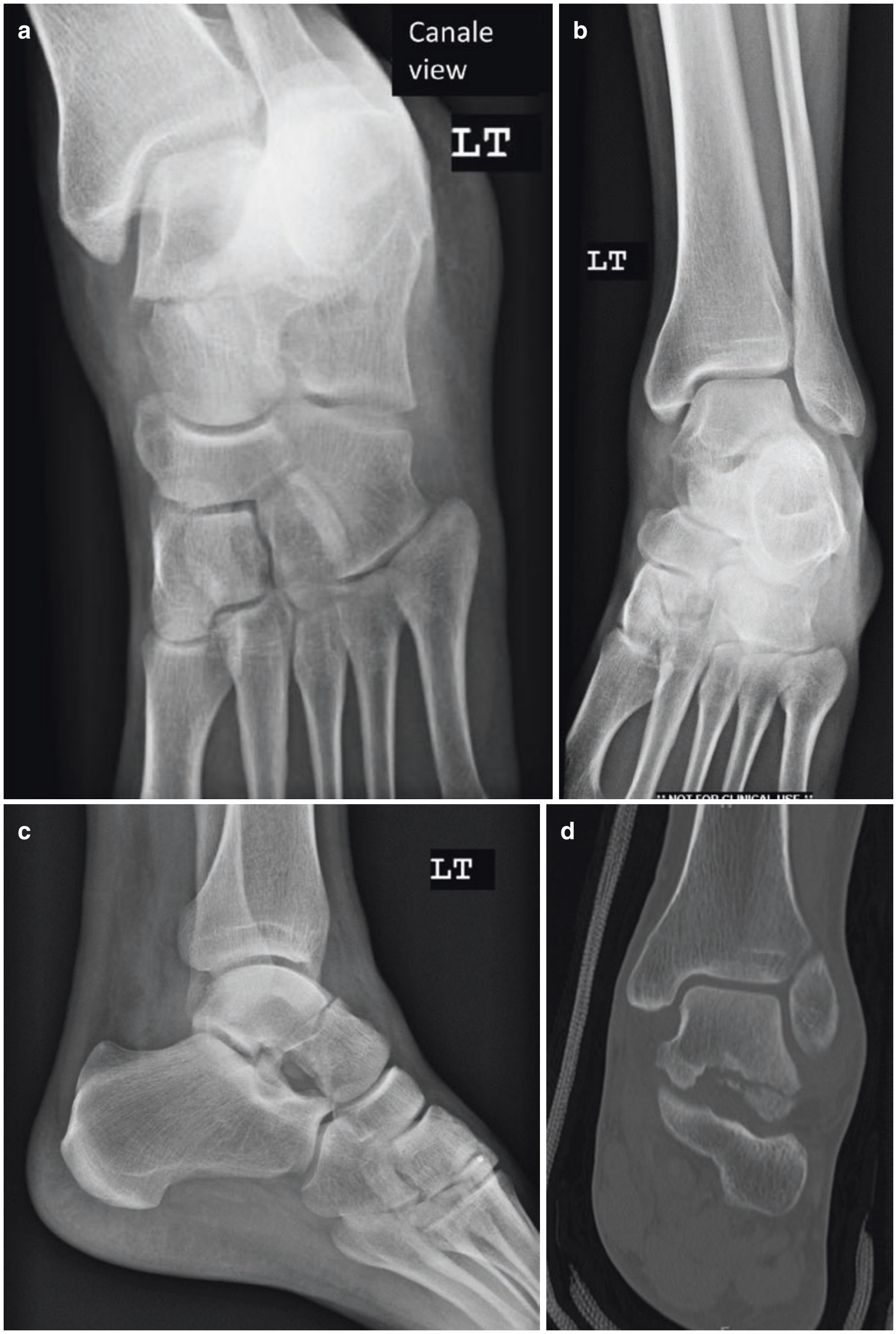

Fig. 27.13 A mildly displaced talar neck fracture in a 25-year-old male as a result of ski injury (**a**–**c**). CT scan demonstrates the extent of the fractures (**d**, **e**). Postoperative images show near-anatomic alignment (**f**, **g**)

Fig. 27.13 (continued)

7–10 days [12]. Fractures that may be considered for conservative treatment include non-displaced talar head fractures, minimally displaced process fractures, and truly non-displaced body and neck fractures, confirmed by CT [7, 11]. Although displaced talar neck fractures require surgical fixation, reduction can be attempted by plantarflexing the ankle and manipulating the heel in inversion and eversion [11]. If the soft tissue is not compromised, splint, elevate, and consider cryotherapy and pneumatic compression to decrease swelling prior to surgery [12].

Indications for Orthopedic Referral

Urgent referral, within hours, should be undertaken for talar neck fractures, Types III and IV, open fractures, and displaced fractures. Displaced injuries with significant stretching of dorsal soft tissues must be promptly reduced

to avoid skin and bone necrosis. Avascular necrosis occurs as a result of initial injury, not as a result from delays in fixation, and the timing of surgery depends on the condition of soft tissues and may be delayed based on these factors [12]. Type II talar neck fracture can be referred on a nonurgent basis [12]. Talar head fractures that exhibit instability of the talonavicular joint or articular incongruency should be referred [11]. Talar body fractures that are displaced or have significant articular involvement should be considered for referral. An extruded talar body needs to be handled emergently [12]. Extra-articular process fractures, lateral and posterior, should be referred for >2 mm of displacement. All other fractures that involve the articular surface should be referred for surgical evaluation [33]. Persistent pain with process fractures should also be referred for consideration of excision [34].

Fig. 27.14 Comminuted and displaced calcaneus and talus fractures in a 23-year-old female as a result of tree strike while snowboarding (**a**, **b**). CT scan demonstrates the extent of the fractures (**c**). Postoperative images show near-anatomic alignment (**d**, **e**)

Fig. 27.14 (continued)

Follow-Up Care

Athletes with non-displaced fractures of the talar head can be converted to a short-leg non-weight-bearing case for 6–8 weeks [11, 12]. Follow-up interval is generally every 1–2 weeks. Talar neck fractures should be placed in a non-weight-bearing short-leg cast in slight equinus and may be changed at 4 weeks to ankle neutral. Non-weight-bearing should be continued for 6–8 weeks. Range of motion and progress from partial to full weight-bearing can occur from 7 to 12 weeks [11, 12]. Follow-up radiographs should be obtained weekly or biweekly for the first 4 weeks to ensure stability – consider operative care of concern for displacement [12]. Posterior and lateral process injuries are generally treated with 6–8 weeks of non-weight-bearing and then gradual progression to full unprotected weight-bearing [34].

Return to Sports

Return to sports after talus fractures is based on both clinical and radiographic fracture healing. Gradual progression can begin once full weight-bearing is allowed and should be individualized based on the fracture and athlete as there are no specific guidelines to determine return to play. This generally will occur over 1–2 months, sometimes longer, once weight-bearing has been allowed.

Complications

Multiple complications may occur from talus fractures; however it is not clear how early versus late recognition of some fractures affects outcomes. Talar head fractures may go unrecognized and can cause loss of medial column support and talonavicular joint instability and posttraumatic arthritis [11]. Avascular necrosis of the talus can occur but nonunion is rare [12]. Type III neck fractures may create medial and posterior protrusion of the talar body which can create compression of the tibial neurovascular structures [11]. Other complications of neck fractures include osteonecrosis, posttraumatic arthritis, malunion, nonunion, and infection [11, 12]. Malunion is common (30%) with varus malunion being most frequent, particularly after closed treatment of Hawkins Type II talar neck fracture [11, 12]. These are typically treated operatively when symptomatic [12]. Complications of talar body fractures include varus malalignment, arthrosis, osteonecrosis, malunion, and skin necrosis/infection, while nonunion of these injuries is infrequent [11]. Talar body fractures with associated dislocation have a higher incidence of osteonecrosis [11]. Osteonecrosis can be observed on radiographs as relative opacity of involved bone caused by osteopenia of neighboring bones secondary to disuse 4 weeks–6 months following fracture or fracture-dislocation [11]. Hawkins sign is a patchy subchondral osteopenia on AP and mortise ankle views seen 6–8 weeks after injury and is reassuring that osteonecrosis is unlikely. This finding is best seen on comparison with contralateral side [11]. MRI is generally not helpful until at least 3 weeks after injury, but may not change initial treatment even if early changes of osteonecrosis are present as initial treatment is conservative and revascularization may take up to 36 months [11]. It may, however, result in the collapse of the entire talar dome [12]. Arthrosis of the subtalar and tibiotalar joints can occur and may limit range of motion. This can be from the initial injury or from abnormal joint mechanics, but is commonly not symptomatic [11]. Both lateral and posterior process injuries have relatively poor vascularity and higher risk of nonunion and persistent pain [12, 33].

Pediatric Considerations

Talus fractures are rare in children, but most are non-displaced fractures of the talar neck and are treated similar to non-displaced fractures in adults [35]. Displaced fractures are usually associated with other injuries and may require

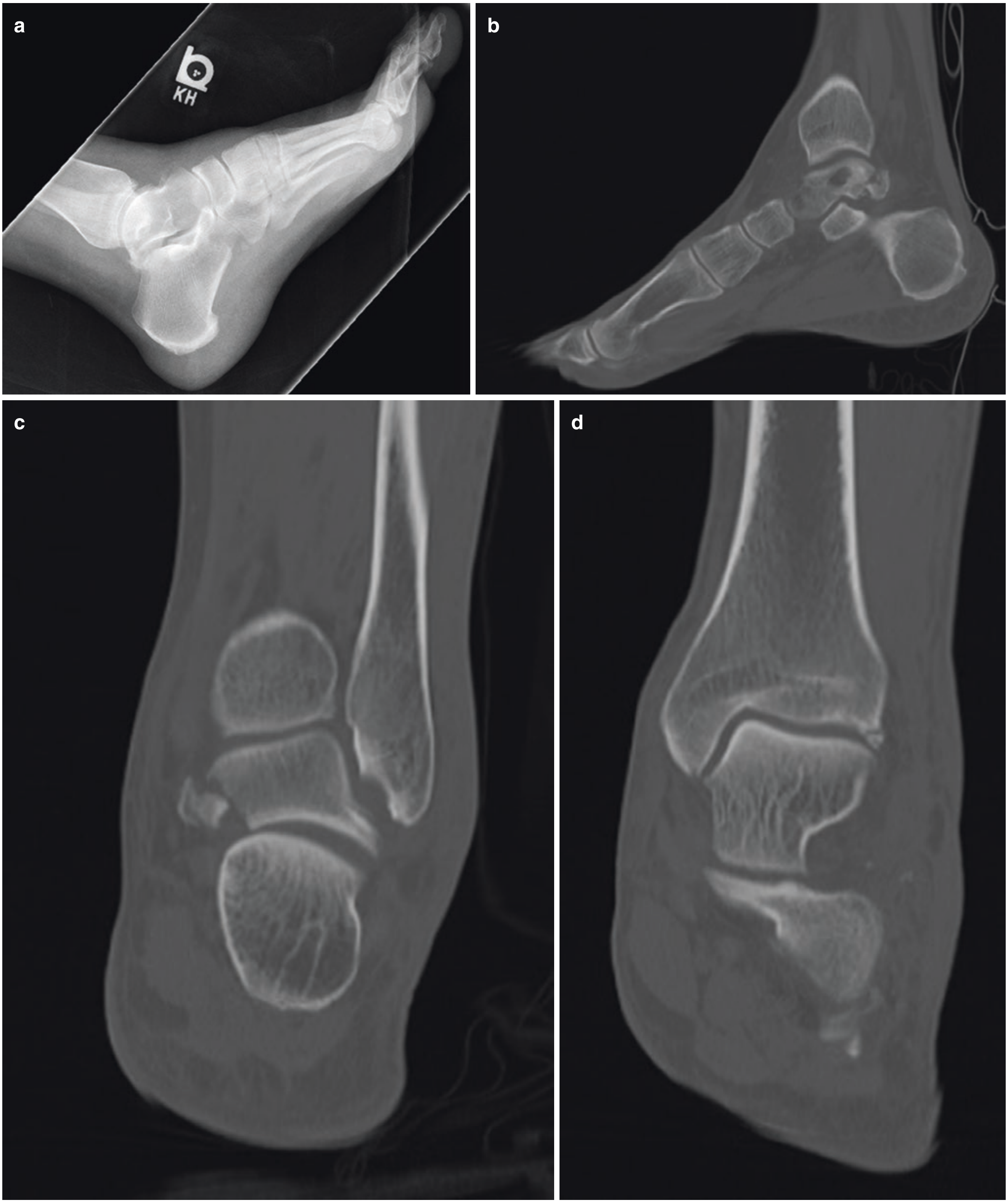

Fig. 27.15 Fracture of the talar body (posteromedial) in a 53-year-old male not visible on plain radiography (**a**). CT images revealed talus fracture (**b**, **c**) as well as avulsion fracture of the lateral distal tibia (**d**). Postoperative images show near-anatomic alignment (**e**)

Fig. 27.15 (continued)

Fig. 27.16 Lateral talar process fracture (open arrow)

Fig. 27.17 Avulsion fracture of the distal dorsum of the talus (arrow) in a 19-year-old male as a result of basketball injury

surgical intervention. The most significant complication is avascular necrosis [35].

Navicular

Mechanism of Injury in Sports

Navicular avulsion fractures (Fig. 27.18) involving the talonavicular ligament occur in extreme inversion and plantarflexion [8, 13, 36]. Avulsions involving the deltoid ligament occur with extreme eversion, and tuberosity avulsions result from acute eversion or valgus injury to the fore-

foot [8, 13, 36]. Avulsion injuries of the medial tuberosity are thought to occur from forceful contraction of the tibialis posterior attachment [13]. Other types usually require high-energy mechanism due to a robust ligamentous network consisting of plantar and dorsal ligaments, the posterior tibial tendon, the plantar calcaneonavicular (spring) ligament, and deltoid ligament. When they do occur, they are caused by crush injuries, axial loading of the plantarflexed foot, or dorsiflexion in the setting of hindfoot eversion [8, 13, 36].

Epidemiology

Navicular avulsion fractures account for 50% of acute tarsal navicular fractures and have high rates of concurrent midfoot and hindfoot injuries [8]. Occult injuries, diagnosed by MRI, to the navicular and cuneiforms have been reported in ice hockey [20].

Table 27.1 Talar neck fracture classification [7, 11]

Type I	Non-displaced
Type II	IIA – Subluxation of subtalar joint
	IIB – Dislocation of subtalar joint
Type III	Talar body displaced from subtalar and ankle joints (>50% are open fractures)
Type IV	Talar body displaced from subtalar, ankle, and talonavicular joints (rare)

Table 27.2 Classification of talar process fractures [32]

Type I: small chip or avulsion fracture (<0.5 cm)	1a – small (extra-articular) fragment of the lateral process of the talus
	1b – small fragment of the isolated medial tubercle of the posterior process
	1c – small (intra-articular) fragment of the lateral process of the talus
Type II: intermediate fragment (0.5–1.0 cm) with some displacement	2a – extends into the subtalar joint but not to the talofibular joint
	2b – isolated fracture of the entire lateral tubercle of posterior process
Type III: a large fracture fragment (>1 cm) with associated damage to both the ankle and the subtalar joints	3a – single large fragment of the lateral process extending from the talofibular articular surface to the posterior facet of the subtalar joint
	3b – comminuted fracture of the entire lateral process
	3c – fracture of the entire posterior process of the talus
Type IV: a severe form of fracture of either of the processes and associated instability or dislocation of the subtalar joint	

Although some authors recommend treatment based on classification, studies have not clarified a preferred classification system [33]

Fig. 27.18 Avulsion fracture of the proximal and dorsal navicular bone (arrow) in a 56-year-old feamle as a result of twisting her foot (**a**). AP view shows a cuboid fracture (open arrow) as well (**b**)

Classification

Acute navicular fractures can be classified based on location and type, avulsion, tuberosity, and body. Body fractures can be further subdivided. Type 1 injuries have a transverse fracture line in the coronal plane, a dorsal fragment <50% of the body, no angulation of the forefoot, or disruption of the alignment of the medial border of the foot. Type 2 injuries are common, and the primary fracture line runs dorsal-lateral to plantar-medial, and the major fragment is displaced medially. Type 3 injuries are comminuted fractures in the sagittal plane disrupting the medial border of the foot and laterally displacing the forefoot [13, 37].

Clinical Presentation

Acute navicular fractures are often subtle and frequently diagnosed on a delayed basis [8]. Avulsion and tuberosity fractures usually present after a twisting injury with swelling either diffusely or involving the dorsomedial midfoot with tenderness over the navicular, limited weight-bearing, pain with push-off, and loss of the longitudinal height of the foot [8].

Diagnosis

For navicular fractures AP, lateral, and oblique views of the foot are typically sufficient; weight-bearing is preferable. Stress views may be used to demonstrate ligamentous injury. Tuberosity fractures must be distinguished from accessory navicular, and comparison radiograph of contralateral foot may be helpful. Tuberosity injuries are best visualized with external oblique views. CT can be helpful to assess the full extent of injury and intra-articular involvement [8].

Initial Management

Fractures involving the body of the navicular often require surgical intervention. Non-displaced fractures or fractures with <2 mm of displacement or <3 mm of medial column shortening can be considered for conservative treatment [13, 36]. A posterior splint can provide comfort and immobilization initially. Athletes should be non-weight-bearing initially. Avulsion injuries can be treated in a CAM walker with weight-bearing as tolerated. If significant swelling exists, some authors recommend a period of non-weight-bearing [13].

Indications for Orthopedic Referral

Open wounds, concern for swelling and compartment syndrome, gross instability, skin compromise, and irreducible dislocations should all be urgent referred for orthopedic intervention. Nonurgent referrals should be made for displacement or joint incongruity >1 mm, medial column shortening >2 to 3 mm, subluxation, and lateral column involvement [13].

Follow-Up Care

As no specific recommendations about length of non-weight-bearing and type of immobilization exist, provider preference may vary. Consideration of a short-leg non-weight-bearing cast for 4–6 weeks with transition to CAM walker for partial weight-bearing is reasonable. Fractures should be followed weekly for 1–2 weeks with radiographs with follow-up at 4–6 weeks for radiographs and possible transition to weight-bearing. Avulsion fractures with minimal swelling can be treated in a CAM walker boot for 4–6 weeks with weight-bearing as tolerated. For injuries with more significant swelling, patients may be made non- or partial weight-bearing for the initial few weeks with some suggesting up to 6–8 weeks [13].

Return to Sports

Patients with dorsal avulsion fractures or tuberosity fractures can typically return to full activity in 2–3 months, whereas body fractures may take up to 4 months [1]. Return is based on fracture healing and return to functional activities and should be individualized.

Complications

Persistent stiffness, pain, loss of hindfoot motion, hindfoot varus secondary to progressive lateral collapse, and posttraumatic arthritis are common complications [8, 36]. Avascular necrosis and nonunion may occur secondary to poor blood supply [8, 36].

Pediatric Considerations

Dorsal avulsion fractures (Fig. 27.19) are the most commonly seen navicular fracture in pediatrics. These injuries generally heal well with conservative treatment with protected weight-bearing for 3–4-weeks [1].

Cuboid

Mechanism of Injury in Sports

Cuboid avulsion fractures are believed to be caused by twisting injuries. Hindfoot inversion associated with forefoot adduction and external tibial rotation resulting in avulsion of the calcaneocuboid portion of the bifurcate ligament [9, 14]. Crush injuries can occur from axial loads transmitted through the fourth and fifth metatarsals, or abduction of the forefoot relative to the hindfoot. Other mechanisms include direct crush or indirect rotational injuries, forcible eversion of the forefoot on the hindfoot, high-energy axial loads with an extended midfoot, or axial loading of the heel with the foot in fixed plantarflexion, compressing the lateral column of the

Fig. 27.19 Avulsion fracture of the proximal and dorsal navicular bone in a 17-year-old male as a result of soccer injury

foot [9, 14]. Comminuted fractures typically occur as a result of direct impact to the lateral foot [14].

Epidemiology

Cuboid fractures are rare and account for half of all midfoot fractures, with an incidence of 1.8 per 10,000 in the United Kingdom annually [9, 14]. Avulsion fractures (Fig. 27.20) are the most common and may be associated with other midfoot fractures (Fig. 27.18), including Lisfranc complex (Chap. 28; Fig. 28.24) [9, 14].

Classification

Cuboid fractures do not have a validated classification system. The Orthopaedic Trauma Association classifies fractures as simple avulsion, minimally displaced, or comminuted which generally includes crush injuries [14]. Nutcracker (compressed) fractures are extensions of crush injuries caused by compression through the lateral column [14]. Others have described classifications based on the pattern of the injury [9]. In this system Type 1 injuries are simple avulsions involving the calcaneocuboid joint. Type 2 injuries are isolated extra-articular injuries involving the body of the cuboid and can be associated with avulsion fractures to the base of the fifth metatarsal. Type 3 fractures are intra-articular within the cuboid body, and Type 4 fractures are associated with midfoot disruption and tarsometatarsal injuries. Type 5 injuries include disruption of the mid-tarsal joint and crushing of the lateral column in Type 5a or lateral and medial column in Type 5b [9]. Type 5a injuries are often seen in conjunction with navicular tuberosity avulsions [9].

Clinical Presentation

Cuboid fractures often present similar to lateral ankle sprains. They present with localized swelling and tenderness with severe pain with movement of the midfoot [1] Ability to weight-bear is variable based on severity of the injury.

Diagnosis

Cuboid avulsion fractures are rarely diagnosed on routine x-rays as fracture fragments are missed or misinterpreted as accessory bones [14]. In addition to standard AP, oblique, and lateral views, comparison views of the contralateral foot may aid in detection of decreased length of the lateral column as well as differences in cuboid shape and position [14]. CT scan can aid in fracture detection as well as classification, but is not always necessary for treatment planning [14].

Initial Management

Cuboid avulsion fractures, Type 1, usually present similar to sprains and can be treated with protected weight-bearing, supportive footwear, or a CAM walker boot, for comfort. Crush injuries should be evaluated for any skin compromise and can be treated with posterior splinting or a CAM walker boot with non-weight-bearing initially [14]. Most other cuboid fractures will require surgical intervention. Fractures with minimal articular involvement and normal morphology can be treated conservatively but may require external fixation if the lateral column is shortened [14]. Type 5a may be treated nonoperatively if the articular surfaces are congruent and the length of the lateral column is maintained [9].

Indications for Orthopedic Referral

Athletes with open fractures, skin tenting, dislocation, severe lateral column shortening, and/or significant articular incongruity all should be referred urgently for evaluation [14]. Type 2–5 fractures usually require surgical intervention, and although some may be treated conservatively, referral should be strongly considered based on provider experience. Most closed fractures should be managed in a delayed fashion to facilitate recovery of associated soft tissue injuries, which are common [14].

Follow-Up Care

Type 1 fractures managed symptomatically can be followed as needed based on the athlete. Repeat radiographs

Fig. 27.20 Avulsion fracture of the cuboid (arrows) in a 38-year-old female as a result of an inversion ankle injury (**a**, **b**). CT scan reveals the extent of the fracture (**c**–**e**)

Fig. 27.20 (continued)

are not generally necessary [9]. Crush injuries that are treated conservatively are managed with immobilization and protected weight-bearing for 6 weeks followed by progressive weight-bearing and mobilization, although some authors recommend 6–8 weeks of non-weight-bearing [9, 37]. Type of immobilization is provider dependent, cast versus CAM walker, and repeat visits and radiographs are generally performed weekly for the first few weeks and then around 6 weeks to assess readiness to progress weight-bearing.

Return to Sports

Type 1 fractures can progress back into sporting activity as pain and function allow. Taping or orthotic support may be helpful. Other fractures will vary based on the individual injury and provider with progression to functional status after healing being most important. Closed treatment with external fixators and/or operatively treated fractures usually take 10–12 weeks to return to full weight-bearing and progress toward return to sports from that time varies [14].

Complications

Posttraumatic arthritis is the most common complication. Residual planus or planovalgus deformity can occur from lateral column shortening. Residual articular incongruity or malunion can lead to alterations in gait and peroneus longus tendinopathy [14].

Pediatric Considerations

Cuboid fractures are rare in children and occur usually with a fall from height, but other mechanisms including injury during soccer have been reported [38, 39]. Treatment is similar to adults with the most common surgical indications being lateral column shortening and open injuries [38].

Cuneiform

Mechanism of Injury in Sports

Cuneiform fracture rarely occurs in isolation (Chap. 28; Fig. 28.24), but when they occur, blunt direct trauma, such as a puck hitting a skate, is the most commonly described mechanism [10]. Avulsion fractures of the medial cuneiform may occur in ankle sprains and are thought to be caused by traction of the tibialis anterior tendon [15]. Combined injuries and fracture dislocations may occur with an axial load to the midfoot creating hyperflexion. The degree of force and associated positions, i.e., supination or pronation, may create associated injuries to additional surrounding structures [15].

Epidemiology

Isolated cuneiform fractures are extremely rare accounting for 1.7% of all tarsal fractures, and occult bone contusions have been found in ice hockey [10, 20].

Classification

Cuneiform fractures do not have a separate classification system and can be described by location, displacement, and other factors such as comminution. Some authors suggest description based on the number of bones affected [15]. Type A involve a single cuneiform. Types B and C involve two or three cuneiforms, respectively. Subtyping in each category is based on fracture versus dislocation with subtype 1 fracture alone, subtype 2 dislocation alone, and subtype 3 fracture-dislocation. Type A injuries almost always involve the medial cuneiform [15].

Clinical Presentation
Cuneiform fractures usually present with difficulty weight-bearing, tenderness, and swelling over the dorsal midfoot but may present with severe deformity if fracture-dislocation is present [10, 15].

Diagnosis
Cuneiform fractures may be missed on plain radiographs due to superimposition of overlapping articulations of mid-tarsal joints [10]. Consideration of CT or MRI is indicated for high clinical suspicion and concern for other associated injuries [10]. Bipartate medial cuneiforms have an incidence of 0.1% and may be mistaken for a fracture [40]. Bipartate cuneiforms have plantar and dorsal segments in the long axis of the foot which is uncommon for fractures [40].

Initial Management
Non-displaced isolated cuneiform injuries can be treated with initial protected weight-bearing in a splint or CAM walker boot. Ankle and foot range of motion can be advised as pain allows [10]. Non-displaced multiple fractures can also be considered for conservative treatment but often involve displacement or other injuries that require interventions [15].

Indications for Orthopedic Referral
Fractures associated with dislocations or displacement should be protected in a non-weight-bearing posterior short-leg splint and referred urgently, within hours, for reduction and fixation. Dislocations are generally reduced as soon as possible while the timing of displaced fractures may vary based on type and degree of swelling [15].

Follow-Up Care
The timing of repeat examination and radiographs varies based on the type of injury and provider preference. Conservatively treated fractures can be followed in 1–2 weeks to evaluate swelling and to re-examine for associated injuries. Repeat radiographs can be considered if there is any concern for stability or displacement. Transition to full unprotected weight-bearing can occur at 4–6 weeks. Healing is usually determined clinically by absence of pain, as repeat radiographs may not show much change for non-displaced injuries [10].

Return to Sports
As pain improves, athletes may progress from low impact activities toward impact exercise based on sport. Ankle and foot strength exercises can be added to range of motion as pain allows [10]. Athletes may be able to return to lower impact sports between 4 and 8 weeks, but a longer time may be needed for sports that involve running and jumping [10].

Pediatric Considerations
Isolated fractures of the cuneiform are rare in adults [10] and thought to be rare in children. Fractures associated with Lisfranc injuries can occur and are commonly associated with sporting activity [41].

Dislocations

Tarsal dislocations are rare in trauma and even more rarely reported in sports. Case reports of isolated dislocations of the calcaneus, navicular, and cuneiforms have been published [15, 42–44]. Subtalar dislocations which include injury to the talocalcaneal and talonavicular joints have been more commonly reported (Fig. 27.21). However, subtalar dislocations are still rare injuries accounting for only 1–2% of dislocations [45]. These injuries usually occur with high-energy mechanism with approximately 75% of injuries occurring in motor vehicle accidents and falls from height [45]. A recent review found 14% of injuries occurred from sports injuries and case reports in volleyball, basketball, and football have been published [44, 46, 47]. The majority (~75%) of dislocations are medial with lateral dislocation being the next most common at approximately 15% of injuries [46, 48]. Anterior and posterior dislocations are rare [46]. Medial dislocations are caused by forced inversion when the foot is in a plantarflexed position and lateral dislocations occur with the ankle in dorsiflexion with forced eversion [45]. These injuries may occur with or without associated fractures. Presentation is usually obvious with significant deformity of the ankle. Standard ankle radiographs (Fig. 27.21) usually show the dislocation but may not elucidate any associated fractures.

Both isolated tarsal bone dislocation and subtalar dislocations require emergent reduction and evaluation for associated injuries [42–44]. Most subtalar dislocations can be treated with closed reduction, but general or regional anesthesia is usually required [44]. CT scan imaging after reduction can help identify associated fractures. Immobilization in a non-weight-bearing cast for 6 weeks is the usual treatment for subtalar dislocations. Return to activity is based on resolution of pain, regaining strength and range of motion. The majority of patients have good outcomes but complications such as avascular necrosis and open injuries can occur [46]. Posttraumatic subtalar and talonavicular arthritis is common [44].

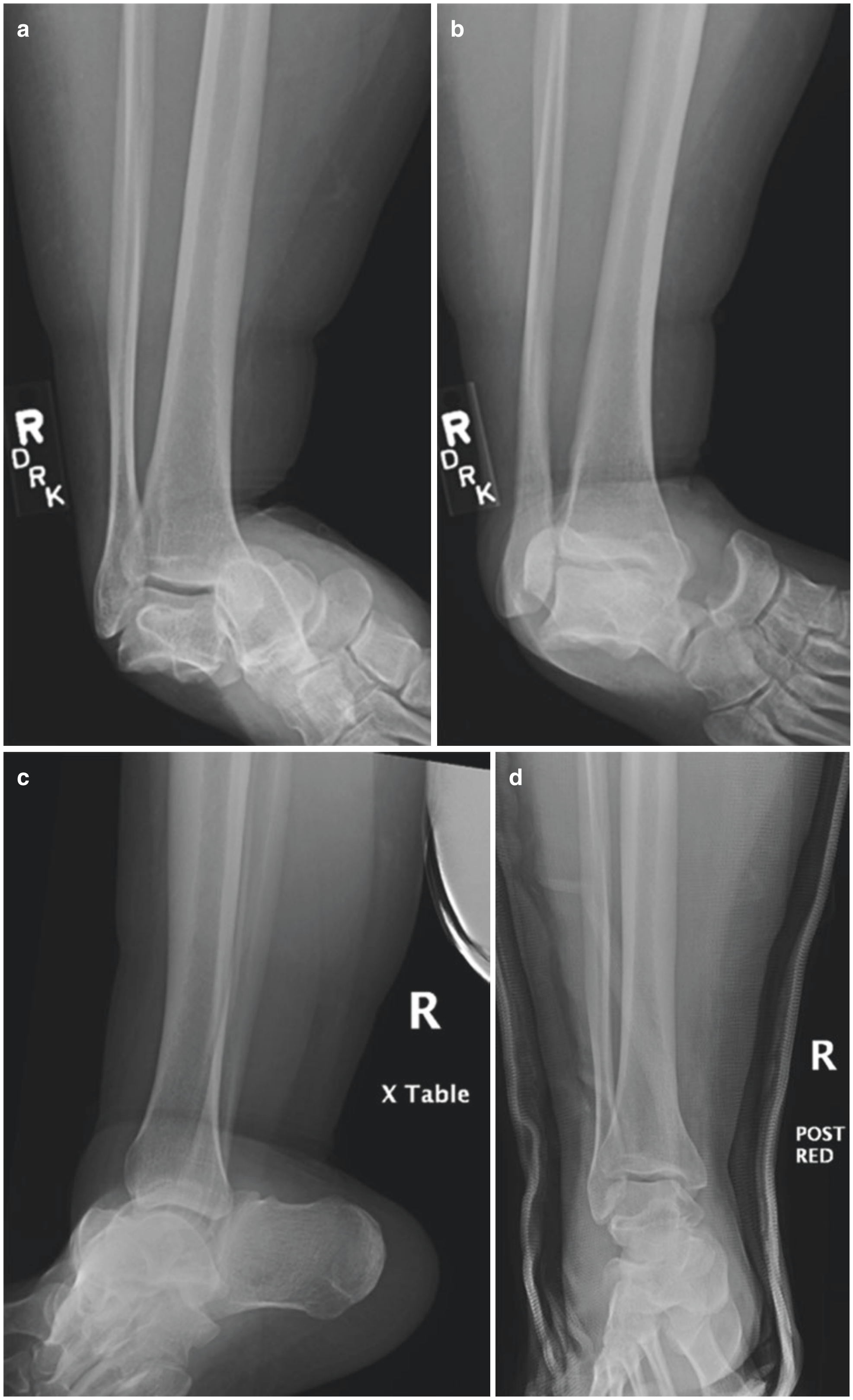

Fig. 27.21 Subtalar (talocalcaneal and talonavicular joints) dislocations in a 44-year-old morbidly obese female as a result of a fall while biking (**a**–**c**). Post-reduction images demonstrate anatomic alignment (**d**, **e**). No fracture was detected on CT scan

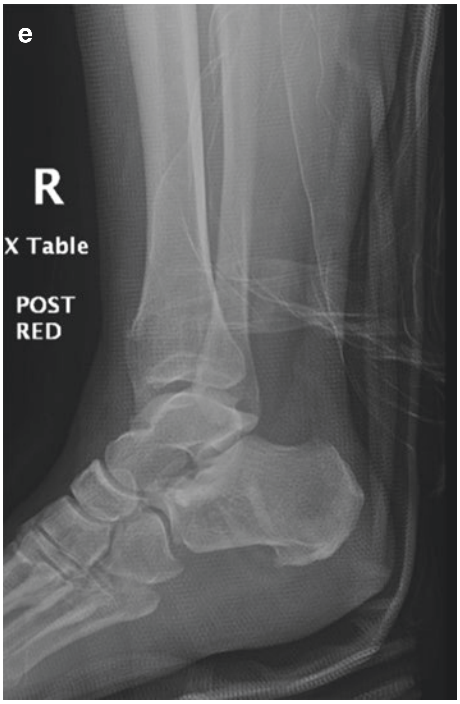

Fig. 27.21 (continued)

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28 Metatarsus

Nathaniel S. Jones, Ellen Kroin, and Adam Schiff

Key Points

- Metatarsal fractures are common in sports.
- Most metatarsal fractures can be treated conservatively.
- Displaced or intra-articular fractures of the first metatarsal should be considered for surgical fixation.
- Pain at the base of the first or second metatarsals should raise suspicion for Lisfranc injuries.

Introduction

Metatarsal (metatarsus) fractures can be seen in a variety of sports and recreational activities. About 80% of metatarsal fractures are non-displaced or minimally displaced and therefore can be managed conservatively in most cases [1]. The goal of treatment is to restore alignment of the five metatarsals to preserve both the longitudinal and transverse arch of the forefoot and maintain a normal weight-bearing distribution under the metatarsal heads [2]. Most athletes can transition to weight-bearing as tolerated and progress back to sporting activity when the fracture heals, generally 4–8 weeks from date of injury. However, return to sports varies based on the fracture and type of sport. Fractures of the first metatarsal, some fractures of the base of the fifth metatarsal, and multiple fractures, particularly those about the base of the second and third, should prompt consideration for consultation.

N. S. Jones (✉)
Department of Orthopaedic Surgery & Rehabilitation, Health Sciences Campus, Loyola University Medical Center, Maywood, IL, USA
e-mail: jonen@lumc.edu

E. Kroin
Department of Orthopaedic Surgery, Loyola University Medical Center, Maywood, IL, USA

A. Schiff
Department of Orthopaedic Surgery, Loyola University Medical Center, Maywood, IL, USA
e-mail: adam.schiff@lumc.edu

Anatomy

The first metatarsal is an important component of the arch of the foot and supports twice the weight of each of the other metatarsals [2]. It is the thickest, widest, and shortest of the metatarsals and has a plantar crista that articulates with the tibial and fibular sesamoids (Fig. 28.1). The base of the first metatarsal articulates with the medial cuneiform. Two powerful extrinsic muscles attach to the base of the first metatarsal (Fig. 28.2). The tibialis anterior inserts on the plantar medial aspect of the base of the first metatarsal, while the peroneus longus attaches onto the plantar lateral base [3]. The tibialis anterior elevates the first metatarsal and the peroneus longus serves to plantarflex the head. There is no inter-metatarsal ligament between the first and the second metatarsal, allowing for independent motion in order to help adaptability to uneven surfaces. The second, third, and fourth metatarsals are broader dorsally than plantarly, forming a trapezoidal shape. The second metatarsal articulates with all three cuneiforms, the third metatarsal articulates with the lateral cuneiform, and the fourth metatarsal articulates with the cuboid. They are all part of the tarsometatarsal (TMT) joint. The bases of these three central metatarsals combine to form a configuration resembling the keystone of a Roman arch in the coronal plane. The second metatarsal is normally the longest and is locked into its position at the notch formed by the three cuneiforms [3]. The second, third, fourth, and fifth metatarsal bases are interconnected both on the dorsal and plantar aspect by transverse ligaments (dorsal, plantar, and interosseous) which act to stabilize each metatarsal with its neighbor. Distally, the

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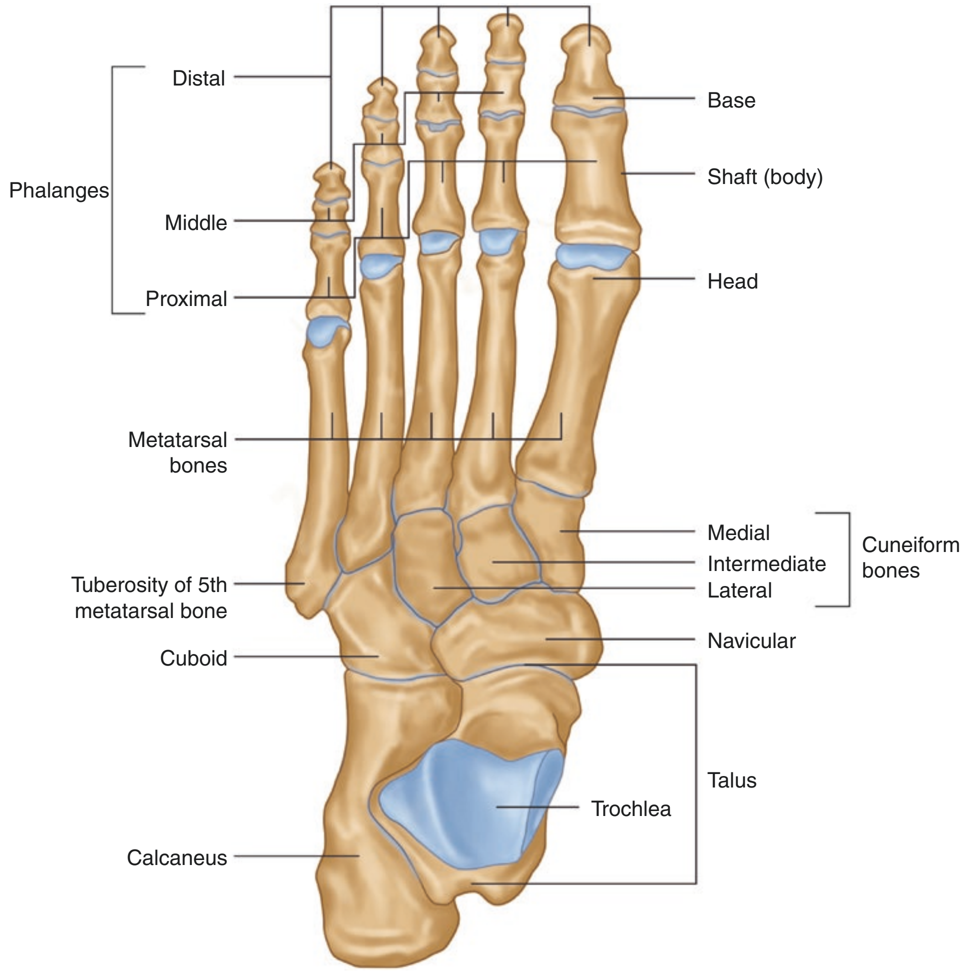
Fig. 28.1 Bony anatomy of the foot



thick transverse metatarsal ligaments connect the metatarsals indirectly by linking the plantar plates of the adjacent metatarsophalangeal (MTP) joints. The Lisfranc ligament runs from the plantar aspect of the medial cuneiform to the base of the second metatarsal, and to a lesser extent to the base of the third, thus inadvertently connecting the lateral four metatarsals to the medial side of the forefoot [4]. There is an increase in range of motion through the TMT joints beginning at the second metatarsal and going out to the fifth. This further allows for adaptability to uneven terrain by the metatarsal heads [4].

The intrinsic dorsal and plantar interosseous muscles originate from the shafts of the metatarsals. Extrinsic muscular attachments are slips from the tibialis posterior tendon, which insert on the plantar base of the second, third, and fourth metatarsals [5]. The combination of these soft tissue structures limits displacement of isolated metatarsal shaft fractures. However, both the plantar flexion force of the intrinsic flexors (musculi interossei and lumbricales) and the extrinsic flexors can force the distal fragment of the metatarsal fracture in a plantar and proximal position.

Like the first metatarsal, the fifth metatarsal has distinguishing characteristics and functions independently from the central metatarsals (Fig. 28.1). It articulates with the cuboid and lateral base of the fourth metatarsal with strong ligamentous attachments. The base of the fifth metatarsal has insertion points for three muscles [3]. The peroneus brevis attaches on the dorsal aspect of the tubercle, while the peroneus tertius attaches on the dorsal aspect at the proximal metaphyseal-diaphyseal junction. The abductor digiti quinti makes up the third muscular attachment. The lateral band of the plantar fascia also attaches to the planter aspect of the tubercle.

The posterior tibial, lateral plantar, and dorsalis pedis arteries contribute to the arterial supply to the forefoot. At the proximal aspect of the first inter-metatarsal space, the dorsalis pedis crosses the Lisfranc joint and dives between the first and second metatarsals to form the plantar arterial arch. A single nutrient artery enters each of the metatarsals from the medial cortex at the junction of the proximal and middle third of the diaphysis to supply the metatarsal shafts. The metatarsal heads are supplied by dorsal and

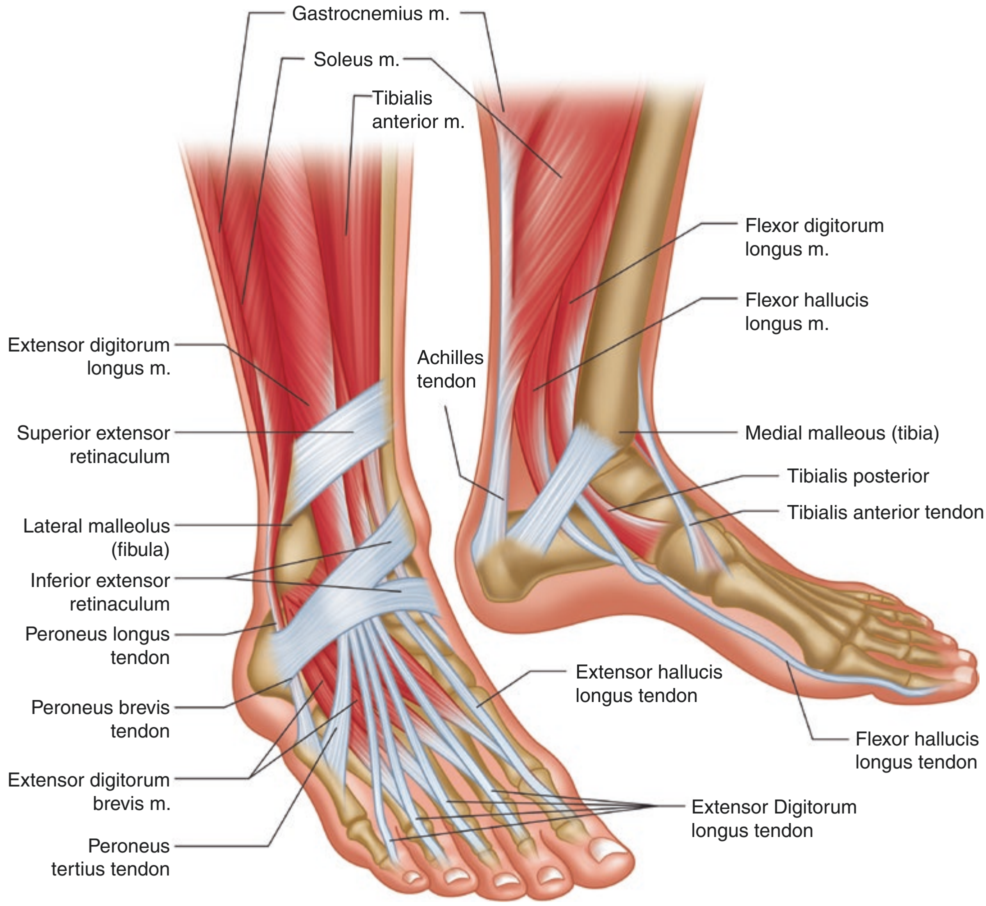


Fig. 28.2 Muscle and tendon anatomy of the foot

plantar metatarsal arteries, which form a vascular ring [6]. Epiphyseal and metaphyseal arteries supply the base of the first through fourth metatarsal. In the fifth metatarsal, there are three possible sources of blood supply: the nutrient artery, the metaphyseal perforators, and the periosteal arteries [7]. An area between the supply of the nutrient artery and the metaphyseal perforators, at the metaphyseal-diaphyseal junction, represents a watershed region which is a site of poor healing [8].

Fractures

Mechanism of Injury in Sports

Common mechanisms of acute metatarsal fractures, in order of frequency, include twisting force with a static forefoot, excessive axial loading or fall from height, direct blow to the foot, and crush injury [2, 3, 9, 10]. Since the first metatarsal is relatively larger and stronger, the most commonly affected metatarsals are the second, third, and fourth. Ballet dancers can sustain a fracture to the distal fifth metatarsal shaft by rolling onto it while raised up on their toes or by landing on it after a jump. This fracture is known as a “dancer's fracture” [9]. Fifth metatarsal fractures are a unique subset of forefoot injuries and their treatment is frequently debated. The fifth metatarsal bone has the widest range of motion of all the metatarsals, except for its base. A sudden inversion mechanism with subsequent pull of the lateral band of the plantar aponeurosis can cause a styloid avulsion (zone I) fracture (Figs. 28.3 and 28.4). These injuries are often missed because their cause of injury is the same as a lateral ankle sprain and their areas of tenderness overlap. True Jones fractures (zone II) are caused by adduction of the forefoot and tensile stress along the lateral border of the metatarsal resulting in fracture at the proximal metaphyseal-diaphyseal junction (Fig. 28.5) [11, 12]. The injury can occur when an athlete lands on the side of their foot in a fall or sustains a direct blow to the area. Zone III fractures, or proximal diaphyseal fractures in the proximal 1.5 cm of the shaft, are rare (Fig. 28.6). However, it usually occurs in athletes due to repetitive microtrauma at the lateral cortex from tensile forces causing a stress fracture. They are commonly lumped together with Jones fractures incorrectly. Repetitive pivoting is a common cause and it is most often seen in basketball players. Like other stress fractures, it is due to chronic overload on the bone or change

Fig. 28.3 Radiographic classification (zones I, II, III) of proximal fifth metatarsal fractures

in routine causing greater stress to the bone. Cavovarus foot deformities also increase stress in the lateral column and predispose to zone II and zone III fractures [13]. Studies in professional football players have also shown that shoes narrower than the foot width may predispose to zone II and III fractures. The fifth metatarsal hangs over the sole of the shoe, thus increasing overload and pressure [14].

Epidemiology

Metatarsal fractures account for 35% of all adult foot fractures and occur most often in patients between 20 and 50 years of age [10, 15]. Sports are involved in the mechanism of injury in 7.9% of metatarsal fracture cases [16]. Overall, in the athletic population, the fifth metatarsal is the most commonly fractured followed by the third and second (Figs. 28.7, 28.8, 28.9, and 28.10). In multiple metatarsal fractures (Figs. 28.11 and 28.12), the third metatarsal is often associated with concurrent fractures of both the second and the fourth, with studies showing that 63% are associated with either one, and 28% are associated with both second

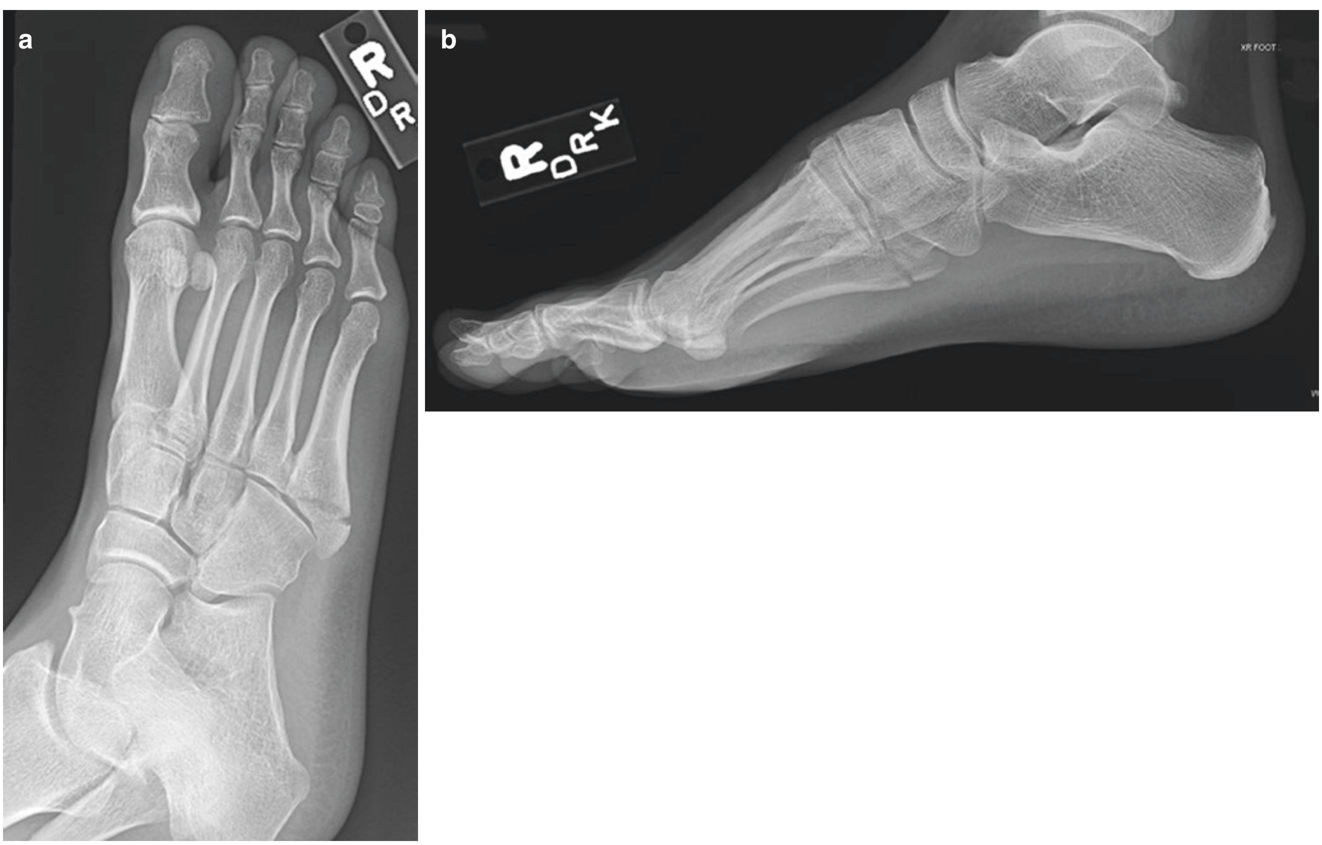


Fig. 28.4 A zone I proximal fifth metatarsal fracture in a 35-year-old male as a result of a mountain bike injury (**a**, **b**)

Fig. 28.5 A zone II proximal fifth metatarsal fracture in a 60-year-old female (**a**, **b**)

and fourth metatarsal fractures [15]. First metatarsal fractures are rare in sports (Figs. 28.13 and 28.14). Studies have shown that of patients who present with metatarsal fractures as a result of a sports injury, soccer is the most commonly associated sport, although fifth metatarsal fractures overall are not common representing 0.5% of injuries in this population [17, 18]. Fractures of the fifth metatarsal account for 68% of all metatarsal fractures [15]. The frequency of each zone has been shown to be zone I> zone II> zone III [15, 16]. In order of frequency, these are commonly encountered in basketball, American football, soccer, baseball, jogging, gymnastics, lacrosse, field hockey, and volleyball [18, 19].

Classification

Fractures of the metatarsal bones are relatively common in both adults and children [2, 20]. The diaphysis (shaft) is the most common location for metatarsal fractures followed by the distal end (Figs. 28.7, 28.8, 28.9, 28.10, and 28.11). There are several ways to classify metatarsal fractures. As with many classification systems, they do not always define treatment nor predict outcome. When separated by anatomic region of the metatarsal, these fractures are subdivided into distal (metatarsal head and subcapital), midshaft, and proximal (base) fractures [2]. Classification should include location, fracture pattern, displacement, angulation, and articular involvement. The Orthopaedic Trauma Association (OTA) classification system describes the fracture pattern of each bone but does not give insight into overall stability or treatment [2, 11]. Its classification is similar to the metacarpals of the hand. It is subdivided into metatarsal, proximal or distal, nonarticular, partial articular, or complete articular and diaphyseal non-comminuted, wedge comminution, or comminuted. The Arbeitsgemeinschaft für Osteosynthesefragen AO classification system divides the fractures between extra-articular (type A), intra-articular (type B), fracture-dislocations (type C), and pure dislocations such as the floating metatarsal (type D) [2, 11]. Fractures of the proximal fifth metatarsal have been classified based on their anatomic description after differences in healing were noted related to the fracture location in relationship to the peroneus brevis tendon [21]. Dameron, and later Quill, classified fifth metatarsal fractures into the proximal tuberosity avulsion fracture (zone I), metaphyseal-diaphyseal junction fracture extending into the fourth-fifth

Fig. 28.6 A zone III proximal fifth metatarsal fracture in a 63-year-old female as a result of a fall

Fig. 28.7 A comminuted and mildly displaced diaphyseal fracture of the fifth metatarsal fracture in a 25-year-old male

inter-metatarsal articulation (zone II), and proximal diaphysis fracture (zone III) [21, 22]. Table 28.1 summarizes these different categories.

The commonly misused term "Jones fracture," first described by Sir Robert Jones in 1902 and later defined by Stewart, is specific to zone II injuries which involves the vascular watershed area [23, 24]. It is a transverse fracture approximately 18 mm from the base of the fifth metatarsal at the meta-diaphyseal junction [25].

The radiographic classification of proximal fifth metatarsal fractures (Fig. 28.3) is as follows: fracture on the lateral aspect of the tuberosity extending proximally into the metatarsocuboid joint (type I), Jones fracture beginning laterally in the distal part of the tuberosity and extending obliquely and proximally into the medial cortex at the fourth and fifth metatarsal base articulation (type II), and fracture distal to the fourth and fifth metatarsal base articulation (type III) [26].

Clinical Presentation

The diagnosis of most metatarsal fractures is fairly straightforward when a thorough history is obtained, a careful phys-

Fig. 28.8 A spiral fracture of the fifth metatarsal diaphysis in a 47-year-old female (**a**, **b**)

ical examination is performed, and radiographic evaluation is acquired. In acute metatarsal fractures, patients typically present with pain that is exacerbated by weight-bearing. Swelling and ecchymosis can also be associated with these fractures and may develop quickly. The pain may initially start out as localized, but as the forefoot continues to swell, localization may become more difficult. Swelling can also become severe enough to produce compartment syndrome of the foot. This risk is greatest in first metatarsal fractures or multiple metatarsal fractures [17]. Pain and swelling out of proportion to radiographic findings, small avulsion injuries at the base of the second metatarsal, as well as plantar ecchymosis should prompt suspicion of a Lisfranc joint injury [1, 4, 11].

Deformity can accompany severely displaced fractures, and it is important to thoroughly examine the skin for signs of tenting and assess neurovascular status. Applying an axial load by compressing the metatarsal head toward the calcaneus can help distinguish soft tissue injury from a fracture. Direct palpation may elicit point tenderness in both injuries, but axial loading generally should not produce significant pain in a soft tissue injury.

Diagnosis

If the mechanism of injury is consistent with an inversion ankle sprain, the Ottawa foot rules can be used to determine the need for imaging of the foot. If athletes have tenderness at the base of the fifth metatarsal or at the navicular bone or inability to bear weight immediately or at the time of the evaluation, then foot plain radiography should be obtained [20]. Other mechanisms or clinical concerns should prompt radiographic evaluation to assess the metatarsals for fractures and any associated shortening, deviation, angulations, or displacement [27]. Three standard x-ray views of the foot in the AP, oblique, and lateral projection should be obtained. The radiographs should include the whole foot to rule out any other potential injuries, but sometimes a second set of radiographs focusing only on the forefoot or a modified lateral view with the metatarsals rotated slightly may be necessary for better visualization [20]. These radiographs should be weight-bearing if possible. This allows for a better assessment of the alignment of the fractures as well as the overall alignment of the foot. The fracture should be visualized on at least two views. The lateral view can be helpful in demonstrating

Fig. 28.9 An intra-articular fracture (arrows) of the fifth metatarsal head in a 27-year-old male as a result of a snowboarding injury (**a**, **b**)

dorsal or plantar displacement of the fracture fragments. It is important to evaluate for apex dorsal displacement because the displaced metatarsal head can cause significant disability if its position is not treated properly [11]. Contralateral foot views or, if tolerated, stress or weight-bearing radiographs can be obtained for further evaluation.

In the office and especially in the sideline setting which often has limited resources, musculoskeletal ultrasound can provide radiation-free imaging that could be accurate, sensitive, specific, and comparable to traditional radiography [1, 28, 29]. In addition, musculoskeletal ultrasound is cheap and portable and provides real-time dynamic imaging that can be used to augment the sideline evaluation. Few studies have addressed the role of adjunctive imaging in metatarsal injuries. CT scan can be considered if there is concern for articular surface congruity, and MRI may provide additional information related to suspected soft tissue injuries, osteochondral injuries, or ligamentous disruption, but it is not usually necessary.

Initial Management

Before initiating treatment, the provider should assess neurovascular status and skin viability and be vigilant for signs of compartment syndrome. Most acute metatarsal fractures are treated conservatively with elevation, icing, analgesia, and immobilization. Initial immobilization can be obtained with a posterior splint, postoperative shoe, or fracture boot [1, 2]. Table 28.2 summarizes the management of first through

Fig. 28.10 A comminuted fracture of the second metatarsal diaphysis in a 63-year-old female

fourth metatarsal fractures. Patients should be instructed to use pain as a guide for weight-bearing, apply ice, elevate the foot, and use analgesics as needed.

Fifth metatarsal fractures are treated based on the zone of injury. Most zone I fractures can be treated similarly to other metatarsal fractures [29–33]. Zone II fractures have traditionally been treated with initial non-weight-bearing; however more recent studies have questioned the necessity of this treatment [34]. Zone III fractures are usually treated surgically (Fig. 28.15). Table 28.3 summarizes the management of proximal fifth metatarsal fractures.

Fig. 28.11 Distal (neck) fractures of the second, third, and fourth metatarsals (arrows) in a 28-year-old male

Indications for Orthopedic Referral

In the acute setting, if the metatarsal fracture is an open injury or associated with any neurovascular compromise, the athlete should be transferred immediately to a hospital with adequate resources for urgent treatment. The provider should also be on alert that a closed injury may be converted to an open fracture if skin necrosis and sloughing ensues days after the initial injury. In general, if the skin appears in jeopardy or if there was a significant crush injury, the provider should strongly consider early referral for consultation.

Anatomic positioning of the first metatarsal is important because it supports one third of the weight-bearing forces of the forefoot. Any displacement of the first metatarsal through the joint or fracture site represents instability and should strongly be considered for fixation [2, 3, 20]. Therefore, patients with even mildly displaced fractures, >3 to 4 mm or >10° of angulation of the first metatarsal, should be considered for referral as these fractures may require closed reduction or operative management [2, 3, 20]. Patients with multiple or displaced proximal fractures of the first to fourth metatarsals should also be considered for referral [35]. These injuries also may be associated with dislocations of the Lisfranc joint [11]. Reduction with anatomic alignment is essential in these fracture-dislocations. Zone II injuries may be considered for operative treatment in athletes, although this aggressive treatment is debatable [31–33]. Other indications for referral include displaced fractures that occur very close to the head, any fracture-dislocations, and intra-articular fractures.

Fig. 28.12 An intra-articular proximal second metatarsal (open arrows) and extra-articular fractures of the third and fourth metatarsal base (arrows) in a 22-year-old male (**a**, **b**)

Follow-Up Care

After acute injury management, follow-up should generally occur within 3–7 days [1, 20]. This allows for reduction of soft tissue swelling. Radiographs should be obtained at the first follow-up visit to assure fracture stability. If fracture position is lost during follow-up, referral to orthopedic surgery is recommended. In general, if the fracture is stable, the patient can be transitioned to a short-leg walking cast or boot. In non-displaced or minimally displaced fractures, the patient can be treated symptomatically in a firm-soled shoe or a postoperative shoe instead of the cast or boot if able to tolerate the pain [1–3, 20, 25]. If a cast is used, it may be discontinued 2–3 weeks after injury depending on athlete's symptoms. Once the cast is removed, the patient should be placed in a firm, well-padded shoe and begin progressive ankle range of motion exercises along with calf stretching and strengthening exercises. Evidence has shown that early mobilization while progressing with weight-bearing as tolerated results in improved function scores compared to immobilization with a cast, with no difference in healing or pain scores [30]. This functionally based treatment in the sports setting allows for a jumpstart in the rehabilitative process without risking the outcome. For zone II fractures, a short-leg non-weight-bearing cast may be applied. Although these fractures have a high rate of union without surgery, this treatment requires an extensive period of

Fig. 28.13 A transverse diaphyseal fracture of the first metatarsal in a 45-year-old male as a result of a dropped heavy object

immobilization. There is debate whether or not weight-bearing should be permitted with most experts recommending non-weight-bearing. However, studies allowing weight-bearing have not shown significant difference in outcomes [34]. The patient should be immobilized for 6–8 weeks and then evaluated out of the cast. If there is no sign of symptomatic or radiographic healing at that time, a referral should be made to an orthopedic surgeon. Average total immobilization time for zone II fractures has been shown to take about 6–10 weeks, and total healing time is typically 12 weeks in these fractures [1]. Physical therapy may be beneficial for range of motion and strengthening after prolonged immobilization.

Repeat follow-up visits should occur every 2–4 weeks, with repeat radiographs obtained at 4–6 weeks to assess healing. Adequate healing is attained if at 6 weeks there is evidence of callus formation on radiographs and the patient no longer has point tenderness. This time is generally longer for zone II fractures. Immobilization can be discontinued once there is clinical and radiographic evidence of fracture healing.

Return to Sports

For conservatively treated fractures, early range of motion and ankle/foot strengthening exercises can be started as soon as pain allows. Cross-training activities are reasonable as tolerated. As with all injuries, return to sports should be a progression of loading toward sport-specific demands while respecting fracture healing, and in most cases can occur between 4 and 8 weeks.

For Jones fractures, after the period of immobilization, a patient may gradually resume activities once both clinical and evidence of radiographic healing have occurred [31–33, 36].

Patients with uncomplicated metatarsal fractures may be expected to return to their pre-injury level of sporting activities once healing has occurred [37].

Complications

Complications may occur if more severe injuries go unrecognized. This includes arterial injury and compartment syndrome, which may lead to loss of the foot or ischemic contractures. Open fractures can lead to osteomyelitis. Failure to recognize more complicated injuries of the tarsometatarsal (TMT) joint, or Lisfranc injuries, can lead to chronic pain with weight-bearing and subluxation of the metatarsals [11]. General complications include malunion, nonunion, and posttraumatic arthrosis of the TMT or MTP joints. Malunion of the first metatarsal is tolerated poorly. It can result in significant morbidity due to the essential role of the first metatarsal as the preferred ray for loading during weight-bearing [2, 3]. Excessive weight-bearing of the lesser metatarsal heads can occur when the first metatarsal is short or not in plantigrade position. These positions lead to increase loading through the lesser metatarsal and MTP joints and can lead to MTP synovitis and rupture of the plantar plate or collateral ligament.

Fig. 28.14 A comminuted diaphyseal fracture of the first metatarsal in a 51-year-old male as a result of a dropped heavy object (**a**, **b**)

Table 28.1 Proximal fifth metatarsal fracture zone classification [11, 22]

Proximal fifth metatarsal fracture zone classification	Anatomy	Mechanism of injury
Zone I	Proximal cancellous tuberosity including the insertion of the peroneus brevis tendon and the calcaneometatarsal ligamentous branch of the plantar fascia and includes the articular surface of the fifth metatarsocuboid joint	Result of an acute episode of forefoot supination with plantar flexion resulting in avulsion-type injuries from a pull from the lateral band of the plantar fascia and peroneus brevis
Zone II	Distal tuberosity with fractures usually extending into the fourth and fifth metatarsal articulation	Result from an acute injury with possible mechanism being a large adduction force to the forefoot with the ankle plantarflexed
Zone III	Begins distal to the fourth and fifth ligamentous structures and extends distally 1.5 cm into the tubular portion of the diaphysis	Result from a fatigue injury to normal bone has been under repeated stress

Table 28.2 Management guidelines for metatarsal fractures (first through fourth) [1–3, 20, 25]

Fracture category	Proximal	Shaft/distal
Initial immobilization	Posterior splint or short-leg walking boot (SLWB)	Posterior splint or SLWB
Patient instruction	Partial weight-bearing as tolerated (as pain allows, low levels of pain 1–3/10 acceptable)	Non-displaced: weight-bearing as tolerated
		Displaced/reduced: partial weight-bearing as tolerated
Follow-up	3–7 days	3–7 days
Long-term immobilization type	Non-displaced without evidence of ligament disruption: SLWC or SLWB	Non-displaced: SLWB, or firm-soled shoe (postoperative sandal)
		Reduced: SLWC or SLWB
Length of immobilization	Partial weight-bearing 4–6 weeks, followed by 2–4 weeks weight-bearing in SLWC or SLWB	Non-displaced: 4–6 weeks
		Reduced: 2–3 weeks partial weight-bearing and then 3–4 weeks with full weight-bearing as tolerated
Indications for referral	Most proximal metatarsal fractures should be considered for referral especially if more than one involved	Fractures with dorsal or plantar angulation >10°, >3–4 mm displacement, open fractures, multiple fractures, unable to reduce, intra-articular fractures, vascular injury, compartment syndrome, fracture-dislocation, high risk for skin necrosis
	Lisfranc joint injuries	
Healing time	6–10 weeks	Non-displaced: 6 weeks
		Reduced: 6–8 weeks
Return to sports	Will vary based on specific injury and degree of pain. If prolonged immobilization, return to sports is delayed, with initial focus on ROM and strengthening as tolerated	Can begin ankle range of motion and calf stretching/strengthening immediately. Once pain-free in SLWC or SLWB or post-op shoe can ride a stationary bike, then at 6–8 weeks can begin running and progress as tolerated

Fig. 28.15 Surgical fixation of a zone III proximal fifth metatarsal fracture with a partially threaded intramedullary screw (**a**, **b**)

Table 28.3 Management guidelines for proximal fifth metatarsal fractures [16, 29–33]

Fracture category	Styloid avulsion (zone I)	Metaphyseal-diaphyseal junction (Jones fracture) (zone II)	Diaphyseal stress (zone III)
Initial immobilization	Firm-soled shoe	Posterior splint with ankle at 90	Posterior splint with ankle at 90
Follow-up	4–7 days	3–5 days	3–5 days
Patient instruction	Weight-bearing as tolerated	Non-weight-bearing (some studies have questioned) [34]	Non-weight-bearing
Long-term immobilization type	Firm-soled shoe, consider SLWC if very symptomatic	SLNWBC ankle at 90	SLNWBC ankle at 90
Length of immobilization	2 weeks	6–10 weeks	Up to 20 weeks
Indications for referral to ortho	Displaced >3 mm and/or comminution	Displaced Nonunion at 3 months Athletes	All cases, especially Torg types II and III Nonunion at 3–4 months Recurrence
Healing time	4–8 weeks	6–12 weeks	Up to 20 weeks
Return to sports	Advance activity as tolerated once pain-free	Slow integration into sports, usually 3–6 months before full return	Slow integration into sports, usually 3–6 months before full return

Pediatric Considerations

Metatarsal fractures account to about 60% of all pediatric foot fractures (Figs. 28.16, 28.17, 28.18, 28.19, 28.20, 28.21, 28.22, and 28.23) [37]. Children have an ability to remodel fractures very well with greater correction potential. A short-leg cast may be a preferred choice of treatment for children based on age, offering greater protection and control of symptoms. Children are able to tolerate up to 20° of dorsal or plantar angulation, but they should be referred for fractures with greater deformity [35]. Other indications for referral are any displaced physeal fractures, as well as Salter-Harris types III, IV, and V fractures. This is because children may be at risk for future growth abnormalities if the physis is injured.

There is a high incidence of unrecognized fractures in younger children, particularly of the first metatarsal due to the subtle nature of the fracture (Figs. 28.21, 28.22, and 28.23) [38]. The physis of the first metatarsal in children can also be confused with a fracture because it is located proximally rather than distally like in the other metatarsals. Radiographic findings should be made with clinical correlation. If there is tenderness, it is safe to treat as though there is a fracture and repeat radiographs in 10–14 days to identify callus. Buckle fractures are also not uncommon and may present with little swelling, so once again, point tenderness can aid in diagnosis.

Children and adolescents are also at risk for osteonecrosis of the metatarsal head known as Freiberg's disease or infraction. This commonly affects the second metatarsal but can affect the third or fourth metatarsal. Children and adolescents will present with localized pain that is exacerbated by weight-bearing. Clinically this condition can be mistaken for an acute facture. However, on radiographs, there is sclerosis and partial collapse of the metatarsal head and will often appear more squared. Nonoperative treatment may include short-leg walking cast or boot with transition to stiff inserts and metatarsal pads with improvement. Children and adolescents should be referred to an orthopedic surgeon if this condition is suspected and nonoperative treatment has failed [39].

In children, avulsions of the styloid are the most common fractures of the proximal fifth metatarsal (Fig. 28.16) like in adults. It is important that these injuries are not misdiagnosed for the normal apophysis that appears during late childhood and early adolescence (Fig. 28.16). The apophysis at the base of the fifth metatarsal is visible between 9 and 11 years in girls and between 11 and 14 years in boys [21]. The normal apophysis acts as a growth center for a tendon insertion. On normal radiographs, the apophysis lies parallel to the long axis of the metatarsal, while a fracture is almost always transverse. If a child experiences an acute injury and has tenderness over the apophysis, it suggests avulsion of the apophysis. Contralateral views can be obtained for comparison but generally do not change approach. Children may also present with apophysitis, which often occurs when there is tenderness but normal radiographic findings and no history of acute injury. This is treated with NSAIDs and rest. Styloid avulsion fractures are treated similar in children as in adults.

Lisfranc Injuries

Mechanism of Injury in Sports

The Lisfranc joint is a joint between the tarsal bones and metatarsal bases that is essential for proper foot function and athletic performance. Unlike the first metatarsal, the other metatarsals are tethered to another, so that when the second metatarsal is injured at the Lisfranc joint, the third through fifth metatarsals are also affected. High-energy Lisfranc injury typically occurs after major trauma (direct blow and

crush injuries) with associated proximal metatarsal fractures (Fig. 28.24) [40, 41]. However, sports-related Lisfranc injuries are usually caused by more subtle low-energy injuries with little to no displacement of the Lisfranc joint such as landing forward on a plantarflexed foot [40–42].

Epidemiology

Lisfranc injury is relatively common among athletes for midfoot sprains. It occurs in 4% of collegiate American football players every year with about one third of whom are offensive linemen [43]. It can happen to football players when another player lands on the plantarflexed and externally rotated foot, further increasing the forces applied to the proximal metatarsals.

Classification

Nunley and Vertullo introduced a classification system for subtle Lisfranc injuries based on clinical examination, diastasis, and loss of arch height on weight-bearing radiographs (Table 28.4) [44].

Tarsometatarsal fracture-dislocations are commonly referred to as Lisfranc fracture-dislocations. These are less common injuries than the purely ligamentous injuries and sports is an uncommon cause [19, 45].

Fig. 28.16 Inversion foot injury in a 12-year-old boy as a result of an American football injury (**a**). Plain radiography (**b**–**d**) shows a zone I fracture (arrows) of the proximal fifth metatarsal. Normal apophysis (open arrows) should not be confused with a fracture as it is usually parallel to the diaphysis

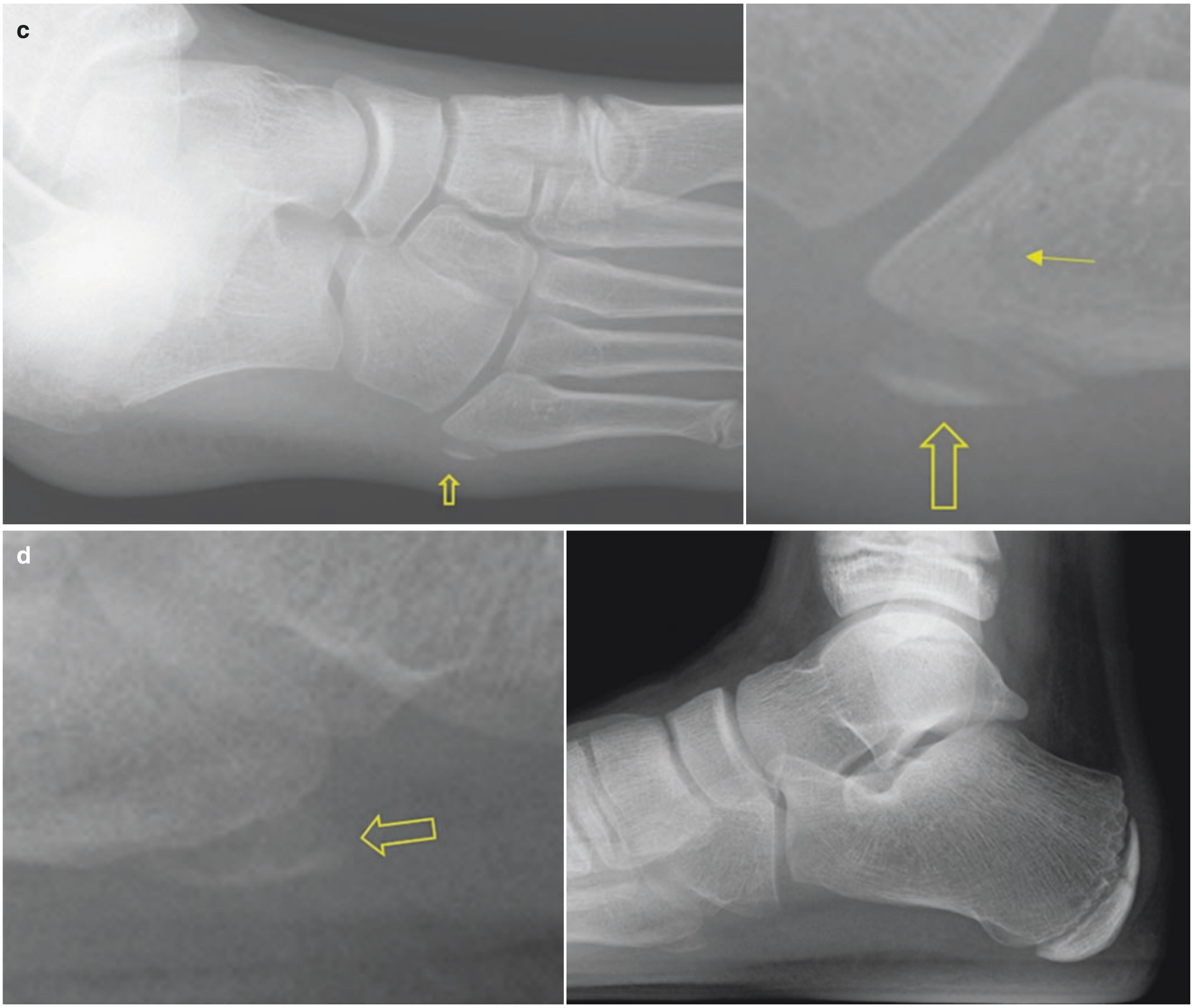


Fig. 28.16 (continued)

Clinical Presentation

Athletes present with severe swelling and pain and are usually unable to bear weight. Due to the interconnectedness of the Lisfranc joint, tenderness can be diffuse. The athlete will have pain and difficulty with push-off or heel raise [4, 31, 42, 46]. In tarsometatarsal fracture-dislocations, a deformity is sometimes appreciated [14, 47]. Plantar ecchymosis can occur but not until days later. On physical examination, there is pain with manipulation of the tarsometatarsal (TMT) joint with first ray hypermobility.

Diagnosis

Careful evaluation for Lisfranc joint injury should be made in the setting of proximal fractures of the first through fourth metatarsal, especially when multiple fractures are present [11, 42]. A standard foot radiographic series (AP, lateral, and oblique views) should be obtained in addition to AP weight-bearing views of both the injured and unaffected foot, which would stress the TMT joint and pick up any widening between the first and second metatarsals or arch collapse.

Lisfranc joint injuries should be suspected when there is an avulsion or fleck fracture at the lateral base of the first metatarsal or the medial edge of the second metatarsal or widening between the first and second metatarsal space (Fig. 28.25). The medial edge of the second metatarsal should align with the medial edge of the middle cuneiform, the medial edge of the third metatarsal should align with the medial edge of the third cuneiform, and the fourth metatarsal base should be in line with the medial edge of the cuboid. It is advised to obtain further advanced imaging in the case of multiple metatarsal base fractures to rule out a Lisfranc

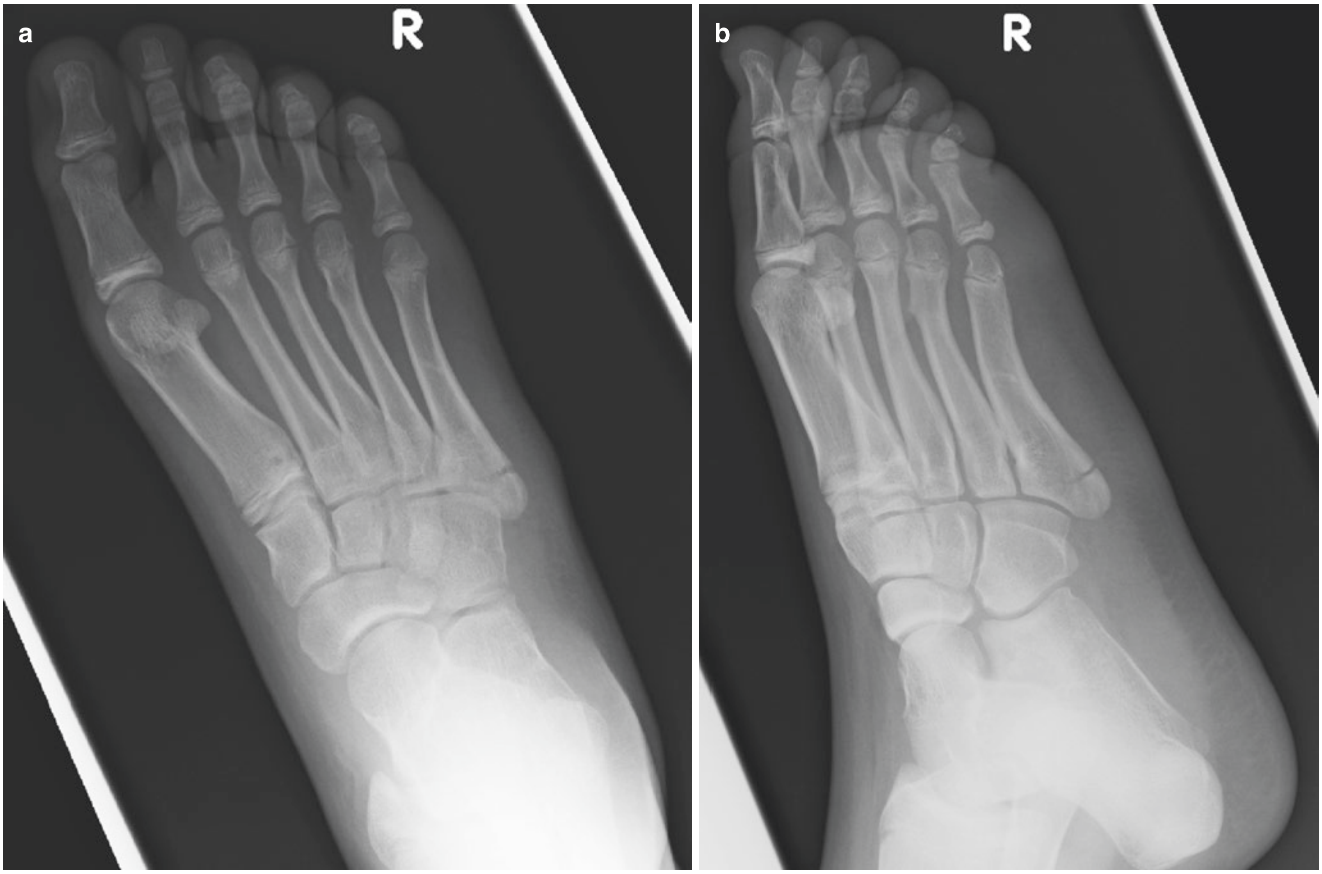


Fig. 28.17 Zone I fracture of the fifth metatarsal base in an 11-year-old girl as a result of twisting her foot going downstairs (**a**, **b**)

fracture-dislocation if unclear on plain radiography [4, 11, 42]. A CT scan can be helpful in the setting of multiple fractures. If radiographs appear normal, but there is a high clinical index of suspicion, MRI is the imaging of choice.

Initial Management

Initial management includes typical symptomatic treatment with ice, elevation, and pain management. Some authors suggest no weight-bearing, while others suggest protected weight-bearing as tolerated [4, 14]. A short-leg posterior splint or CAM walker boot can provide comfort in the first days. Athletes with fracture-dislocations should be referred within hours for further evaluation and treatment [14].

Indications for Orthopedic Referral

All Lisfranc injuries should be considered for referral. Stage 1 injuries can be considered for conservative treatment and based on provider experience may not need referral. There are no studies comparing different treatment protocols, and usual treatment is partial protected weight-bearing with a cast or boot, with progression to full weight-bearing as pain allows. Supportive inserts may be considered. Unstable injuries, fracture-dislocations, or conservatively treated injuries with persistent pain and disability should be referred for surgical consultation. Both timing of intervention and surgical procedure are debated and currently left to the discretion of the operating surgeon. Surgical procedure is usually screw fixation with some techniques now using suture alone or combined with screw fixation [4, 14].

Follow-Up Care

Conservatively treated injuries should be followed for improvement in pain and swelling and progression from initial protected weight-bearing. Ankle and foot range of motion exercise can be started early with transition to strengthening exercises as pain allows. The timing of follow-up care will depend on the athlete, injury, and provider preference.

Return to Sports

Depending on the severity of the injury, return to sports can take weeks to months [42]. With a mild sprain of the Lisfranc ligament, recovery is much quicker, and athlete can

Fig. 28.18 Displaced diaphyseal fractures of the second and third metatarsals in a 17-year-old male as a result of a fall

Fig. 28.19 Fractures of the proximal second, third, and fourth metatarsals (arrows) in a 7-year-old girl

Fig. 28.20 Fractures of the distal (neck) third and fourth metatarsals (arrows) in a 7-year-old girl

be treated with protected weight-bearing in a cast or boot taking 8–16 weeks to recover [4, 42, 44, 46]. In a fracture-dislocation of the Lisfranc joint, the athlete has to be non-weight-bearing for a prolonged period of time with return to sports sometimes taking up to 12 months. In athletes where there is documented instability, as defined by the presence of diastasis or subluxation, surgery is often recommended to aid in a more rapid recovery [4, 42, 44, 46].

Complications

Complications of proximal metatarsal and Lisfranc injuries are common and sometimes may be related to delayed recognitions of the injury. When this occurs, the arch is at higher risk of collapse due to loss of stability provided by the Lisfranc joint. Posttraumatic arthritis and persistent pain are common. Acute complications include skin necrosis, vascular injury, and compartment syndrome [4, 42, 44, 46].

Pediatric Consideration

Lisfranc injuries in pediatric athletes are rare and are most commonly seen in boys. Fall from height is the most com-

Fig. 28.21 A Salter-Harris II fracture (arrows) of the proximal first metatarsal in a 9-year-old boy as a result of a tree strike while sledding

mon mechanism [48]. In general, management of the pediatric Lisfranc injuries is similar to the adults' injuries. Non-displaced injuries, with or without fractures, can be treated with initial non-weight-bearing splint for 7–10 days and transition to a non-weight-bearing cast for additional 4 weeks. Transition to protected full weight-bearing can occur over another few weeks [48]. Injuries with joint widening or fracture-dislocation should be protected and referred for surgical evaluation.

Metatarsophalangeal Joint Dislocations

Dislocations of the MTP joints are relatively rare injuries with an unknown incidence in sporting activity but have been described in motor vehicle accidents [49]. The most common mechanisms for this injury include stubbing the toe or axial loading through the toes [49, 50]. The first toe is the most commonly reported MTP joint to dislocate likely due to its anatomic position. However, dislocations of the lesser toes have been reported. Case reports of multiple MTP joint dislocations have also been described [50]. First MTP joint dislocations are usually dorsal, and classification description is based on ligamentous integrity and presence or absence of a sesamoid

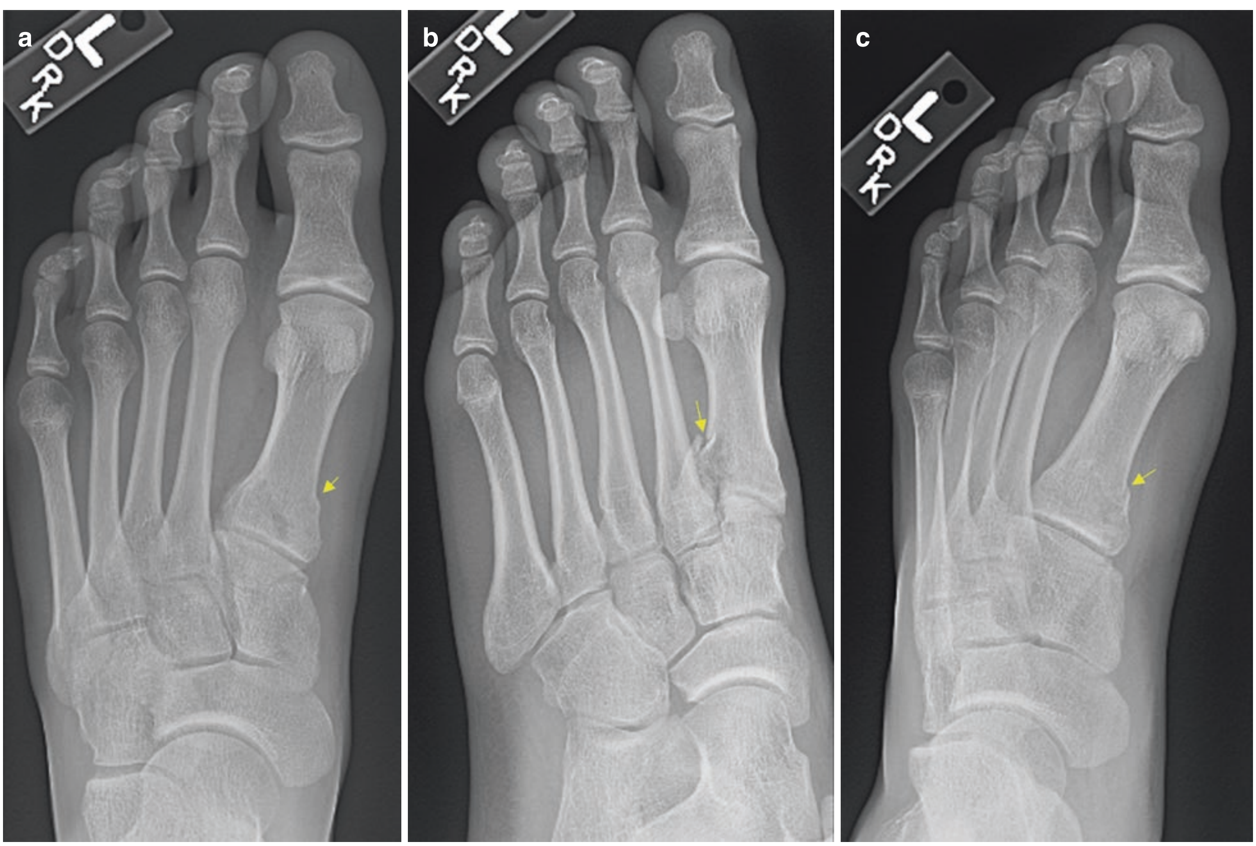


Fig. 28.22 An intra-articular proximal first metatarsal fracture (arrows) in a 15-year-old boy as a result of a mountain bike injury (**a–c**)

Fig. 28.23 Salter-Harris II fractures (arrows) of the proximal first metatarsal and the distal second metatarsal in a 10-year-old boy (**a**, **b**)

Fig. 28.24 A significant injury in a 29-year-old male as a result of a tree dropped on his foot (**a**, **b**). Displaced comminuted fracture with significant shortening of the second metatarsal head due to proximal fracture fragment retraction. Joint space widening of the second MTP joint. Lateral subluxation of the second metatarsal base relative to the intermediate cuneiform, compatible with Lisfranc ligament injury. Mildly displaced third metatarsal head comminuted fracture with mild apex lateral and mild third MTP joint space narrowing. Lateral dislocation of the third metatarsal relative to the lateral cuneiform. Dislocation of the fourth metatarsal relative to the cuboid, with increased joint space laterally. Comminuted fracture of the cuboid. Medial subluxation/dislocation of the medial cuneiform relative to the navicular. A fracture of the medial navicular. Significant soft tissue swelling

fracture. Based on Jahss classification, in type I, the intersesamoid ligament is intact and there is no sesamoid fracture [51]. In type II, the intersesamoid ligament is ruptured with (type IIb) or without (type IIA) the sesamoid fracture [51].

Table 28.4 Lisfranc injury classification [44]

Lisfranc injury classification	Description
Stage 1	Midfoot/Lisfranc sprain with no diastasis or loss of arch height
Stage 2	Associated with 2–5 mm of diastasis between the first and second metatarsal without arch height collapse
Stage 3	Greater than 5-mm diastasis and collapse of the arch height

Lateral and plantar dislocations may also occur but are even more rare [52, 53]. No specific classification system is used to describe lesser toe MTP joint dislocations. Closed reduction of these injuries can be attempted with stabilization of the ankle and midfoot and axial traction, adding dorsiflexion as needed, on the proximal phalanx [50]. Anesthesia may be required but no studies address local versus regional or the use of procedural sedation and will depend on the individual injury and provider's comfort level. Fractures may be irreducible and require open reduction. Treatment of these injuries is based upon expert opinion. Most first MTP joint injuries require surgical stabilization of a ruptured sesamoid ligament or to facilitate reduction [53]. However, some injuries may be treated without surgical stabilization if ligamentous structures are intact

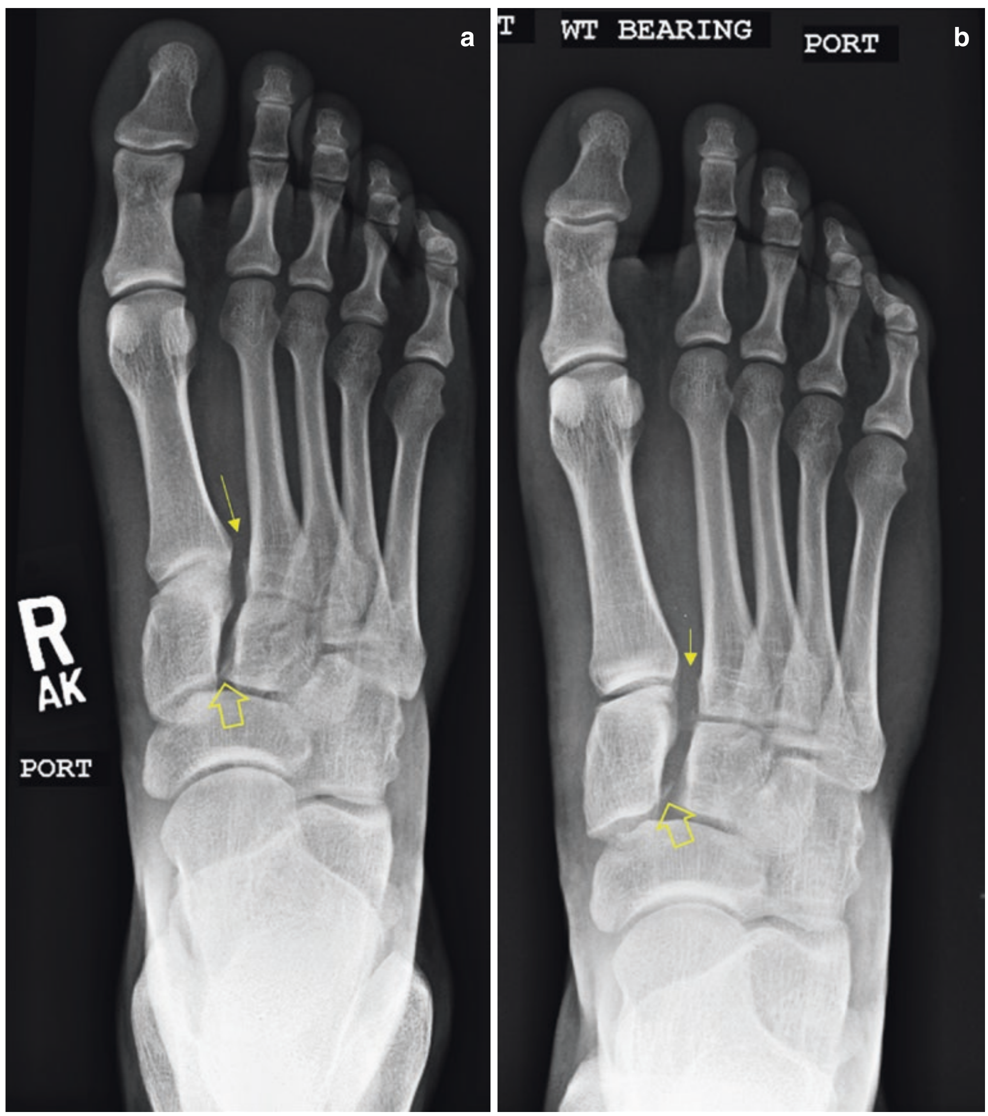


Fig. 28.25 Lisfranc injury (arrows) in a 19-year-old male as a result of a mountain bike injury (**a**). Weight-bearing radiograph reveals widening of the Lisfranc complex (**b**). There is also an avulsion fracture of the medial cuneiform (open arrows)

[53]. Lesser toe injuries may be treated without surgery if reduction is obtained. Most treatment protocols suggest protection for 2–3 weeks with a posterior splint extending past the toes with non- or partial weight-bearing and transition to weight-bearing over the next subsequent 2–3 weeks for both surgical and conservatively treated injuries. Residual stiffness, posttraumatic arthritis, and occasionally instability are potential complications. Return to sporting activity is based upon soft tissue healing and return to normal strength and range of motion with limited pain, but no specific timeframes or guidelines exist.

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Toe

29

Bryant Walrod

Key Points

- Most toe fractures come from direct trauma.
- Toe fractures can be treated conservatively in most cases.
- Intra-articular fractures or fractures with significant displacement may require closed or open reduction.
- Return to play is based on comfort of athlete in most cases.

Introduction

Toe injuries are relatively common in sports. Toe fractures are the most common fractures of the foot and represent approximately 8–9% of all fractures [1, 2]. Fracture of the lateral four toes is approximately four times as common as fracture of the great toe [3]. However, the hallux has a more significant role in terms of balance and weight bearing. During the final stage of foot contact during ambulation, 40% of the body weight is imposed on the toes [4]. The great toe bears an estimated 70% of the forces applied to the toes. Therefore, the morbidity associated with fractures of the great toe is potentially higher compared to that of the lesser toes although studies have shown similar outcomes [5]. The vast majority of toe fractures result from either axial loading, for example, stubbing one's toe, or from a crush injury, such as dropping a heavy object directly on the toe [6]. Less commonly, hyperextension or hyperflexion of the toe may lead to an avulsion injury or a spiral fracture [7]. Blunt trauma is likely to result in a closed toe fracture. In contrast, sharp trauma, for instance, from a blade or serrated device, is more prone to result in open toe fractures.

Toe fractures are frequently comminuted, especially when the distal phalanx is involved. They often demonstrate intra-articular involvement and it is not uncommon to fracture multiple toes concurrently. Most fractures show minimal to no evidence of displacement and can be managed nonoperatively by a patient's primary care physician. In a study of 339 toe fractures, 95% showed less than 2 mm of displacement, and all were managed conservatively with good outcomes [5].

Anatomy

The foot is comprised of numerous soft and hard tissue structures, which collectively provide the means for balance, weight-distribution, propulsion, and ambulation [4]. Key components to the effective execution of these functions are the toes. Each foot possesses five toes, with each toe consisting of a set of phalanx bones or, collectively, phalanges [8]. In structure, the phalanx is comprised of a base proximally, a shaft (body), and a head distally. The proximal phalanx of each toe is connected to the foot by means of articulation with a metatarsal bone, forming the metatarsophalangeal (MTP) joint. The individual phalanges of each toe articulate with one another in flexion and extension via interphalangeal joints. These joints are surrounded and supported by a joint capsule. The first toe, the hallux (also commonly referred to as the "great toe" or "big toe"), contains two phalanx bones, a proximal phalanx and a distal phalanx. The remaining four toes each possess a proximal, middle, and distal phalanx, with the proximal and middle phalanges forming a proximal interphalangeal joint (PIP joint) and the middle and distal phalanges forming a distal interphalangeal joint (DIP joint). On occasion, the middle and distal phalanges may be congenitally fused, resulting in only two phalanges [6]. This most commonly occurs in the

B. Walrod (✉)
Department of Family Medicine, The Ohio State University, Wexner Medical Center, Columbus, OH, USA
e-mail: Bryant.walrod@osumc.edu

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fifth toe, less commonly in the fourth toe, and more rarely yet in the third toe. This condition, a common variant of the norm, is referred to as symphalangism [9]. Additionally, the first metatarsal bone typically has two accompanying sesamoid bones located beneath its distal head and adjacent to the first MTP joint [6, 10]. These sesamoids are small, pea-shaped bones. They are embedded in a capsular ligamentous complex and within the flexor hallucis brevis tendon, one located lateral to and the other medial to the distal aspect of the first metatarsal. Common variants include a single sesamoid bone, as well as multipartite sesamoid bones. A bipartite sesamoid is present in 10% of individuals, and 25% of people with bipartite sesamoid have the condition bilaterally [10]. Additional sesamoid bones are rarely present in other flexor tendons beneath the distal heads of the lateral four metatarsals.

Fractures

Mechanism of Injury in Sports

Toe fractures in sports occur with similar mechanism to non-sport-related injuries. These include blunt trauma such as getting the foot stepped on by an opponent or direct impact such as kicking an opponent or equipment (e.g., goalpost) [15]. Hyperflexion or extension injuries may also occur and are more likely to cause avulsion or spiral-type injuries where blunt trauma is more likely to create transverse or intra-articular injuries [5, 6]. The mechanism of injury for a sesamoid fracture is often a direct blow when jumping or hyperextension and the medial sesamoid most commonly affected [10, 12].

Epidemiology

Despite their frequency, representing approximately 10% of fractures, there is little data on the incidence with many reviews combining foot and ankle injuries and several reviews reporting no injuries from sporting activity [16–18]. In two reviews of soccer players, toe fractures accounted for approximately 5% of injuries in both elite player and in injuries seen in the emergency room [19, 20]. In recreation motorsports, ATC (all terrain cycle) and motorbike, toe fractures represented approximately 2% of extremity fractures [21]. In childhood fractures of the great toe, 28% were associated with sporting facilities, with soccer being the most common. Salter-Harris II injuries of the proximal phalanx were the most frequent fracture type in children [15]. Fractures in gymnastics, taekwondo, basketball, American football, running, and ballet have also been reported [15, 22–24].

Classification

Fractures of the toes are commonly divided into fractures of the great toe, lesser toes, and sesamoids. This is done from a practical standpoint, as fractures of the lesser toes do not generally require treatment beyond symptomatic care, while fractures of the great toe may require longer periods of protection and immobilization or surgical intervention in some cases [7, 10–12]. Sesamoid fractures also may require closer follow-up and longer return to play [10]. Classification of toe fractures is based on the morphology of the fracture (e.g., transverse and oblique) along with other descriptive characteristics such as intra- or extra-articular comminution and displacement (Figs. 29.1, 29.2, 29.3, 29.4, and 29.5). The Arbeitsgemeinschaft fur osteosynthesefragen (AO) classification is most commonly used for descriptive purposes and uses the above parameters but does not guide clinical treatment [13].

Clinical Presentation

Following injury, most patients initially experience severe pain, which may improve over a period of hours to days into more

Fig. 29.1 Intra-articular fracture of the distal fifth proximal phalanx in a 16-year-old male as a result of a mountain bike injury

Fig. 29.2 Comminuted fracture of the fourth proximal phalanx

of a dull, throbbing sensation. They generally demonstrate pain with weight bearing or inability to bear weight on the foot of the affected toe. A fractured toe will likely demonstrate evidence of significant swelling, particularly when compared to the same digit on the uninjured foot [3, 7]. They also may present with ecchymosis and distal injuries may be associated with a subungual hematoma (Fig. 29.6) [3]. Increased tenderness with axial loading, as well as passive and active range of motion, is

Fig. 29.4 Oblique fracture of the second proximal phalanx

Fig. 29.3 Fracture of the base of the first through third proximal phalanxes with intra-articular involvement

Fig. 29.5 Avulsion fracture of the proximal second middle phalanx in a 16-year-old male as a result of a mountain bike injury

Fig. 29.6 Ecchymosis with toes trauma

revealed on palpation of the fractured toe. Crepitation is also commonly recognized. Despite these findings, the fracture site in a closed fracture is difficult to localize as movement and palpation typically elicit diffuse tenderness. Conversely, open and displaced toe fractures can generally be characterized quite easily. Rotation, angulation, and shortening of the toe coincide with most displaced fractures. Outside of severe displacement or a dissecting injury, damage to an artery or nerve is rare with an isolated toe fracture. Sesamoid injuries will typically present a history of injury to the first MTP joint, either direct or indirect [12]. Patients will typically complain of pain with ambulation and with palpation. Pain is noted with palpation of the affected sesamoid, and the pain is worse with passive extension of the great toe and with resisted flexion of the great toe. Patients will also often complain of increased pain when asked to go up on their toes. There may also be associated ecchymosis, edema, hematoma, or crepitus with palpation.

A thorough examination for a suspected fracture begins with inspection [2]. Visualization of the surrounding skin for open wounds is a good place to start in order to grossly distinguish between an open and a closed fracture. While ecchymosis and subungual hematoma are common associated findings, careful evaluation and documentation of skin appearance is crucial as skin sloughing and necrosis can develop following an intense crush injury. This could allow for transition of a closed fracture to an open fracture with subsequent skin breakdown [3, 25]. The examiner should also assess for rotational deformity of the toe by comparing nail bed alignment to that of the uninjured toe on the contralateral foot [7]. Assessment of the integrity of the nail and nail plate is necessary as laceration of the nail plate may be accompanied by a fracture of the distal phalanx [25]. Neurovascular status, including sensation to light touch and capillary refill, should be documented.

Diagnosis

Radiographic imaging is typically necessary to diagnose, characterize, and/or distinguish a suspected fracture from other injuries to the toe. The differential diagnosis includes ligament sprain, contusion, dislocation, tendon injury, and additional soft tissue abnormalities. A dedicated set of three x-rays consisting of AP, lateral, and oblique views are traditionally obtained [2, 7, 10]. Overlying shadows frequently present on the lateral view can make adequate visualization a challenge, and as such, the oblique film can be more useful in the diagnosis. Given that toe fractures are regularly comminuted, can involve the intra-articular space, and periodically exhibit involvement of multiple toes, proper assessment when ordering and reviewing imaging should allow for visualization of the adjacent toes, as well as the joint both proximal and distal to the suspected fracture site (Fig. 29.7). If sesamoid fracture is suspected, clinicians may also consider 40° medial oblique and lateral oblique to evaluate the medial

Fig. 29.7 Transverse fractures of the third through fifth middle phalanxes

Fig. 29.8 Sesamoid view

and lateral sesamoids, respectively (Fig. 29.8) [10]. It is often helpful to obtain similar views of the contralateral sesamoid bones to help distinguish sesamoid fractures from bipartite sesamoids which are often bilateral [10, 12]. Ultrasound has also been proposed in the evaluation of extremity fractures. Studies looking at foot and ankle injuries have found excellent sensitivity and specificity for metatarsal fractures but have not specifically looked at toe injuries [26, 27]. Studies have shown good negative predictive value with ultrasound, but a positive finding still often requires radiographs for characterization of the fracture [28]. CT scan and MRI are sometimes employed for perioperative planning and evaluation of associated soft tissue injuries or to differentiate acute versus chronic injuries in some cases but are normally not required for management decisions [10, 12].

Initial Management

Lesser Toe Fractures

Most lesser toe fractures do very well with conservative management [3, 5, 7, 10]. Treatment options are based on comfort and range from normal foot wear to buddy taping or postoperative shoes [6, 7, 10]. Buddy taping has been shown to work well, but patients may not be compliant with taping and should be warned about possible skin problems between opposing digits (Fig. 29.9) [29]. Based on the comfort of the healthcare provider, closed reduction of many displaced phalanx fractures can be performed safely in the office. A digital block is most commonly used for anesthesia. Traction can be applied to reduce both angular and rotational deformities in most cases with the occasional need for application of other forces (varus/valgus) [6]. Once satisfactory reduction is obtained, treatment is the same as non-displaced and minimally displaced fractures. Table 29.1 outlines the treatment recommendations of lesser toe fractures.

Great Toe Fractures

Presentation and evaluation of great toe fractures is the same as lesser toe fractures, but great toe fractures often have increased pain and longer recovery [2]. In addition, fractures of the great toe should get special attention because of its role in weight bearing, balance, and locomotion. Therefore, there is a concern for higher morbidity, long-term pain, and disability when comparing greater toe fractures to lesser toe fractures [10, 15]. Despite these considerations, reviews have not shown significant differences in outcomes between great toe and lesser toe fractures [7]. Non-displaced or minimally displaced extra-articular fractures (Fig. 29.10) and non-displaced intra-articular fractures (Fig. 29.11) can be treated similar to lesser toes fractures [2, 3, 6, 10, 12, 15]. Table 29.2 outlines management of great toe fractures.

Sesamoid Fractures

Sesamoid fractures most commonly involve the medial sesamoid with a transverse fracture pattern being the most common pattern [12]. It is very rare to have a fracture of both the medial and lateral sesamoid although case reports have been published [14]. Distinguishing a bipartite hallux sesamoid from a fractured hallux sesamoid can be challenging. Fractures (Fig. 29.13) tend to have sharp or jagged

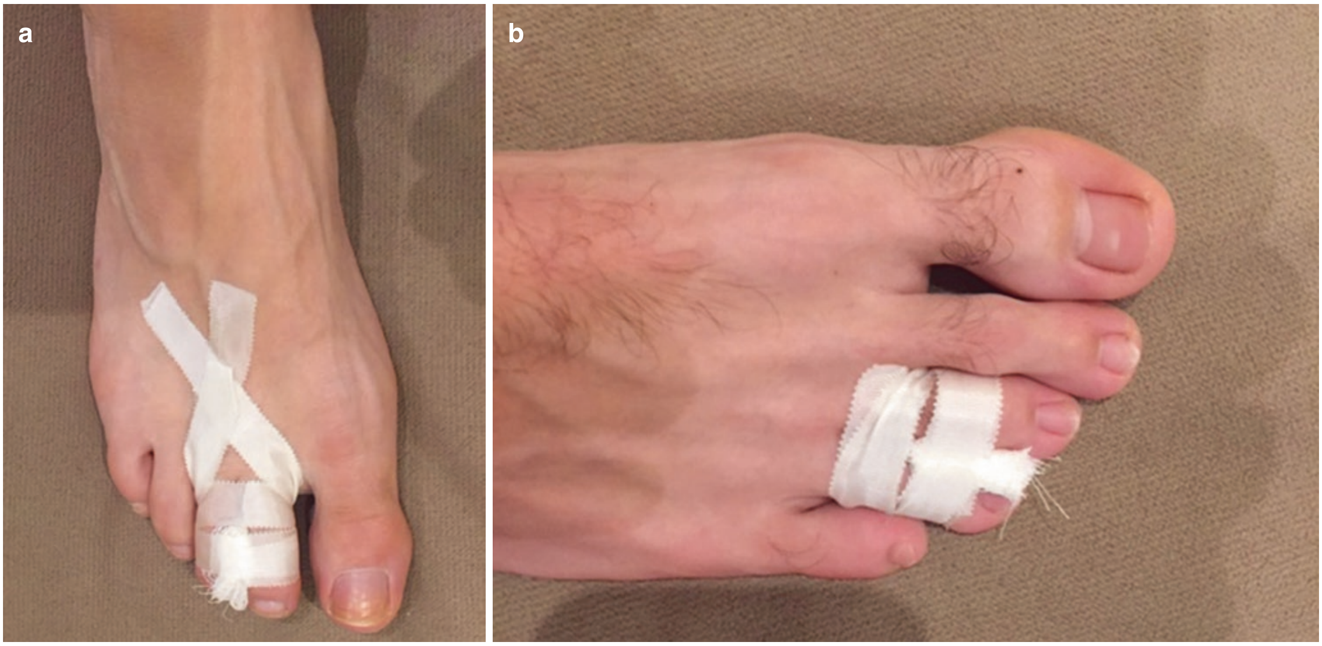

Fig. 29.9 Buddy tapping (**a**, **b**)

Table 29.1 Treatment of lesser toe fractures [6, 7, 10, 29]

Initial immobilization	Buddy tape to adjacent toe (consider a cotton wisp or a folded 2 × 2 to prevent skin maceration)
	Firm soled or post-op shoe especially if there is significant pain with ambulation
Patient instructions	Weight-bearing as tolerated
Follow-up	Consider if significant pain >3 weeks
	Consider repeat radiographs at 1 week for:
	Displaced fractures
	Greater than 25% of the joint surface involved
	The fracture needed to be reduced
Long-term immobilization	Transition to regular shoes as pain allows
	Continue buddy taping as needed
Healing time	3–4 weeks (may have pain for 6–8 weeks)
Indication for referral	Open fracture of the proximal phalanges
	Open fracture of the distal phalanges with gross contamination
	Consider in a poorly controlled diabetic patient or an immunocompromised patient
	Fracture dislocations; displaced intra-articular fractures
	Fractures that do not maintain proper alignment with buddy taping
	Lesser toe fractures with angulation of >20° in the dorsoplantar plane, >10° in the mediolateral plane, or >20° of rotational deformity
	Severe crush injuries
	Overlying skin necrosis
	Vascular compromise
	Additional referral after initial treatment may also be necessary for cases exhibiting delayed complications of osteomyelitis, persistent pain after healing, and malunion. Surgical intervention may be necessary to manage mal-aligned or persistently painful joints
Return to play	As pain allows

Fig. 29.10 Oblique fracture of the proximal phalanx of the first toe

edges without cortication, whereas bipartite sesamoids (Fig. 29.14) have rounded fragments with cortication. Fracture fragments tend to “fit into” each other, while the bipartite sesamoids often do not line up [10]. The medial sesamoid is often larger than lateral sesamoid. In a bipartite sesamoid, the medial sesamoid is often much larger than the lateral sesamoid bone [10, 12]. Sesamoid fractures are treated similar to great toe fractures. Walking boot or walking cast with weight bearing as tolerated (WBAT) for 4–6 weeks based on pain is the typical treatment course [10, 12]. When pain allows transition into a rigid shoe or an orthotic to unload the first ray should be considered. Table 29.3 summarizes the treatment of sesamoid fractures.

Fig. 29.11 A mildly displaced and intra-articular oblique fracture of the distal phalanx of the first toe in a 47-year-old male as a result of a mountain bike injury (**a**, **b**)

Indications for Orthopedic Referral

While primary care physicians can manage the majority of toe fractures, there are specific instances when referral for orthopedic evaluation is warranted. Due to its significant role in weight bearing and stability, far more referrals are necessary for the hallux when compared to the lesser four toes in order to prevent deformity and decreased range of motion which may affect a patient's ability to resume normal activity and functionality [7]. Tables 29.1, 29.2, and 29.3 highlights fractures that should be given consideration for referral based on provider's experience. Referral of sesamoid fractures is indicated for failure of conservative treatment with persistent pain beyond 8 weeks.

Follow-Up Care

Non-displaced lesser toe fractures do not need follow-up in most cases unless pain persist longer than 3–4 weeks [11]. Great toe fractures, displaced and reduced fractures, and sesamoid fractures will usually be followed initially in 1–2 weeks followed by evaluation every 2–4 weeks. However, this is based on provider preference and individual injury.

Return to Sports

There are no evidence-based guidelines for return to play after toe fractures. In most cases athletes may progress back into training and competition as pain allows. For fractures requiring surgical intervention, radiographic evidence of healing may be indicated. Basic requirements include minimal to no pain and normal or near-normal joint motion and strength. Functional evaluation may include normal walking and running gaits.

Complications

When evaluation and treatment are initiated early and prompt referral is placed as indicated for surgical consideration, outcomes are generally favorable [5]. With proper management, the risk of complications can be mitigated, and most patients

Table 29.2 Management of great toe fractures [6, 7, 10, 12, 15]

Initial immobilization	Walking boot or post-op shoe
	Buddy taping
	Crutches if needed
Patient instructions	Weight bearing as tolerated
	If walking boot, remove for range of motion exercises several times a day
Follow-up	Consider repeat x-rays at 1 week if there was intra-articular involvement or if reduction was needed at initial evaluation
	If pain last longer than 6 weeks
Long-term immobilization	Transition to regular shoes as tolerated
	Encourage stiff soled shoes during transition
Healing time	3–6 weeks
Indications for referral	Open fractures
	Intra-articular fractures involving >25% of the joint of the hallux (Fig. 29.12)
	Fractures with >2 mm of displacement
	Consider in a poorly controlled diabetic patient or an immunocompromised patient
	Fracture dislocations, displaced intra-articular fractures
	Severe crush injuries
	Overlying skin necrosis
	Vascular compromise
	Additional referral after initial treatment may also be necessary for cases exhibiting delayed complications of osteomyelitis, persistent pain after healing, and malunion. Surgical intervention may be necessary to manage malaligned or persistently painful joints
Return to play	Based on patient comfort in 4–8 weeks
	Can consider turf toe insert

return to pre-injury functionality. Patients with intra-articular fracture are more prone to develop long-term complications. Degenerative joint disease, chronic pain, angular deformity, and digital edema are long-standing setbacks which can result from these injuries [6]. More acutely, persistent pain, malunion, and nonunion may prevent return to play progression as these can lead to difficulty with ambulation and discomfort wearing shoes. Osteomyelitis remains a significant concern for open fractures and those with extensive crush injuries triggering secondary necrosis.

Pediatric Considerations

With the exception of first toe fractures in older children and correction of rotational deformity in the hallux, very few toe fractures in children require reduction [15, 22, 24]. Natural remodeling of the bone will result in acceptable or near-perfect alignment. Children with displaced fractures of the first toe should be considered for referral [15]. Based on practitioner experience, pediatric fractures involving the physis, with the exception of non-displaced Salter-Harris I or II fractures, should prompt consider-

Fig. 29.12 Comminuted and intra-articular fracture of the proximal phalanx of the first toe

Fig. 29.13 Medial sesamoid fracture

Fig. 29.14 Bipartite sesamoid

Table 29.3 Treatment of sesamoid fractures [10, 12]

Initial immobilization	Walking boot or post-op shoe
	Crutches if needed
Patient instructions	Weight bearing as tolerated
	If walking boot remove for range of motion exercises several times a day
Follow-up	Repeat x-rays may not be needed but can be done at provider discretion
	Consider if pain last longer than 6 weeks
Long-term immobilization	Transition to regular shoes as tolerated
	Encourage stiff soled shoes during transition
	Can use insert with first ray support (turf toe insert)
Healing time	6–8 weeks
Indications for referral	Pain persistent beyond 8 weeks
Return to play	Based on patient comfort
	4–8 weeks
	Can consider turf toe insert

ation of referral to an orthopedic surgeon (Fig. 29.15) [6, 22]. Pediatric toe fractures are generally well-healed in 3 weeks.

Dislocations

Toe dislocations are uncommon in sports. The PIP and DIP joints can sublux or dislocate (Fig. 29.16) as a result of sports injuries. The dislocations are almost always dorsal (Fig. 29.16), but lateral, volar, and complex dislocations can occasionally happen. Some dislocations may be associated with fractures. Toe dislocations can be managed similar to finger dislocations which are discussed in Chap. 20.

Fig. 29.15 Salter-Harris III fracture of the proximal phalanx of the first toe

Other Toe Injuries

Injuries to toes are relatively common. The exact incidence is unknown as most athletes do not seek medical attention. General concepts of soft tissue injuries such as lacerations, sprains, and strains are discussed elsewhere in the book. Toe ligament (e.g., collateral ligaments) injuries are usually mild and can be managed conservatively.

Subungual Hematoma

In the evaluation of a toe injury, there is often a subungual hematoma noted on inspection (Fig. 29.16) [15, 25]. These injuries commonly occur in association with distal phalanx fracture and can increase the pain and discomfort of the injury [3]. Treatment of a subungual hematoma has some variation in practice with a recent trend toward less aggressive management as cosmetic outcomes of trephination alone versus nail removal and nail bed laceration repair have shown similar cosmetic and function outcomes [25, 30].

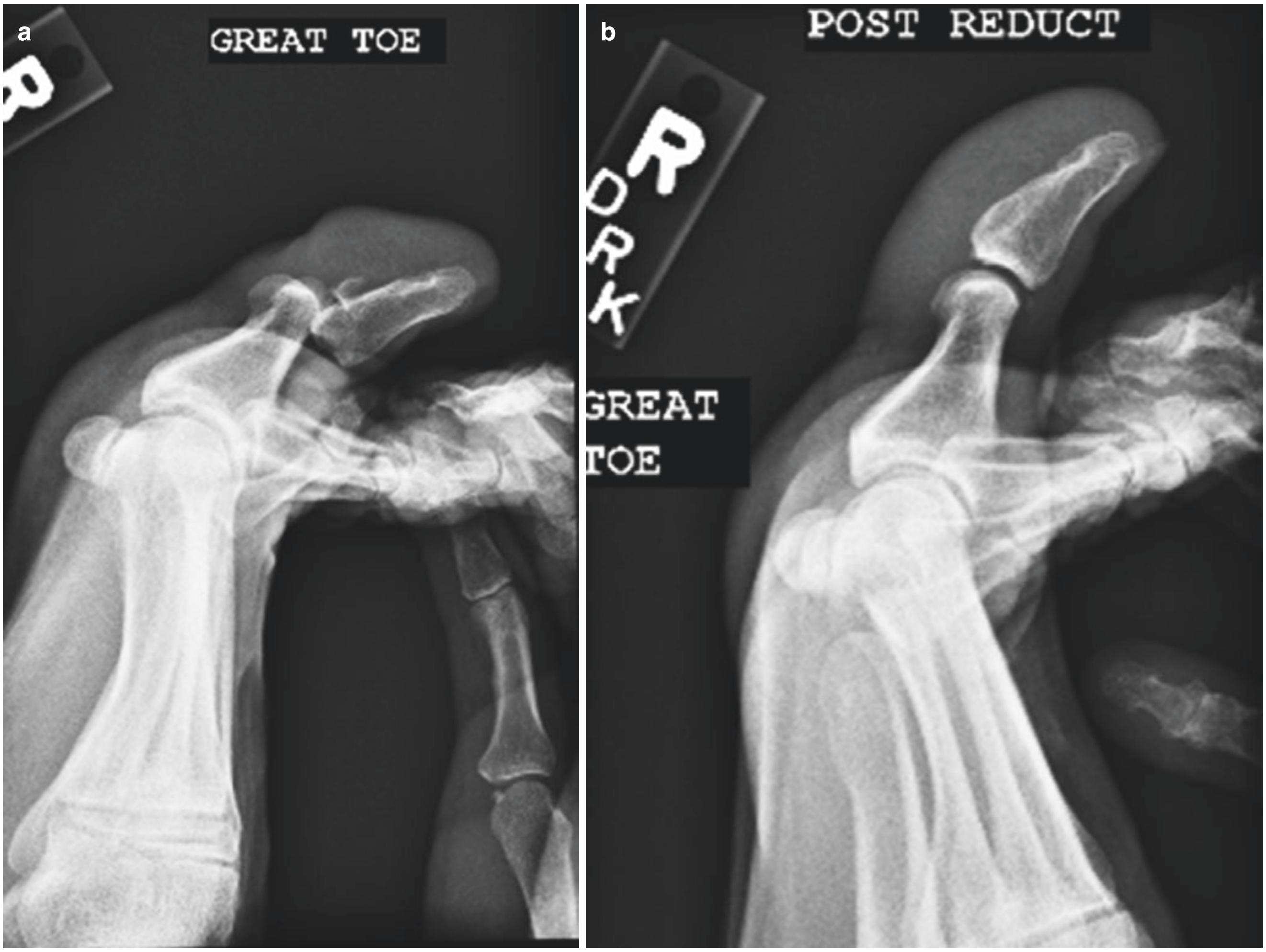

Fig. 29.16 Posterior dislocation of the DIP joint of the first toe in a 55-year-old male as a result of an alpine sliding injury (**a**). Post-reduction radiograph shows anatomical alignment (**b**)

Fig. 29.17 Subungual hematoma

Fig. 29.18 Trephination of a subungual hematoma

Consider trephination of the hematoma if it is within the first few days after injury and for patient comfort. Several techniques have been described in the literature (e.g., electrocautery, 18-gauge needle, heated paper clip), and all appear to be safe and effective (Figs. 29.17 and 29.18) [25, 30].

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Part V

Acute Sports-Related Bones and Joints Trauma: Axial Skeletal

Matthew Gammons

Cervical Spine

30

Matthew G. Zmurko, Matthew Gammons, and Morteza Khodaee

Key Points

- Emergency action plans should include preparation for spinal injuries.
- Coordination with local emergency medical services is paramount as local spinal immobilization protocols will vary.
- Spinal precautions should be taken with all suspected spinal injuries.
- Return to play decision should be made on an individual basis.

Introduction

Although relatively rare, cervical spine injuries account for a large portion of serious injuries in sports [1]. The relative flexibility of the cervical spine in comparison to the thoracic and lumbar regions likely accounts for this difference. The cervical spine can be divided into the upper cervical spine (i.e., craniocervical junction and C1–C2) and the lower cervical spine (C3–C7). Because of anatomic differences, the injury patterns vary between upper and lower cervical spine injuries [2]. Direct axial load to the cervical spine accounts for the largest portion of serious cervical fractures, particularly of the lower spine. Flexion, extension, and rotation may also create serious injury but often result in more soft tissue disruption or avulsion-type fractures [2]. Acute treatment of suspected injuries requires pre-event planning, communication, and spinal immobilization [3]. Subacute treatment and return to play vary based on location and injury and should be individualized.

Anatomy

Differences in the anatomy account for varying injury patterns between the upper cervical spine and the lower spine which has similarities to the upper thoracic spine [2]. The upper cervical spine has many soft tissue attachments but articulates through C1 (atlas) and the occipital condyles. The dens of C2 (axis) articulates with the posterior aspect of the C1. C1–C2 also articulate laterally through synovial joints (Fig. 30.1). The atlantooccipital (AO) joint is generally limited from motion by its osseos articulation where the atlantoaxial (AA) joint relies more on ligamentous support (Fig. 30.2). From the C2–C3 articulation down the lower cervical spine is similar to the thoracic spine, with the spinous process of C7 being the most prominent of the cervical spine and second most prominent overall (T1 is the most prominent).

The lower cervical spine has similar anatomy to the thoracic and lumbar spine but relies more on soft tissue supports, muscles, and ligaments as it lacks other inherent stability. C3–C7 vertebral bones consist of the vertebral body, which is connected to the posterior elements by two pedicles (Fig. 30.3). The posterior elements are made up of the lamina and spinous process. Like the thoracic spine, the ligamentous structures support the anterior and posterior spine as well as an interspinous ligament between the spinous processes (Fig. 30.4). Additionally, C1–C6 vertebrae have transverse foramen to allow passage of the vertebral arteries. Vertebral artery usually passes anterior to the C7 vertebrae to enter the foramen at C6 but will occasionally enter at C5 (Fig. 30.1).

M. G. Zmurko
Vermont Orthopaedic Clinic, Rutland Regional Medical Center, Rutland, VT, USA
e-mail: mzmurko@rrmc.org

M. Gammons
Concussion Clinic, Vermont Orthopaedic Clinic, Killington Medical Clinic, Rutland Regional Medical Center, Rutland, VT, USA
e-mail: mgammons@rrmc.org

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

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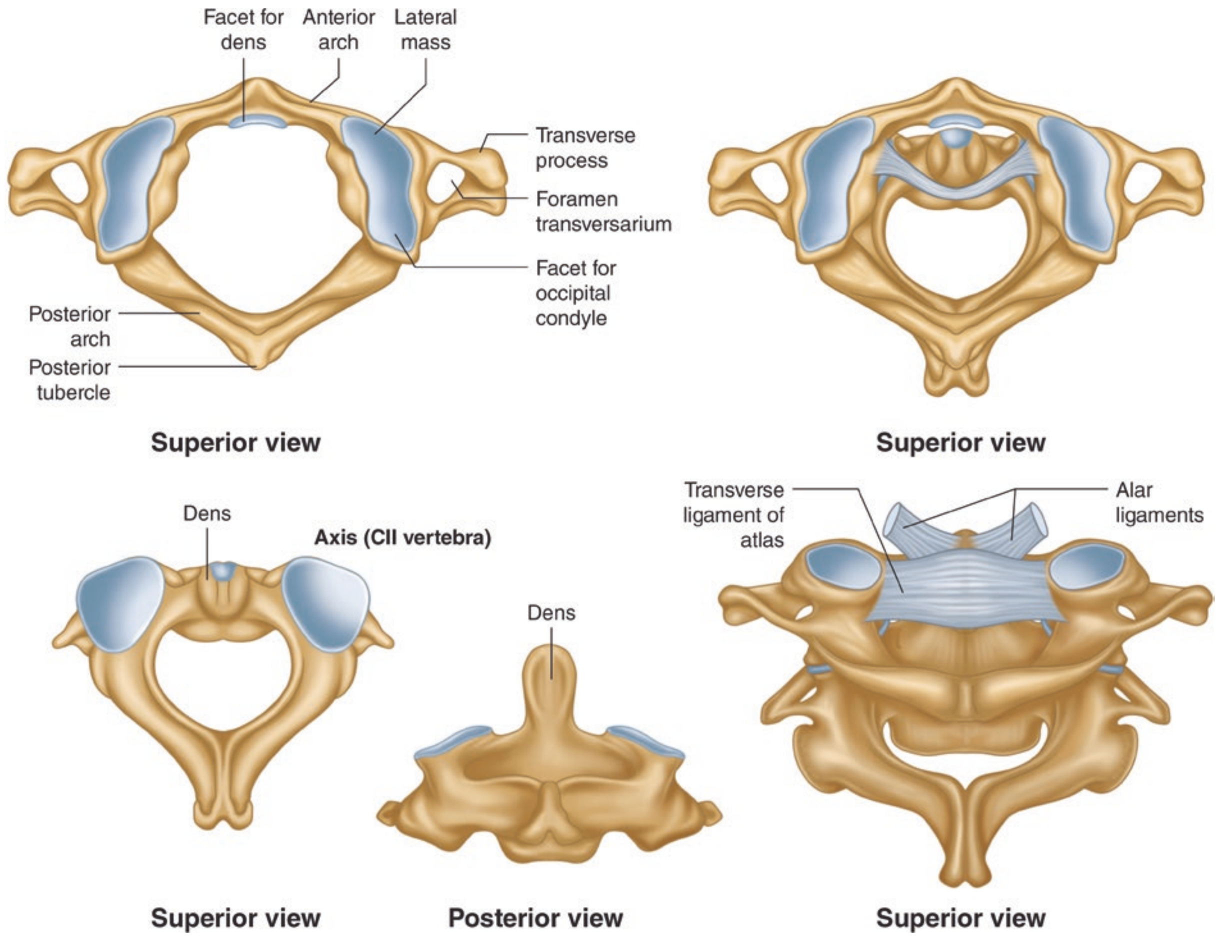


Fig. 30.1 Upper cervical spine bone anatomy (**a**). Schematic articulation in lateral (**b**) and posterior (**c**) views

Fractures

Cervical spine fractures are divided by four main anatomic areas, the occipital condyles, the atlas, the axis, and the lower cervical spine (C3–C7). Each has different classifications and treatments.

Mechanism of Injury in Sport

Overall axial loading mechanisms appear to account for the greatest number of spinal fractures [4]. This may occur when the crown of the head hits another player, an object such as the boards in ice hockey, or the ground and the force of the body compresses the spine. Rotational and distraction mechanisms are less likely given rotational and distraction injuries are less common. Occipital condyle fractures occur in blunt trauma that creates axial loading alone or combined with rotation or lateral bending and have been reported in recreation activities such as sledding and bicycling [5]. Atlas fractures occur with compression and axial loading and have also been reported in recreation activities such as diving (shallow water) and skateboarding. Axis injuries occur with forceful hyperextension or hyperflexion combined with axial loading. Lower cervical spine injuries are generally axial loads or direct impact in the case of spinous process injuries [4, 6].

Epidemiology

Despite their overall rarity, sports activities account nationally for about 10% of severe spinal cord injuries [7]. In 2016, sports accounted for approximately 50% of direct catastrophic reported to the National Center for Catastrophic Sports Injury research [1]. In a study of high school sports, cervical spine injuries occurred at a rate of 3.04 per 100,000 athletic exposures [8]. Fractures account for a small portion (2.6% of overall injuries) but account for 22% of severe injuries [8]. Boys' hockey had the highest proportion of fractures at 20%, followed by boys' basketball, baseball, and wrestling. Although football had the highest rate of overall injury, fracture rates were comparatively low at approximately 3% [8]. Over a 20-year period, cervical spine fractures accounted for 1.7% of fatalities in high school and college American

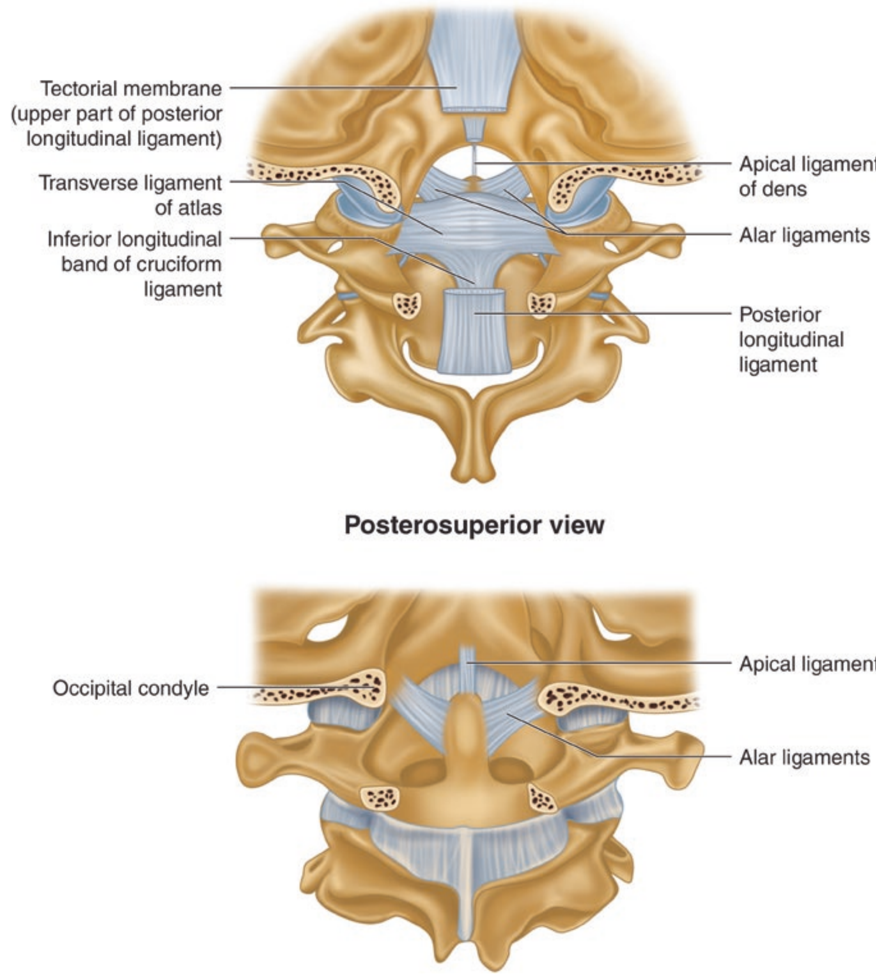


Fig. 30.2 Upper cervical spine ligamentous anatomy (**a**, **b**)

football [9]. Low rates of fracture have been reported in both men's and women's NCAA hockey [10]. In recreational skiing and snowboarding, reported rates of spinal cord injury (fracture rates not reported) are low as well (0.075 for every 1000 skier/snowboarder days) [11].

Upper cervical spine injuries are less common in sports (4.6% of fractures in one study with mountain biking being the most frequently reported cause) [12]. Most axis fractures occur from motor vehicle accidents; however, they have been reported in hockey, football, and diving accidents [13]. The axis was most commonly reported fractured level in a series where 1% of the injuries were sports related but C5–C7 made up the greatest overall number of injuries [14]. Spinous process fractures have been reported in multiple sports [6, 15–19].

Classification

Occipital Condyle

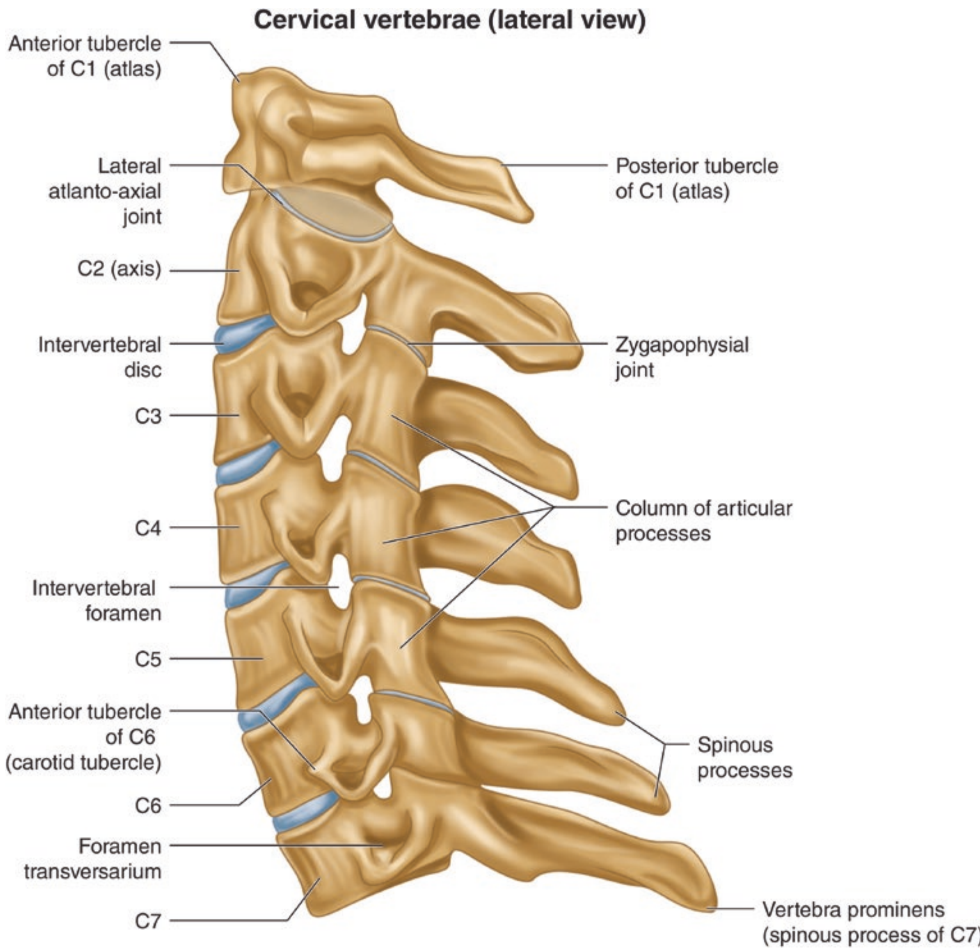
Occipital condyle fractures may be relatively uncommon in blunt trauma patients, with a reported incidence up to 3%, but data on their occurrence in sports is lacking [2, 5, 20]. They have been reported in both adult and pediatric population [21]. They usually result from high-energy blunt trauma and have been increasingly recognized with the use of CT scan for the evaluation of cervical spine trauma. Anderson and Montesano classification system is demonstrated in Fig. 30.5 [22].

Type I injuries are thought to be from axial loading. Types II and III are believed to be rotational and possible lateral bending [2, 20, 22, 23].

Atlas

Fractures of the atlas represent approximately 11% of cervical spine fractures in adults [13]. They are commonly associated with other fractures especially the axis (Fig. 30.6) [24]. Mechanism usually involves direct compression of the skull most commonly resulting in fractures to the anterior and posterior arches. Jefferson's fractures occur when the lateral masses are affected [13, 24, 25]. In addition to assessment of bony injury, it is important to assess the integrity of the transverse ligament in C1 fractures as it provides anterior stability to the C1–C2 joint. Many classification systems have been

Fig. 30.3 Schematic lateral view of the cervical spine bone anatomy



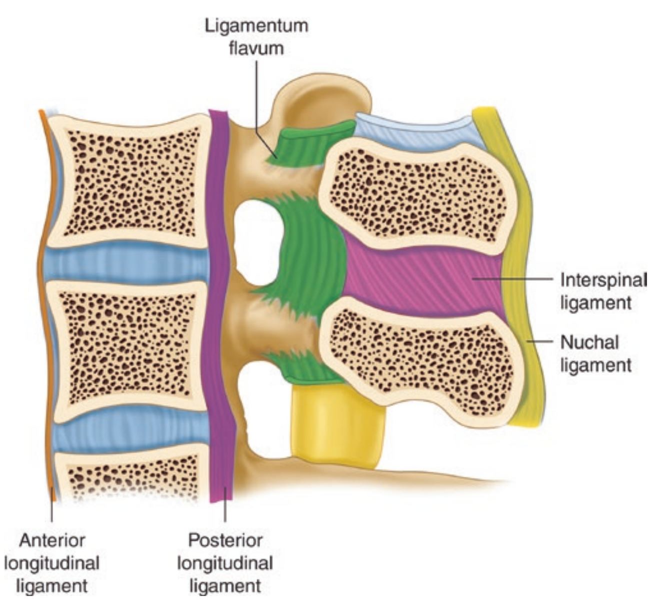


Fig. 30.4 Lower cervical spine (similar to thoracic and lumbar spine) ligamentous anatomy

used, including Jefferson's original description; however, the most clinically relevant system is description of the pattern of fracture (Fig. 30.7) [13, 24, 25].

Posterior arch injuries generally occur with lower-energy mechanisms and are commonly associated with hyperextension. The vertebral arteries have a sulcal groove that likely creates a weak point in the structure. They may also be associated with additional injuries of the upper spine [13, 24, 25].

Burst fractures, also known as Jefferson's fractures, generally occur with high-energy injuries and direct axial loading. These fractures are classically described as having four parts with fracture through both the anterior and posterior arches; however two- and three-part fractures are more commonly seen. Stability depends on the integrity of the transverse atlantal ligament [13, 24, 25]. Anterior arch fractures may be either horizontal, hyperextension injury or vertical, compression injury. They can occur bilaterally in association with Jefferson's fractures [13, 24, 25].

Lateral mass fractures generally occur with distraction forces with asymmetrical compression. These fractures may

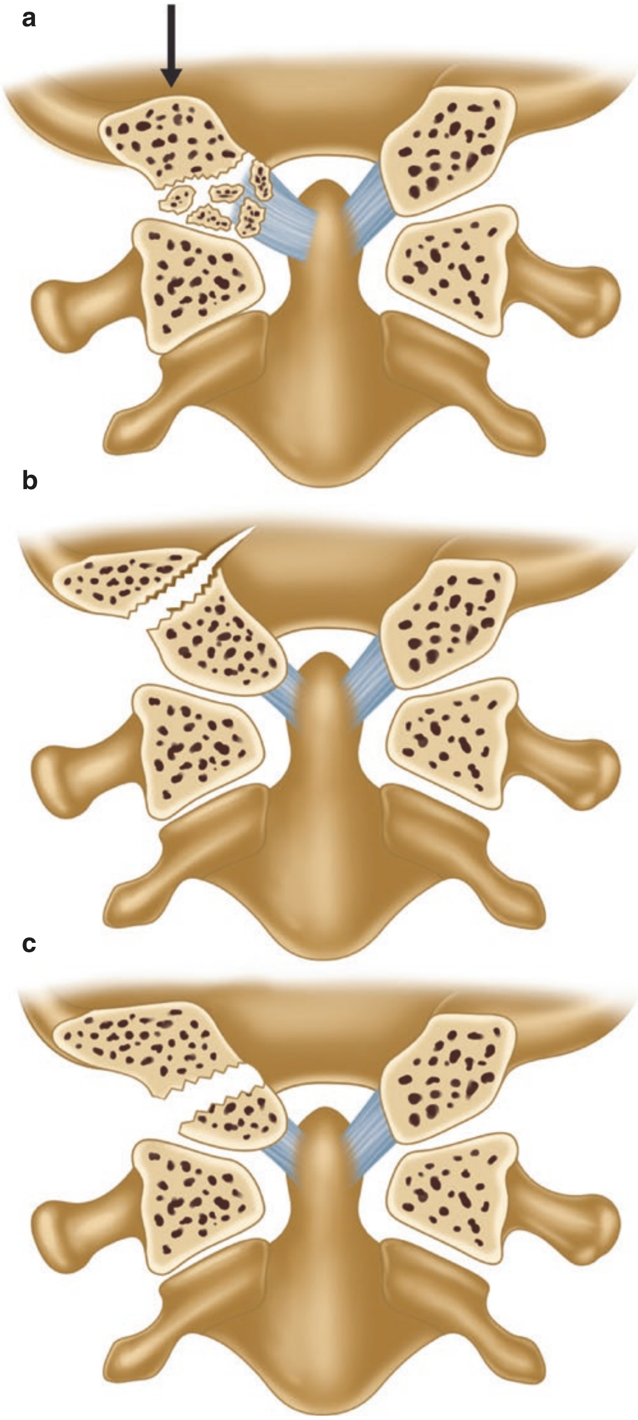


Fig. 30.5 Anderson and Montesano occipital condyle fracture classification. Type I – comminuted without fragment displacement (**a**). Type II – linear fracture (**b**). Type III – avulsion fracture with fragment displacement (**c**)

extend in the transverse process or displace laterally and limit neck motion. Avulsion injuries to the contralateral transverse process may occur with severe lateral flexion injuries. They may also be comminuted involving both compression and flexion forces and be associated with injury to the transverse atlantal ligament. Injuries to the transverse atlantal ligament may also occur with and without bony injury [13, 24, 25].

Axis

Fractures of the axis commonly involve the odontoid process (dens) which represents up to 20% of cervical spine injuries. Fractures are divided into odontoid, traumatic spondylolisthesis (hangman's fracture), and other C2 body fractures [26, 27].

Odontoid Process Fractures

It is believed that odontoid process fractures are created by shear forces, both hyperflexion and hyperextension. Hyperflexion injuries may create anterior displacement (C1–C2 dislocation) and increase the risk of spinal cord injuries. Hyperextension injuries may create posterior displacement but overall carry less risk of spinal cord injury (Fig. 30.6) [26, 27]. The most commonly used classification was described by Anderson and D'Alonzo (Fig. 30.8) [26, 27].

Hangman's Fracture

Haughton first described bilateral axis pars interarticularis fractures (Fig. 30.9) in people who were executed by hanging, hence the commonly referred name [28]. In hanging there is an extension and distraction mechanism, and this differs from modern fractures that are hyperextension and compression injuries seen in motor vehicle accidents and falls. These modern fractures have been termed traumatic spondylolisthesis and tend to be less lethal than the hanging injuries [26]. The Levine-Edwards classification system is most commonly used and is based on the mechanism of injury (Fig. 30.10 and Table 30.1) [29].

Other fractures of the axis body are usually described by the orientation (Figs. 30.11, 30.12, and 30.13) [30]. Type I fractures are oriented in the coronal plane, Type II in the sagittal plane, and Type III in the transverse plane. These fractures are usually non-displaced and rarely involve the spinal cord in the injury [31].

Lower Cervical Spine

The lower cervical spine or subaxial spine (C3–C7) has similar classification to thoracolumbar spine injuries (Fig. 30.14). The Arbeitsgemeinschaft für Osteosynthesefragen (AO) is most commonly cited (Table 30.2) [2, 32].

Similar to the thoracolumbar spine, the AO classification does not determine treatment. The Subaxial Injury Classification (SLIC) is used to better guide treatment options (Table 30.3) [33, 34].

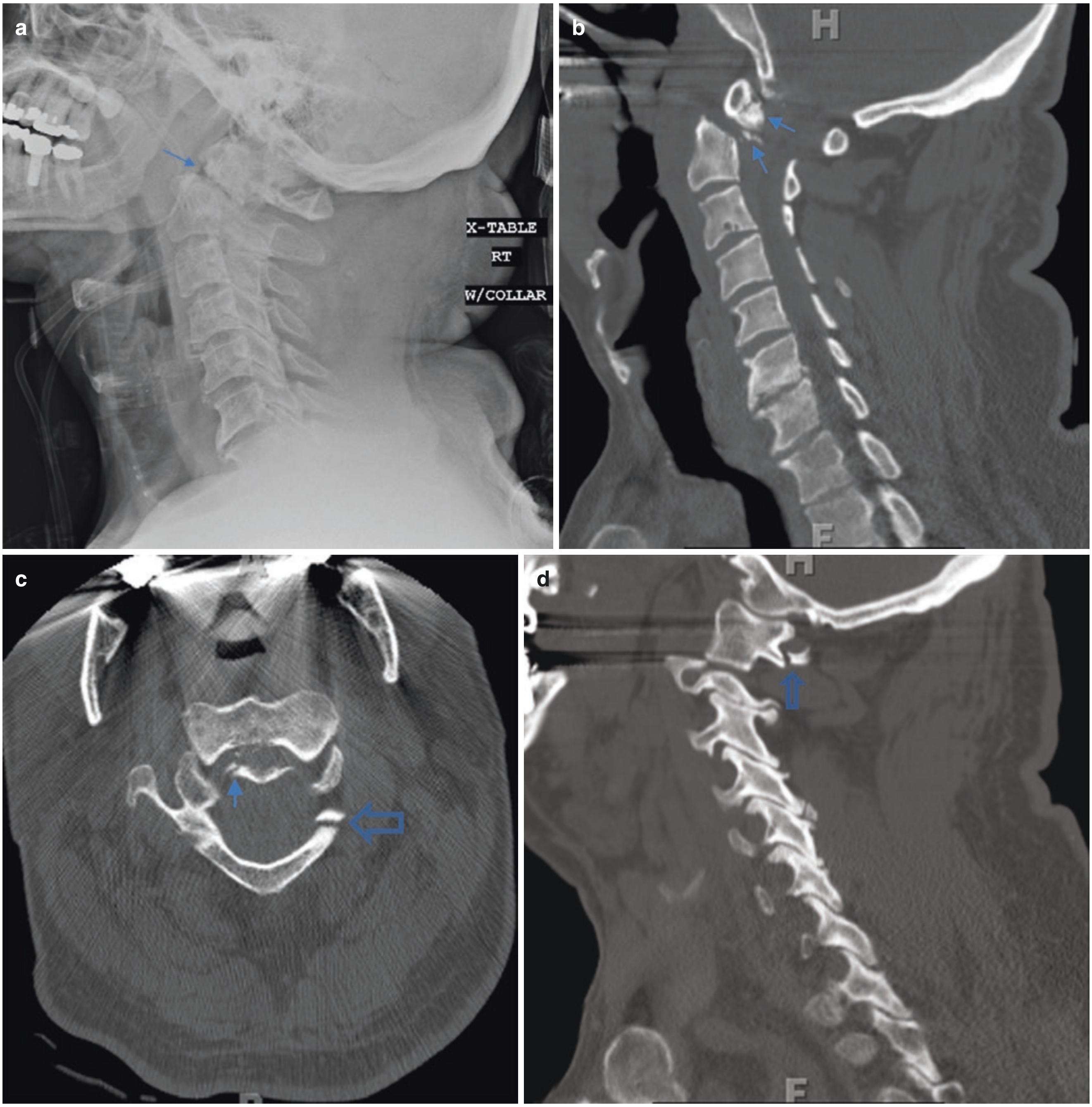


Fig. 30.6 Oblique fracture of the base of the odontoid process with 7 mm posterior displacement (arrows) in a 68-year-old male as a result of a ski injury (**a**–**e**). There are also fractures (arrowheads) through the right and left proximal posterior arch of C1 (**d**–**f**). Postoperative radiographs show near-anatomic alignment (**g**, **h**)

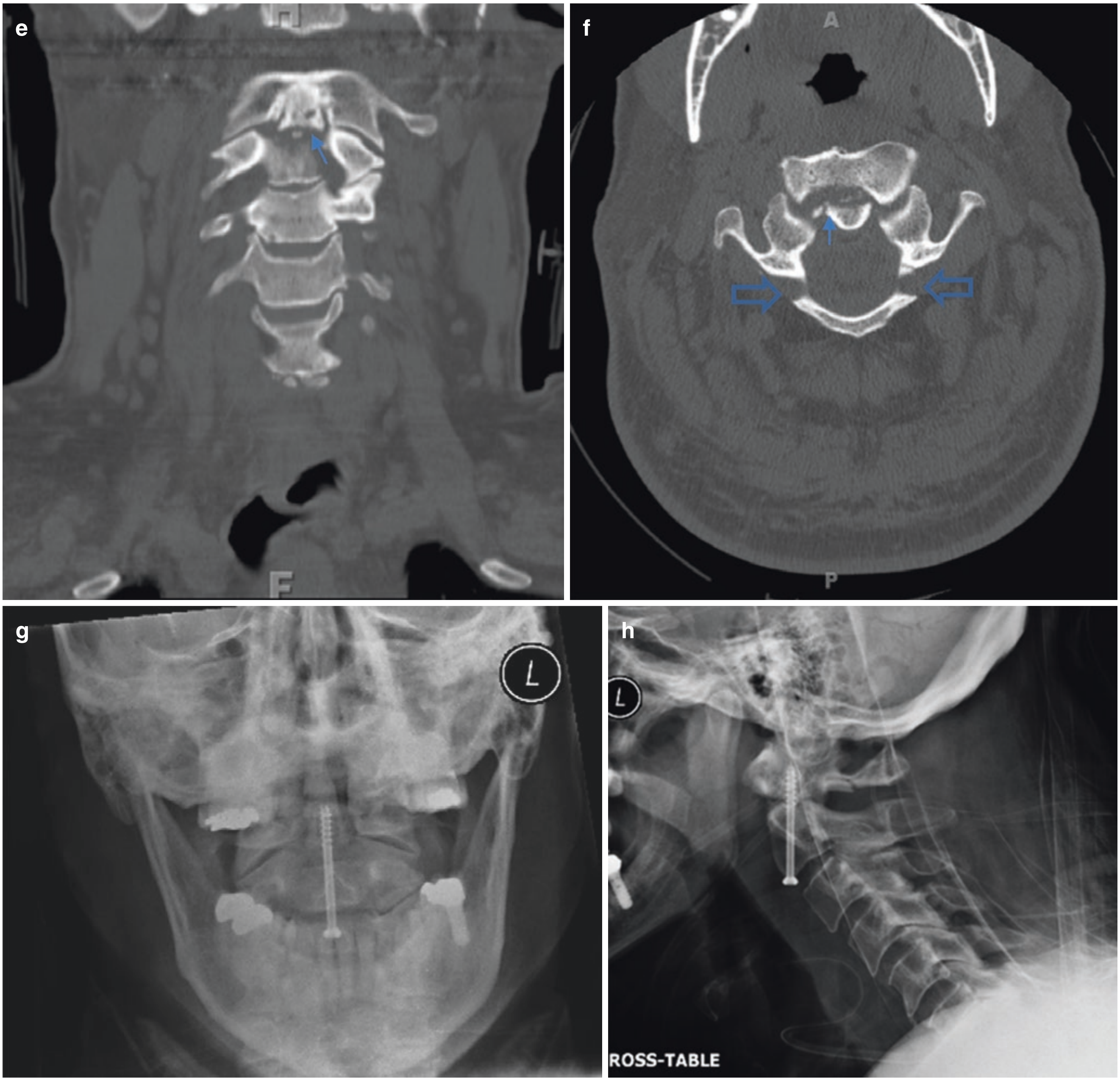


Fig. 30.6 (continued)

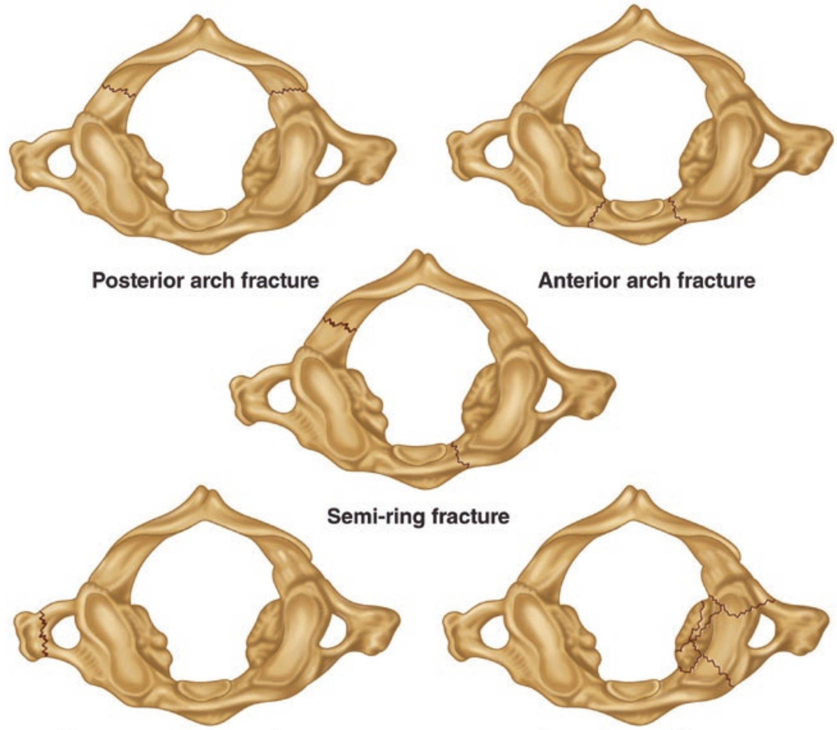
Although the clinical predictive values using this system appear to be good, there have been mixed results on interrater reliability [35–39].

Spinous process injuries of the lower cervical spine are often referred to as clay-shoveler's fractures (Fig. 30.15). These most commonly occur in the lower cervical spine (C7 is the most frequent one) and historically were described as fatigue-avulsion fractures but may occur with direct impact or forced hyperflexion (Figs. 30.16 and 30.17). There is no separate classification for these injuries [6].

Clinical Presentation

Athletes who sustain cervical spine fractures usually present with pain and often associated muscular spasm [24]. Point tenderness of the midline cervical spine is commonly present and should alert the provider to the possibility of significant cervical spine injury. Neurologic involvement will generally reflect the level of the involvement with severe injuries presenting with both upper and lower extremity neurological findings. Athletes who present symptoms hours to days after

Fig. 30.7 Atlas fracture classification



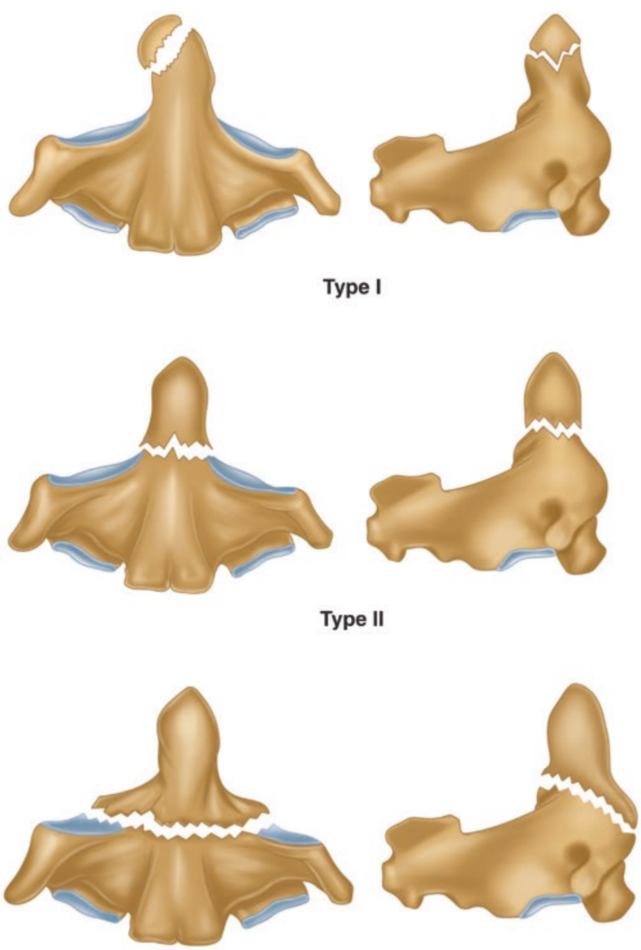


Fig. 30.8 Odontoid fracture classification

an injury are generally unlikely to have significant fracture or neurological injury but may have more stable injuries such as spinous process fractures that present with point tenderness.

Initial Management

With all spine-related injuries, the clinical evaluation begins with the protocols established and learned from Advanced Trauma Life Support (ATLS) regarding the athlete's airway, breathing, and cardiac functions [3, 40]. However, ideally the care begins prior to any injury. An emergency action plan should include a plan on how to immobilize and transfer the injured athlete. In addition, it should include information on local resources and emergency medical services (EMS) protocols. This is imperative as there may be difference and changes in EMS protocols as evident by recent recommendation regarding the use of spine boarding [41]. Pregame medical team meetings should be performed prior to competition. Ideally healthcare providers will have practiced immobilization techniques, helmet and pad removal, and transfer techniques. Spinal immobilization should be entertained in all cases of suspect spinal injury in a symptomatic athlete, and a conservative approach should be taken with respect to the individual athlete, injury mechanism, and provider experience.

Please refer to Chap. 4 for spinal immobilization for details.

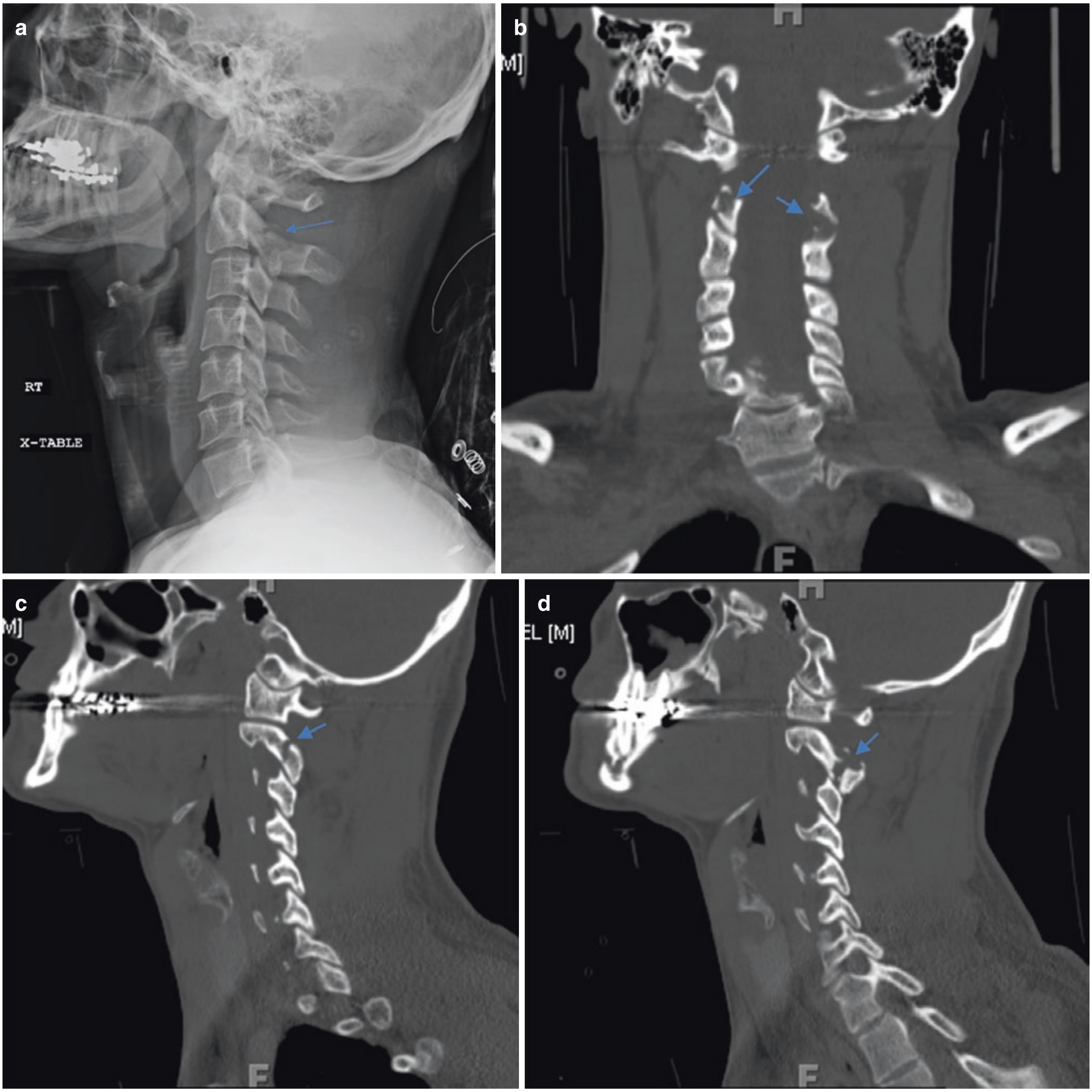


Fig. 30.9 Bilateral C2 pars interarticularis fractures (arrow) in a 31-year-old male as a result of a ski injury (**a**). A coronal CT view (**b**) shows the fractures better. Sagittal views demonstrate right (**c**) and left (**d**) pars interarticularis fractures (arrows)

Diagnosis

Imaging of suspected spinal injuries has evolved over time but is not without controversy in its application. The Eastern Association for the Surgery of Trauma (EAST) guidelines suggest CT as the primary imaging modality for cervical spine clearance [42]. While most facilities have gone away from the use of plain radiography in favor of CT scan, there are significant differences in application of suggested guidelines [43]. Both National Emergency X-Radiography Utilization Study (NEXUS) and Canadian C-spine rules can be used to determine need for imaging [44]. However, these imaging rules may not pick up stable injuries that are considered clinically nonsignificant in the general population but may be significant in the athlete. These include avulsion injuries, small compression injuries, and other stable fractures. The rules may identify athletes who may safely defer emergent imaging, but imaging may still be considered as

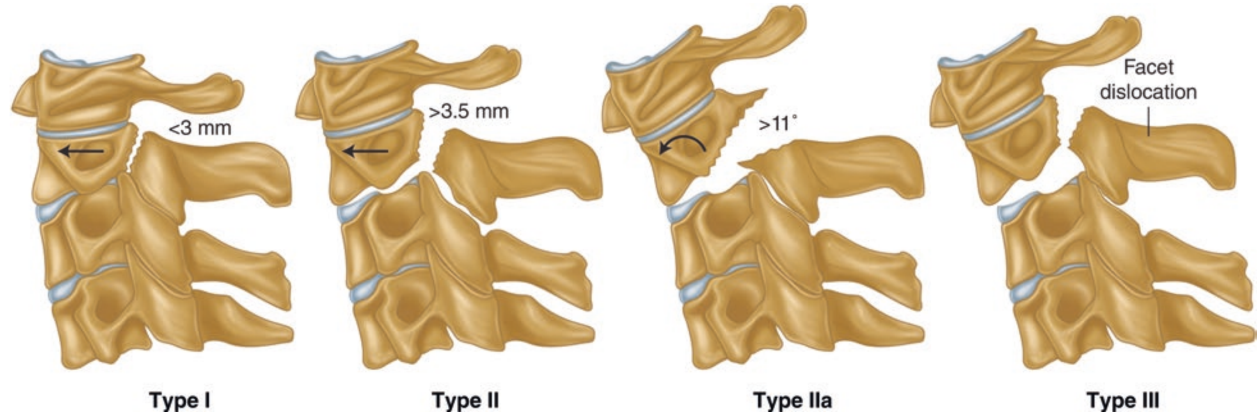


Fig. 30.10 Levine-Edwards hangman's fracture (traumatic spondylolisthesis) classification

Table 30.1 Levine-Edwards classification of hangman's fractures [29]

Type	Mechanism
I	Hyperextension-axial loading <3.5 mm of translation
II	Hyperextension-axial loading with anterior flexion and compression
	Significant translation or angular deviation
IIa	Hyperextension-axial loading with anterior flexion and distraction
	Minimal translation with wide angulation
III	Flexion compression with wide angulation
	Disassociation of C2–C3 facets

injuries may have impact on athletes ability to return to play. It should be noted that imaging rules such as NEXUS and the Canadian C-spine rules have not been validated in the out-of-hospital setting.

Plain Radiography

Plain radiography has the advantage of being readily available. They are generally not recommended in the evaluation of cervical spine trauma, unless the CT scan is not available [42]. The reported sensitivity is approximately only 50% compared to 98% for CT scan [42]. Indication for use includes motion artifact on CT imaging and patients <14 years of age [45]. Lateral imaging is the most important image to evaluation normal alignment (Fig. 30.18), and flexion and extension views are generally not indicated [45–47].

CT Scan

Multidetector CT has largely replaced radiographs as the imaging modality of choice in spinal injuries. Although overall study quality is low to moderate, CT imaging has high reported sensitivity and specificity (>95%) for both stable and unstable injuries [42, 48]. Most guidelines (e.g., EAST) recommend clearance of the cervical spine with negative CT imaging. In younger populations, due to the concerns for radiation exposure, imaging guidelines (e.g., NEXUS and Canadian C-spine) encourage the use of imaging rules to determine need for imaging [45]. In general, CT without contrast is recommended [48].

MRI

The role of MRI after cervical spine trauma is not fully understood with varying recommendations [48]. The importance of MRI findings or ligamentous and other potentially unstable soft tissue injuries are not clear, and false-positive findings are commonly present. MRI is generally obtained if there are findings on CT scan associated with neurological deficit (Fig. 30.14) [48]. MRI should be performed without contrast and as urgently as feasible. For patients with negative CT findings but persistent pain, MRI can be considered. The role of MRI to detected clinically significant unstable spinal injuries is not clear, but most recommendations do not suggest its use as it may not improve healthcare outcomes at increased cost [49].

Imaging for Obtunded or Unreliable Patients

Most guidelines suggest that negative CT alone is adequate to clear obtunded or unreliable patients with suspected cervical spine injuries. However the overall quality of evidence is low to moderate and others have recommended varied protocols in this population. Because definitions of obtunded and unreliable are not clear, one may consider additional imaging or treatment in selected cases although most patients can be cleared with negative CT scanning [48].

Indications for Orthopedic or Neurosurgery Referral

Most cervical spine fractures should be referred for orthopedic or neurosurgical consultation. This includes upper spine injuries and subaxial injuries with SLIC scores ≥4. Subaxial injuries with SLIC scores of 3 are managed conservatively,

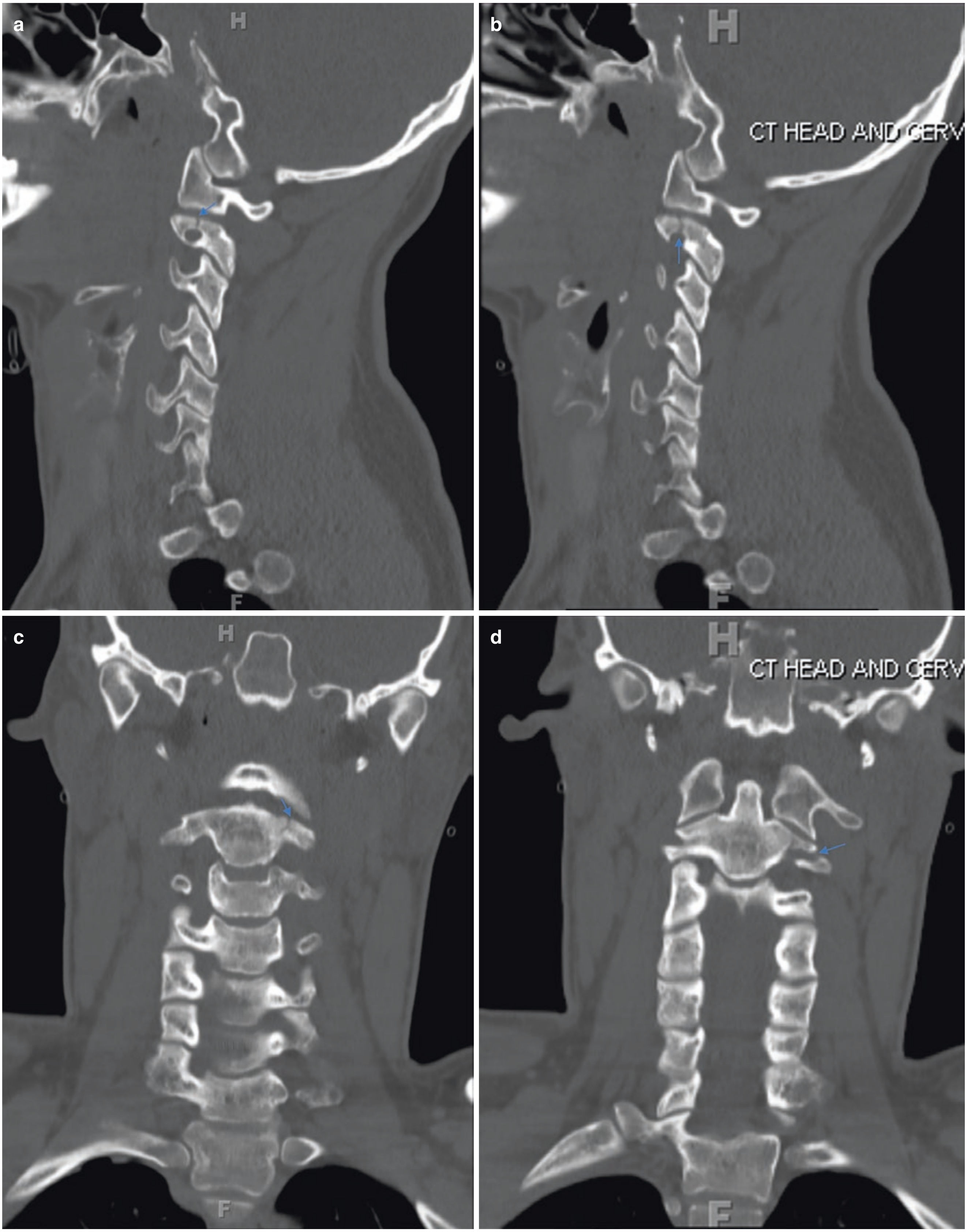


Fig. 30.11 A non-displaced left C2 lateral mass fracture (arrows) with extension into the left foramen transversarium only visible on CT (**a**–**f**) in a 23-year-old male as a result of a tree strike while skiing

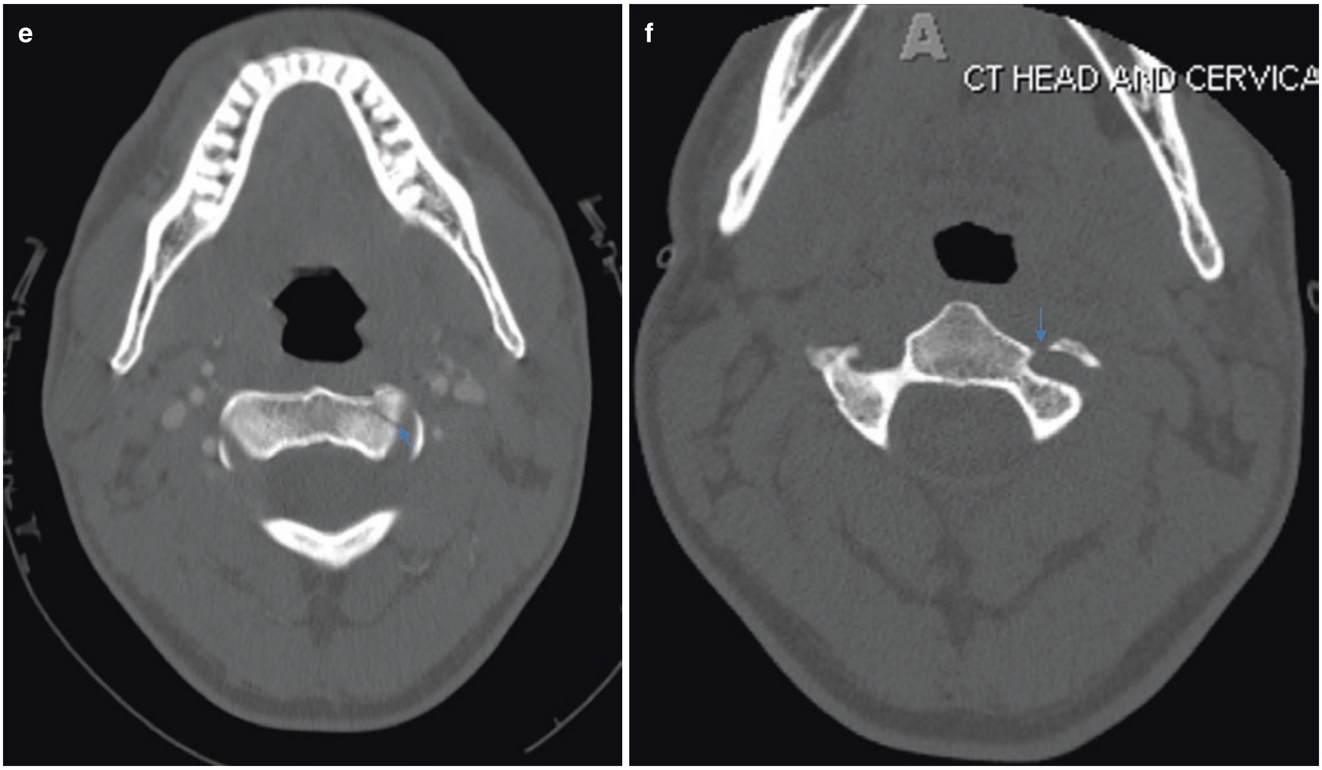


Fig. 30.11 (continued)

and referral should be based on provider's comfort level and experience [17, 18, 23, 47]. Most spinous process fractures can be managed without referral unless there is persistent pain and surgical excision is being considered [51].

Follow-Up Care

For stable injuries there are no evidence-based recommendations for follow-up care. Spinous process fractures are managed symptomatically and may only need follow-up if symptoms are persistent beyond 4–6 weeks. Conservatively treated upper and lower spine fractures will generally be followed every 1–2 weeks for the first month and every 2–3 weeks until healing has occurred. The follow-up interval may vary based on the individual patient and provider experience.

Athletes with more severe injuries, SLIC ≥4, should be managed on an individual basis with provider's experience and preference playing a large role in determining follow-up intervals and necessary imaging. Management and follow-up care of specific fractures are discussed below.

Occipital Condyle Fractures

It is generally agreed that Type I and II fractures can be treated with immobilization. The type of immobilization varies from halo collar to rigid or semirigid cervical collars [20]. These options are based on provider's preference with length of immobilization generally in the 2–3-month range [50].

For unstable injuries (Type III), options include halo vest or fusion of the occipital cervical joint [20, 50]. As there are no specific guidelines to determine which treatment is more appropriate, treatment should be individualized. If conservative treatment is undertaken, immobilization is usually continued for 12 weeks [20, 50].

Atlas Fractures

Most isolated atlas fractures can be treated conservatively with immobilization [24]. Type of immobilization varies by provider's preference as outcome studies are lacking. Athletes with transverse atlantal ligament (TAL) injuries and bony avulsion generally do well nonoperatively, while pure ligamentous injures may require surgical intervention [24].

Strong consideration for referral should be given for most of these injuries given the lack of consensus guidelines and potential for associated injuries. Surgery, either osteosynthesis or C1–C2 arthrodesis, is considered for displaced fractures or multiple fractures or if associated with C2 injuries. Treatment of injuries and surgical indication are covered in Table 30.4 [13, 24].

Axis Fractures

Odontoid fractures can generally be treated with cervical immobilization unless there is displacement [26, 27]. Both

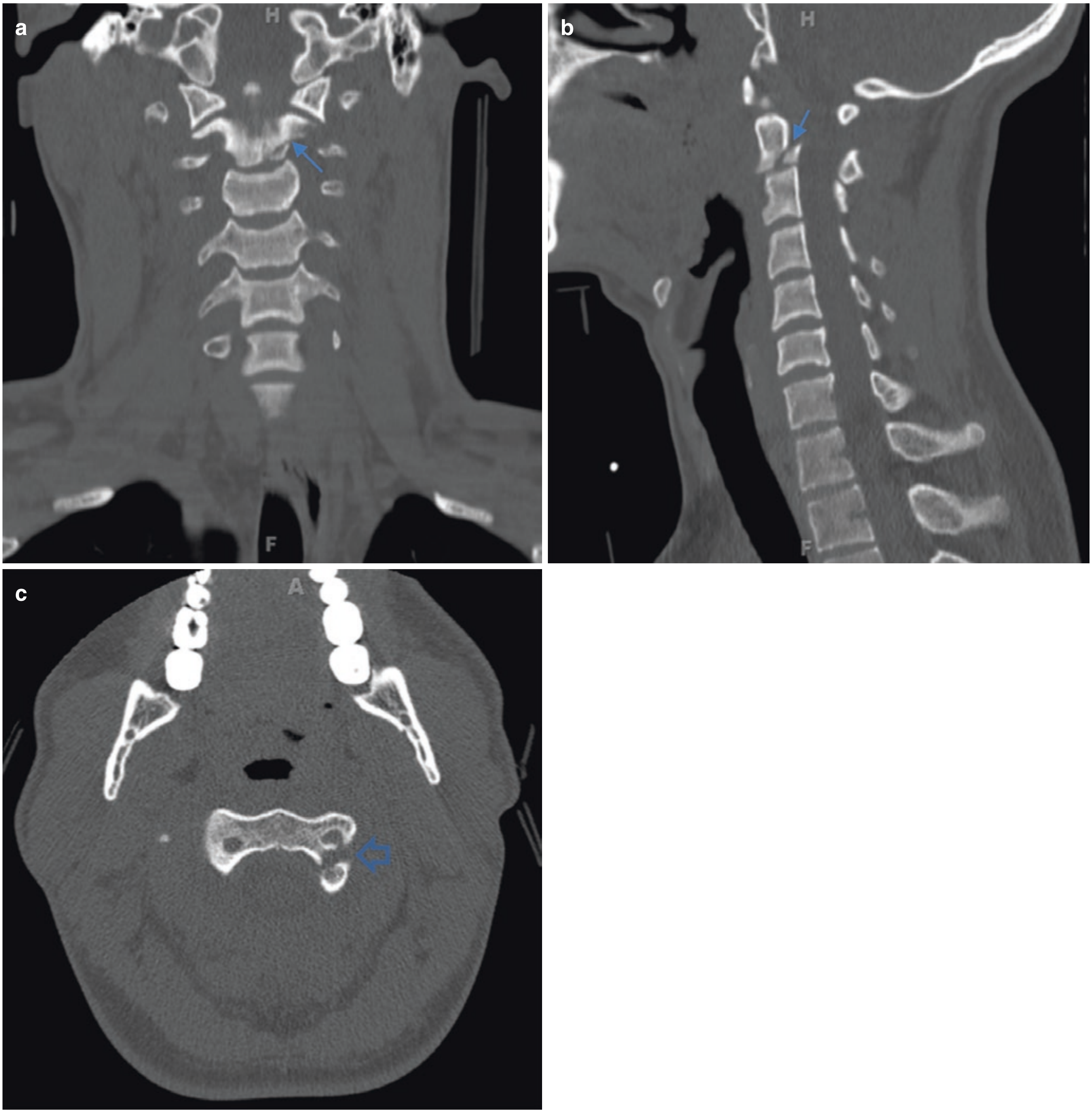


Fig. 30.12 A comminuted fracture of the C2 vertebral body (arrows) through the left transverse foramen (open arrow) only visible on CT (**a**–**c**) in a 20-year-old male as a result of a ski injury

rigid cervical and halo immobilization have been used with no quality evidence to guide immobilization choice. Type II odontoid fractures in patient 50 years old and older generally do better with surgical stabilization [27]. Type I fractures overall tend to do the best with union rates nearly 100% in most studies. Type III injuries also tend to do relatively well with immobilization with union rates reported between 50% and 99% [27]. Type II injuries have a higher risk of nonunion, 50–75%. Both age, >50 years old, and degree of displacement, >5 mm, increase the risk of nonunion [26, 27]. Surgical intervention should be considered in this group as well as for failure of conservative treatment of other fractures. Fusions of C1–C2 or odontoid screw fixation (Fig. 30.6) are the preferred treatment [26, 27]. It is important to note that most of patients in these studies were older with younger patients defined as <50–55 years of age. Table 30.5 summarizes treatment of odontoid fractures.

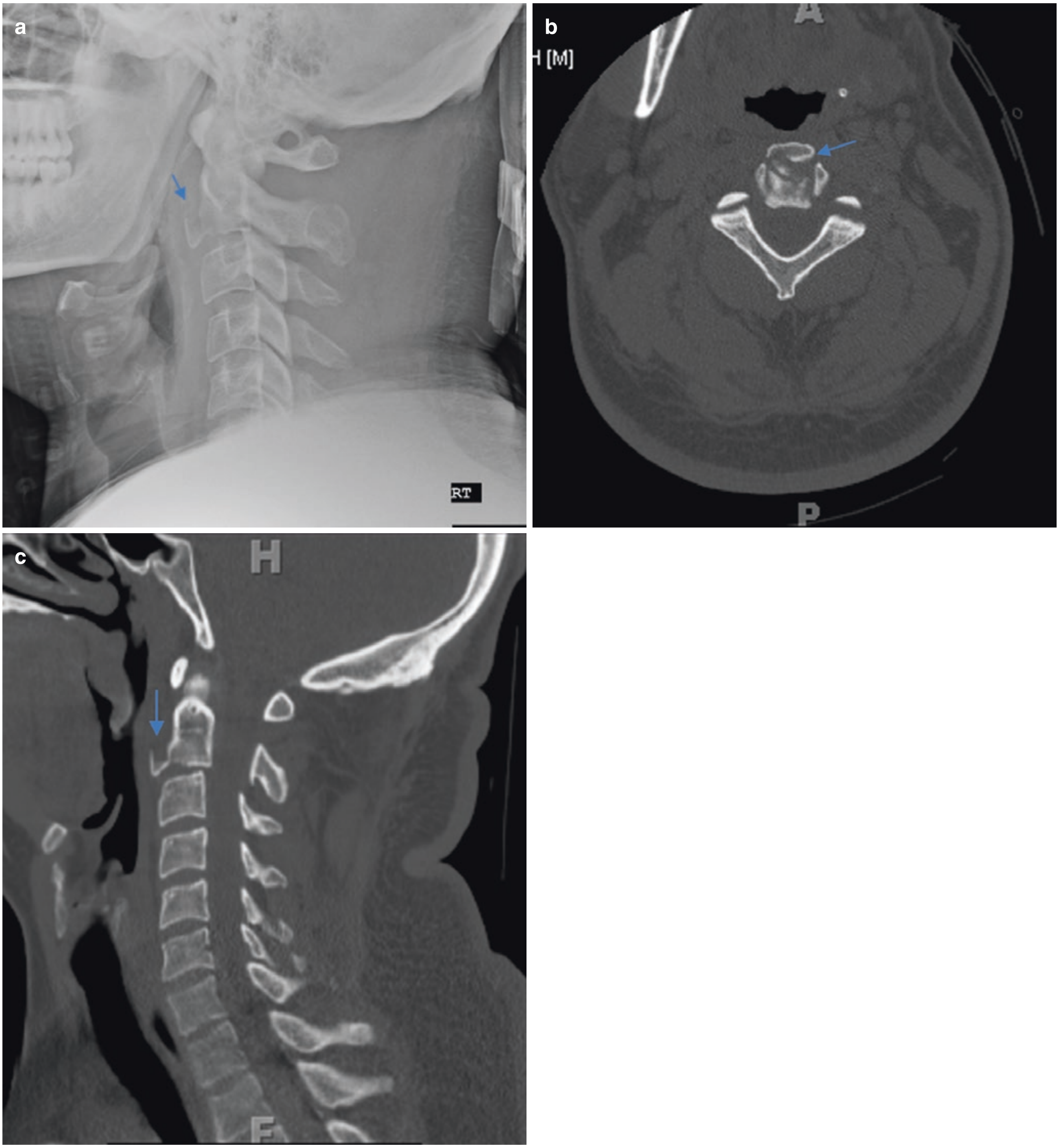


Fig. 30.13 An anterior and inferior C2 body fracture (arrow) in a 28-year-old male as a result of a mountain bike injury (**a**). CT images also demonstrate the fracture (arrows) as well (**b**, **c**)

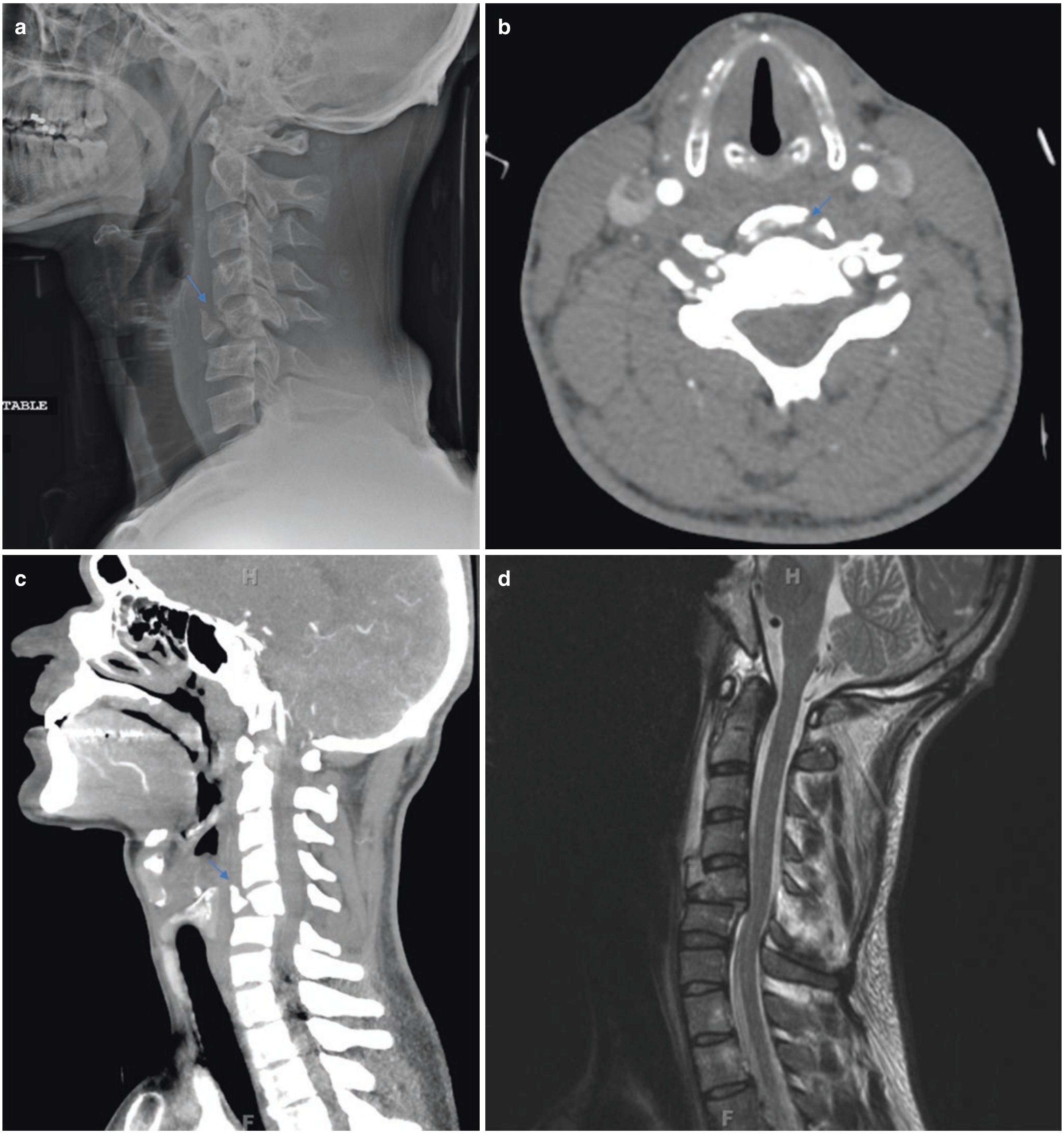


Fig. 30.14 A three-column unstable C5 fracture/dislocation (arrow) in a 32-year-old male as a result of a mountain bike injury (**a**). CT (**b**, **c**) and MR (**d**, **e**) images demonstrate the extent of the fracture with narrowing of the spinal canal and mild cord compression. Postoperative radiograph shows near-anatomic alignment (**f**)

Fig. 30.14 (continued)

Table 30.2 AO subaxial spine fracture classification [2]

Type A – compression/ burst injuries	Axial compression
	Anterior element injury
	Posterior elements intact
Type B – tension band injuries	Injury to posterior elements
Type C – displacement injuries	Injury to anterior and posterior elements creating displacement of structures

Table 30.3 Subaxial Injury Classification and Severity Score (SLIC) [33]

Morphology	No abnormality	0	Imaging
	Compression	1	X-ray
	Burst (add to compression)	13	CT
	Distraction	4	
	Translation/rotation		
Disc-ligamentous complex	Intact	0	Imaging
	Indeterminate: isolated interspinous widening or MRI signal only	1	MRI
	Disrupted: widening of the anterior disc space or facet dislocation	2	
Neurological status	Intact	0	
	Nerve root injury	1	
	Complete spinal cord injury	2	
	Incomplete spinal cord injury		
	Continuous spinal cord compression	3 +1	

Although high-quality evidence is lacking, most hangman injuries can be treated with initial conservative treatment with either rigid cervical or halo immobilization with both conservative and surgical intervention leading to high union rates, 94% vs 99% [26, 27, 29]. Traction may be considered in some injuries prior to immobilization. Levine IIa and III should be considered for surgical referral [27]. Surgery involves C2–C3 fusion with the approach, anterior or posterior, being based mostly on surgeon preference [27, 29]. Table 30.6 summarizes treatment of hangman's fractures.

Atlas body fractures tend to have minimal displacement and low risk of neurologic injury. Type I and II injuries can generally be treated with rigid immobilization, halo or rigid collar, for 10–12 weeks. Type III injuries are more commonly treated operatively but the majority (>75%) are treated conservatively [31]. Recommendations suggest using degree of displacement to guide type of immobilization, <3 mm rigid collar, >3 mm halo, but there is significant variation. Most injuries should be considered for referral based on provider's experience. Surgical indications are also not clear, but many suggest displacement >3 mm, evidence of subluxation, and patients between ages 40 and 65 should be considered for surgical fixation [31].

Lower Cervical Spine Injuries

Treatment of the lower cervical spine fractures is similar to the thoracic and lumbar spine; however, classification alone

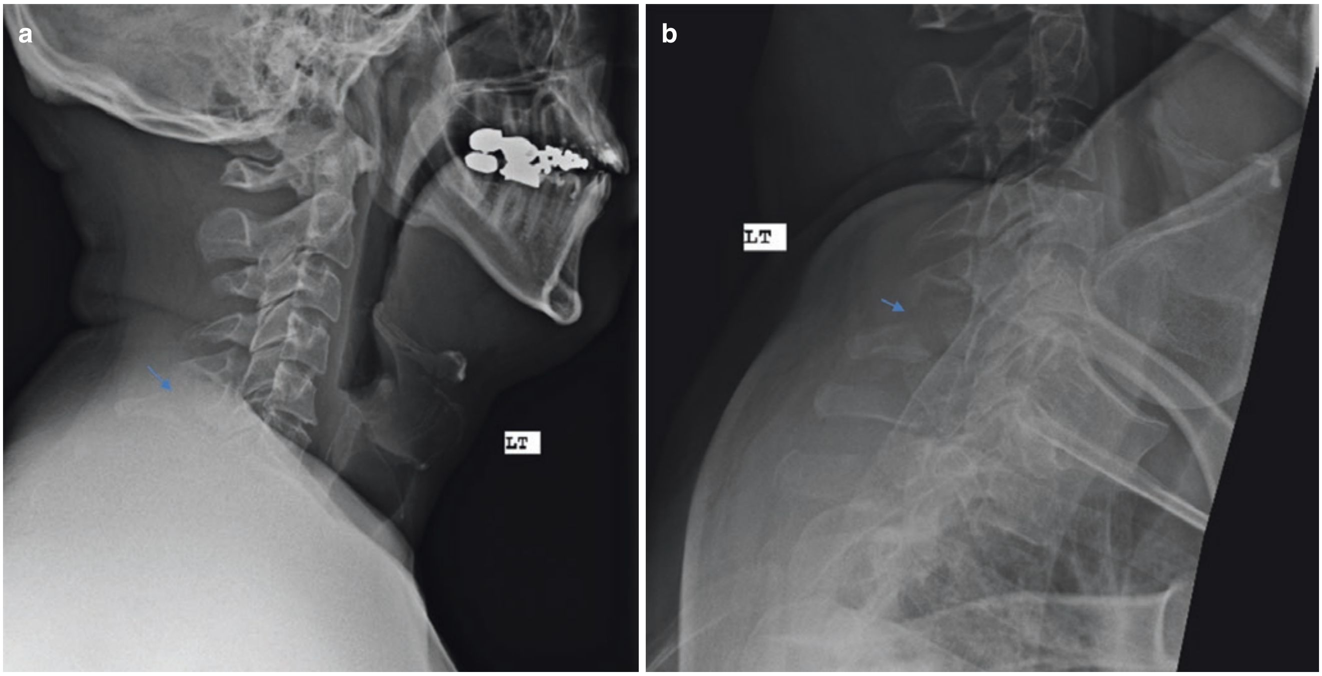


Fig. 30.15 C6 spinal process (clay-shoveler) fracture in a 46-year-old female as a result of a ski injury (**a**, **b**). The fracture (arrows) is more visible on swimmer's view (**b**)

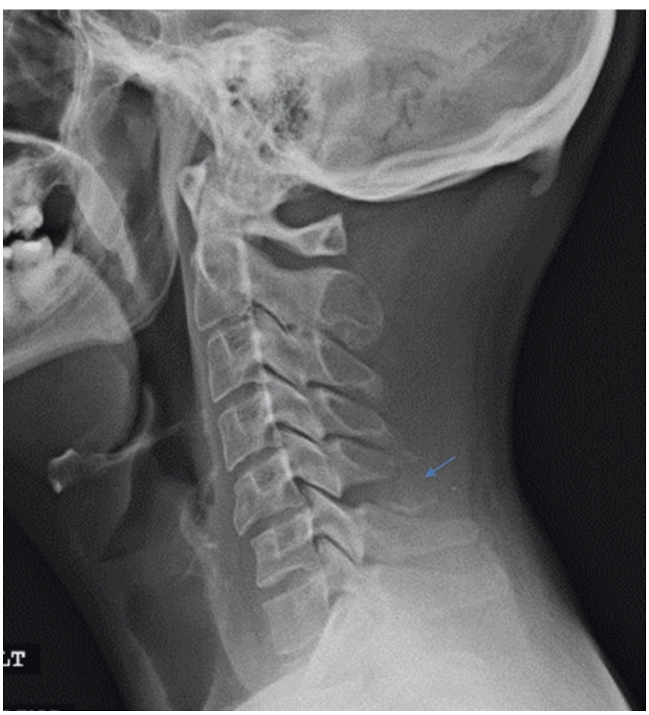


Fig. 30.16 An avulsion fracture of the C5 spinous process in a 26-year-old male as a result of a mountain bike injury

is not used to determine treatment. Using the SLIC classification (Table 30.3), injuries can be scored based on fracture morphology, ligamentous integrity, and presence or absence of neurologic findings. Athletes with a SLIC score of <3 are generally treated conservatively [33, 34, 39, 47]. Patients with a SLIC score of 4 may be considered for either conservative or surgical intervention with no consensus on how best to approach this group [33, 34, 39, 47]. SLIC scores of >4 (Fig. 30.14) are considered unstable injuries and operatively stabilized [33, 34, 39, 47]. Earlier surgical stabilization (<24 h) has been shown to improve outcomes highlighting the need for early recognition of these injuries and expedient transport to facilities capable of surgical care [46]. Surgical approach and treatment are determined by the individual patient and surgeon's experience and preference with both anterior and posterior approaches reporting similar neurological outcomes [39, 47]. Table 30.7 summarizes the treatment of subaxial spine injuries.

Return to Sports

There are no evidence-based guidelines for return to play with cervical fractures [54–58]. The determination of safe return to play varies based on the injury, desired sport, and individual athlete and provider. Thoughtful discussion should always include gaps in our understanding of safe return balancing the level of participation, age, emotional, and sometimes financial consequences of not allowing an athlete to continue in their sport. Expert opinion has defined contraindications for return to contact or collision sports [54–58]. Athletes with single-level fusion of the lower cervical spine can be considered for return to play; however,

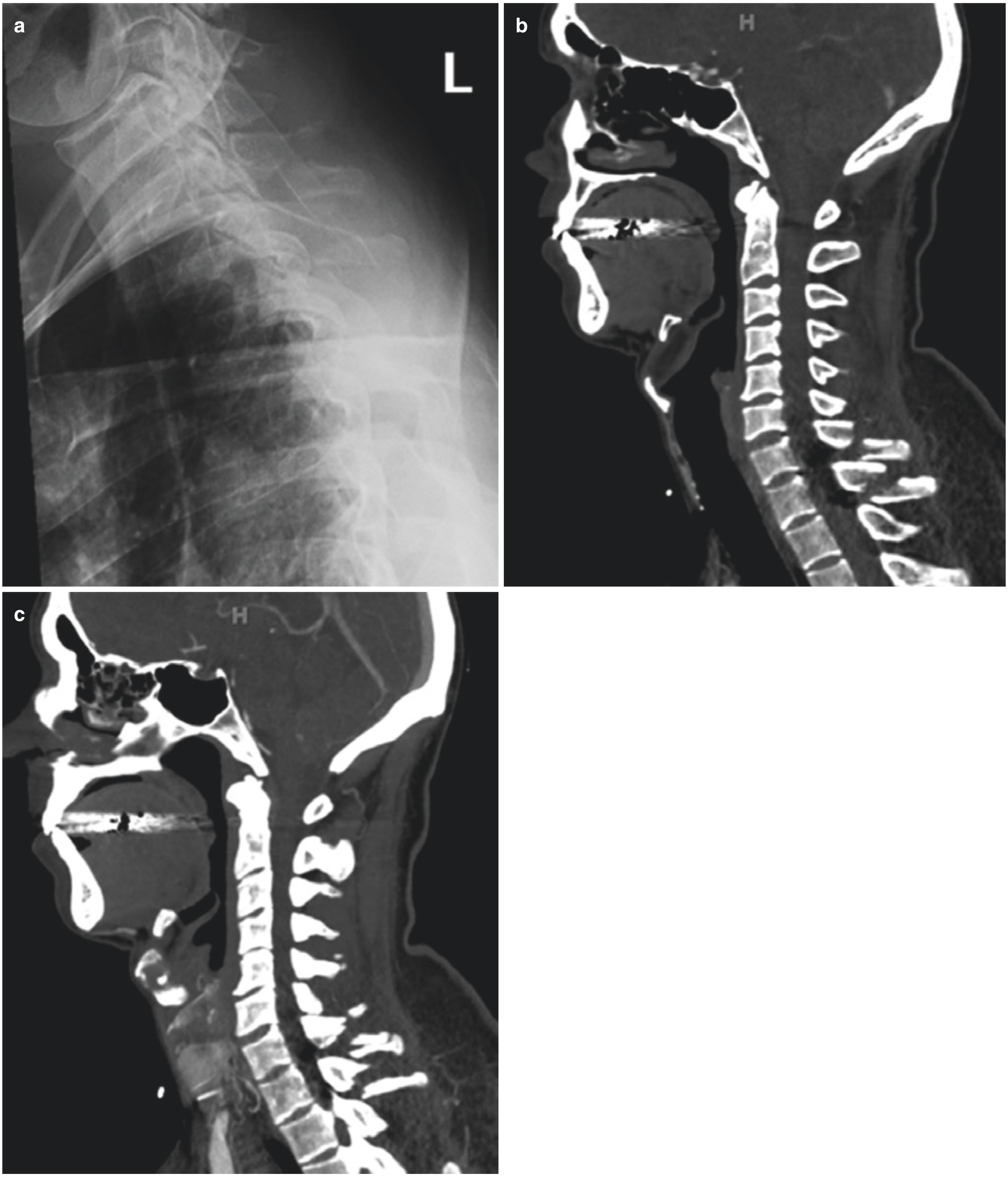


Fig. 30.17 C5, C6, C7, and T1 spinal process fractures in a 47-year-old male as a result of a ski injury (**a**–**c**)

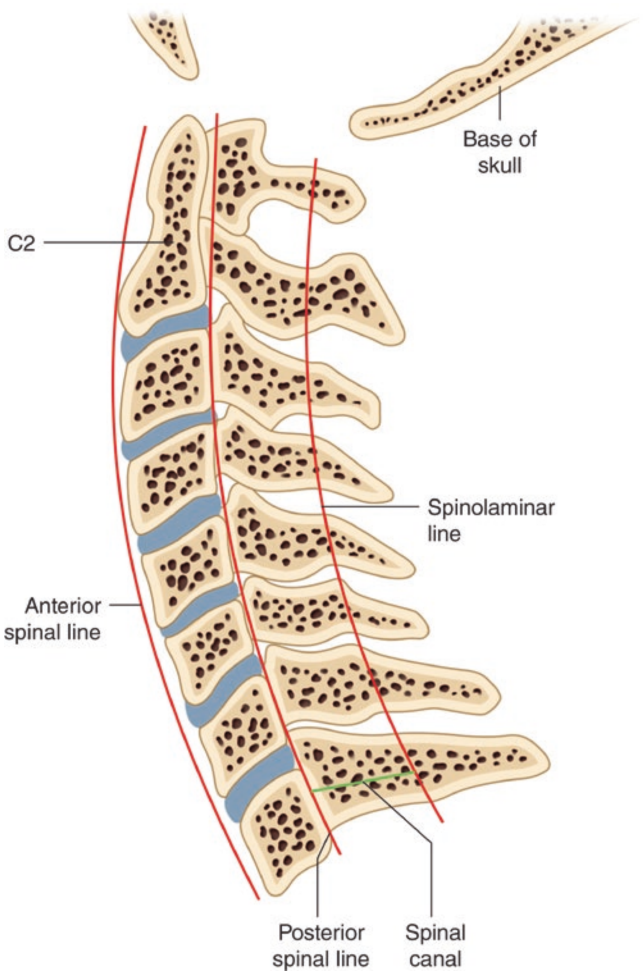


Fig. 30.18 Schematic lateral cervical spine view

Table 30.4 Treatment of atlas fractures [13, 24]

Pattern	Treatment	Referral or surgical indication
Posterior arch fracture	Rigid collar	Displaced fracture >5–7 mm
	10–12 weeks	Multiple fractures
	Halo vest for unstable or multiple injuries	Fractures associated with TAL injuries
Anterior arch fractures	Rigid collar	Displaced fracture >5–7 mm
	10–12 weeks	Multiple fractures
	Halo vest for unstable or multiple injuries	Fractures associated with TAL injuries
Transverse lateral mass fracture	Rigid collar or halo vest	Comminuted or multiple fractures
	10–12 weeks	
Transverse atlantal ligament (TAL)	Rigid collar or halo vest	Pure ligamentous injuries
	Rigid immobilization	
	10–12 weeks	

Table 30.5 Treatment of odontoid fractures [26, 27]

Fracture	Treatment	Referral or surgical indications
Type I	Rigid cervical collar	Nonunion
	Halo immobilization	
	10–12 weeks	
Type II	Rigid cervical collar	>50 years old
		>5 mm of displacement
	Halo immobilization	Nonunion
	10–12 weeks	
Type III	Rigid cervical collar	Nonunion
	Halo immobilization	Patient/provider preference
	10–12 weeks	

Table 30.6 Treatment of hangman's fractures [26, 27, 29]

Fracture	Treatment	Indications for referral
Type I	Rigid immobilization	Nonunion
	Most commonly halo but rigid cervical collars have been used	
	10–12 weeks	
II	Rigid immobilization	Nonunion
	Most commonly halo but rigid cervical collars have been used	
IIa	Can consider halo immobilization for poor surgical candidates	Most fractures should be considered for surgical referral
III	Consider halo immobilization for poor surgical candidates	Most fractures should be considered for surgical referral

Table 30.7 Subaxial spine treatment based on SLIC score [33, 39, 47, 63]

Score	Treatment	Notes
<4	Nonoperative	Spinous process, require symptomatic treatment only
	Rigid cervical collar	
	6–12 weeks	Collar is considered a comfort measure
4	Nonoperative	Individual patient and injury may determine consideration for surgical intervention
	Rigid cervical collar	
	6–12 weeks	
	Operative considered	
>4	Operative	Surgical intervention based on injury pattern and provider preference

studies mostly include athletes who have had fusion for disc issues rather than fractures [59]. Athletes have high levels of return (~75%) after fusion for disc issues, but athletes with fractures return at much lower levels [56–59]. Given the overall low risk of cervical spine injury in sports, data is lacking on the small number of athletes who have returned to pre-injury competitive level and their associated risk of re-injury.

Basic criteria for return to play in all injuries are resolution of pain, return to normal motion and strength, and in most injury radiographic evidence of healing and stability. While flexion and extension views are often recommended for stability evaluation, it is unclear how they should factor into return to play decisions. Most experts suggest evidence

Table 30.8 Return to play recommendations for cervical spine fractures [54–58]

Fracture	Recommendation	Notes
Atlas	Relative contraindication	C1–C2 fusion is an absolute contraindication
		Instability is an absolute contraindication
Axis	Relative contraindication	C1–C2 fusion is an absolute contraindication
		Instability is an absolute contraindication
Subaxial spine	Nonoperative	Persistent neurologic injury is considered an absolute contraindication
	Relative contraindication	
	Single-level fusion	Transient neurologic dysfunction is considered a relative contraindication
	Relative contraindication	
	Multilevel fusion	Instability is an absolute contraindication
	Absolute contraindication	
Spinous process	No restriction	

of instability as an absolute contraindication to return to sports [57, 58]. Table 30.8 summarizes recommendations for return to play.

Complications

Persistent pain, malunion, nonunion, and loss of motion are the most common complications. Based on the type of fracture and treatment options, nonunion rates can be as high as 50% [27]. Delayed surgical intervention for higher-grade injuries may increase risk of poorer neurologic outcomes [39, 47]. Posterior surgical approach has higher risk of wound complications, while anterior approach is associated with an increased risk of swallowing difficulties [47].

Pediatric Considerations

There are several areas of consideration when evaluating a pediatric athlete with suspected or confirmed cervical spine injury. Sports injuries account for approximately 50% of fractures with subaxial injuries being most common followed by upper cervical spine [4]. Anatomically there are ossification centers that can be confused with fractures in children. The atlas has ossification centers in the body and both lateral masses. The axis additionally has ossification centers in the body, lateral masses, and the odontoid. The lower cervical spine has centers in the body and each of the neural arches. These centers allow significantly more mobility accounting for the increased risk of upper cervical spine injuries in children less than 8 years of age. After age 8 years, these centers close, and the spine and associated injury patterns more closely resemble the adult spine [52, 53].

Imaging with plain radiography is still indicated but several normal variants exist (e.g., psuedosubluxation of C2–C3 or C3–C4). In psuedosubluxation the posterior laminar line remains intact where in true fracture it is disturbed [52, 53].

Because of increased flexibility, children may be at risk for spinal cord injuries in the absence of fracture or ligamentous injuries. These injuries have been called SCIWORA (spinal cord injury without radiographic abnormality). Not all injuries are associated with MRI findings. MRI findings do not correlate consistently with outcomes. Acute treatment of SCIWORA is similar with spinal precautions taken. Most injuries are managed nonsurgically with up to 12 weeks of rigid cervical collar [4, 52, 53].

Soft Tissue Injuries

Increased mobility of the cervical spine makes it more susceptible to strains and sprains than other parts of the spine [60]. While these injuries are commonly reported in sports, their true incidence is unknown. They are commonly a diagnosis of exclusion after more serious spinal injury has been ruled out [61]. The terms whiplash and cervical strains and sprains are sometimes used interchangeably. While athletes may experience similar injury pattern, whiplash-associated disorder (WAD) is most commonly associated with motor vehicle accidents, but it can occur with sporting activity [62]. WAD is defined as a collection of symptoms (e.g., pain, stiffness, and neurologic signs) that occurs with an acceleration and deceleration force usually in motor vehicle accidents [63]. Athletes may experience similar symptoms with similar mechanism of injury in both acceleration/deceleration forces and forces applied to the neck during muscle contraction creating overload of the soft tissue structures [61]. Common complaints include pain, stiffness, and sometimes neurologic symptoms [60]. In more severe injuries, significant muscle spasm may be present. Athletes will present with tenderness to palpation along paraspinal muscles and ligamentous attachments with varying degrees of loss of range motion. Pain and limitation in range of motion may worsen over hours to days.

Neurologic findings should prompt further evaluation if not already undertaken based on the initial presentation of the athlete. Radiographs are not always necessary but may be performed if clinical suspicion for instability exists. Standard AP and lateral views allow for assessment of alignment. The addition of flexion and extension views has not been shown to add to the clinical evaluation of neck trauma; however, they can be considered in athletes where more subtle instability may affect return to play decisions [46, 61]. Other imaging modalities including MRI are usually reserved for

the evaluation of more severe spinal injuries and should be considered if neurologic symptoms are present even if they are transient [60]. Treatment should include early mobility and active neck exercises as these have been found to be beneficial in reducing pain and returning to function [63]. Immobilization has not been found to be helpful as it should be limited as much as possible if used. Other modalities such as heat or ice and nonsteroidal anti-inflammatories as well as cervical manual therapy can be used on an individual basis [61]. Return to play should not occur until normal range of motion and strength return.

Neurologic Injuries

Traumatic Brachial Plexopathy

Injuries to the brachial plexus or cervical nerve roots, also commonly called stingers or burners, are one of the most frequent neurologic injuries seen in sports [64]. The frequency of this injury in many sports is unknown, but studies report an incidence of 20–65% in American football with recurrence rates as high as 90% [64–66]. A study of rugby players found approximately one third of high school and university rugby players experienced this injury during a season [67]. Although the exact mechanism is not clear, it is believed these injuries are caused by a traction injury to the brachial plexus and cervical nerve root compression or from a direct compressive force [64]. Traction injuries occur with the lateral bending of the neck and depression of the opposite shoulder. Nerve root compression tends to occur with the neck with lateral bending of the neck with hyperextension. Nerve root injuries most commonly occur at the C5 and C6 levels and a narrow foramen is thought to be a risk factor. Athletes will commonly present with burning pain that radiates down the arm accompanied by varying degrees of weakness and numbness or paresthesia [64]. Initial evaluation should ensure there are no other findings concerning for more central injury. Spinal precautions should be taken if there are any concerns. Examination for weakness of the upper extremity can help determine clinical severity of the injury with more mild injuries presenting only with transient numbness. More severe injuries can occur which can cause symptoms lasting from weeks to months and in rare circumstances there can be permanent injury. Electromyography (EMG) can be used after a few weeks to help classify nerve injury (Chap. 38). Outside of the concern for acute cervical spine injury, the role of imaging is generally limited to persistent symptoms or recurrent episodes, generally defined as two or more injuries. Standard radiographs (AP and lateral views) of cervical spine can help evaluate for alignment and other pathologies. The addition of other views, odontoid and flexion/extension, can be considered in individual athletes. Cervical MRI is the imaging of choice as it allows assessment of multiple tissues and the measurement of the mean subaxial cervical space available for the cord (MSCSAC). This measurement is obtained by subtracting the AP diameter of the cord from the AP diameter of the spinal canal at the disc level on a sagittal view. Measurements less than 5.0 mm are believed to be significant risk factor for recurrent stingers [64]. Return to play is based on resolution of symptoms and return to full range of motion and strength. A large percentage of players can return to competition the same game. While the average return to play is reported at approximately 3 days, most athletes return within 24 h with 80% that do not experience any loss of time [67, 68].

Acute Spinal Cord Injuries

While fortunately rare between 7% and 9% of new cases of spinal cord injuries are caused by sporting activity [68, 69]. These numbers reflect both organized and unorganized sports (e.g., diving) with the number in organized sports at approximately 900 injuries occurring over a 35-year period in high school and collegiate sports in the United States [1]. The cervical spine accounts for the majority (~60%) of spinal cord injuries, which usually involve unstable cervical spine fractures and dislocations [68, 69]. Transient injuries without bone injury can also occur and are termed transient quadriplegia or cervical cord neurapraxia (CCN) [69]. It is believed that spinal stenosis is a risk factor for CCN and it is graded based on length of deficit. The deficit in Grade I is less than 15 min, in Grade II is greater than 15 min but less than 24 h, and in Grade III it is more than 24 h [69]. Preparation for spinal emergency starts with an emergency action plan. This plan should include local and regional resources for spinal cord injury care. Transportation to a facility capable of definitive care should be the goal when possible as early surgical intervention, within 24 h, for appropriate patients improves outcomes [70]. After transportation the American Spinal Injury Association (ASIA) scoring system can be used to classify clinical impairments. This score includes measurement of both motor and sensory components based on the level of the injury. An impairment score can then be calculated and has shown good reliability [71]. ASIA scores at 72 h have been shown to be the most reliable and predictive of outcomes [71]. Common complications aside from the neurologic injury include hemodynamic instability, most commonly hypotension [72]. Monitoring of respiratory function and temperature are also important [72]. The role of corticosteroids in acute traumatic spinal cord injury has not been fully determined but should not be used in general but may be considered in individual cases [72]. Case reports of the use of hypothermia have prompted interest in this modality as a treatment of

spinal cord injury, and while it appears to be safe, outcome data is lacking at the moment limiting its clinical use [73]. Return to play of athletes with spinal cord injury is variable and based on the degree of recovery. Athletes with CCN can be considered for return to sports as it is not believed to be a risk factor for permanent neurological injury [69]. MRI evaluation to look for functional stenosis should be considered. Functional stenosis is considered by some to be a contraindication to contact and collision sports, but true risk is not clear [74]. Athletes with evidence of ligamentous instability and symptoms longer than 36 h and those with abnormal cord findings are also generally not recommended to return to collision and contact sports. Athletes that do return should be counseled on the risk of recurrence (e.g., about 50% in American football) as part of the return to play discussion [69].

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31 Thoracic Spine

Matthew G. Zmurko

Key Points

- Thoracic spine injuries are relatively rare in sports but account for approximately one third of neurologic injuries.
- Spinal precautions should be taken when an acute injury is suspected with immobilization and transport for further evaluation.
- Return to play is based on the individual circumstance as no clear guidelines exist.

Introduction

In general population, lower thoracic injuries (T10–T12) account for a large portion of spinal fracture and injuries [1]. These injuries occur most commonly in motor vehicle accidents and falls but are occasionally seen in sports [1]. The thoracic spine, due to its limited mobility, accounts for less overall injuries and fractures, when compared to the cervical and lumbar regions. The majority of the spinal fractures occur at the cervicothoracic and thoracolumbar transition zones. Mechanism of injury includes compression or distraction/rotation [1]. Isolated avulsions of the spinous process may occur from direct trauma or forceful muscle contraction and are also seen in sports. Despite the decreased frequency of thoracic spine fractures, thoracic spine fractures often result in more severe neurologic injury, as they contribute to approximately one third of all spinal cord injuries [2]. Sports-related injuries appear to be a lesser risk for neurological complication; however, because of the potential risk, suspected injuries should be treated with spinal precautions and referred for further evaluation [3].

M. G. Zmurko (✉)
Vermont Orthopaedic Clinic, Rutland Regional Medical Center, Rutland, VT, USA
e-mail: mzmurko@rrmc.org

Anatomy

The biomechanics and anatomy of the thoracic spine differs from the cervical and lumbar spine. The thoracic spine consists of 12 vertebral bodies that are aligned in a kyphotic nature versus the lordotic curvature of the cervical and lumbar spines (Fig. 31.1). Each thoracic vertebral bone consists of the vertebral body, which is connected to the posterior elements by two pedicles (Chap. 32; Fig. 32.1). The posterior elements are made up of the lamina and spinous process (Chap. 32; Fig. 32.2). These elements of the vertebral bone form the spinal canal in which the spinal cord travels through and supplies two thoracic nerves, which travel on the underside of each of the corresponding ribs. Ligamentous support adds additional stability and includes anteriorly the ligamentum flavum (lies within the vertebral foramen), the interspinous ligament between the spinous processes, and posteriorly the supraspinous ligament (Chap. 32; Fig. 32.1). The spinal cord typically ends at the level of the first lumbar vertebra where it then forms the cauda equina (Fig. 31.2). The cauda equina supplies the nerves to the lumbar and sacral regions of the spine.

Between each of the vertebral bodies is the intervertebral disc, which is the anterior articulation between the two vertebral bodies (Chap. 32; Fig. 32.3). The intervertebral disc is a fibrocartilaginous joint, which consists of an outer fibrous layer called the annulus fibrosus that surrounds an inner gel-like substance called the nucleus pulposus. This joint primarily distributes approximately 80% of the compressive forces across the joint. Posteriorly, the bilateral facet joints are synovial joints aligned vertically in the coronal plane to allow for rotation and lateral bending of the thoracic spine.

Each of the 12 vertebral bodies of the thoracic spine articulates bilaterally with a corresponding rib. The ribs from T2 to T10 articulate with two facet joints, one on the corresponding vertebra and the second with the adjacent superior vertebra. The T2–T10 vertebrae have a third articulation between the tubercle of the rib located distal to the rib head

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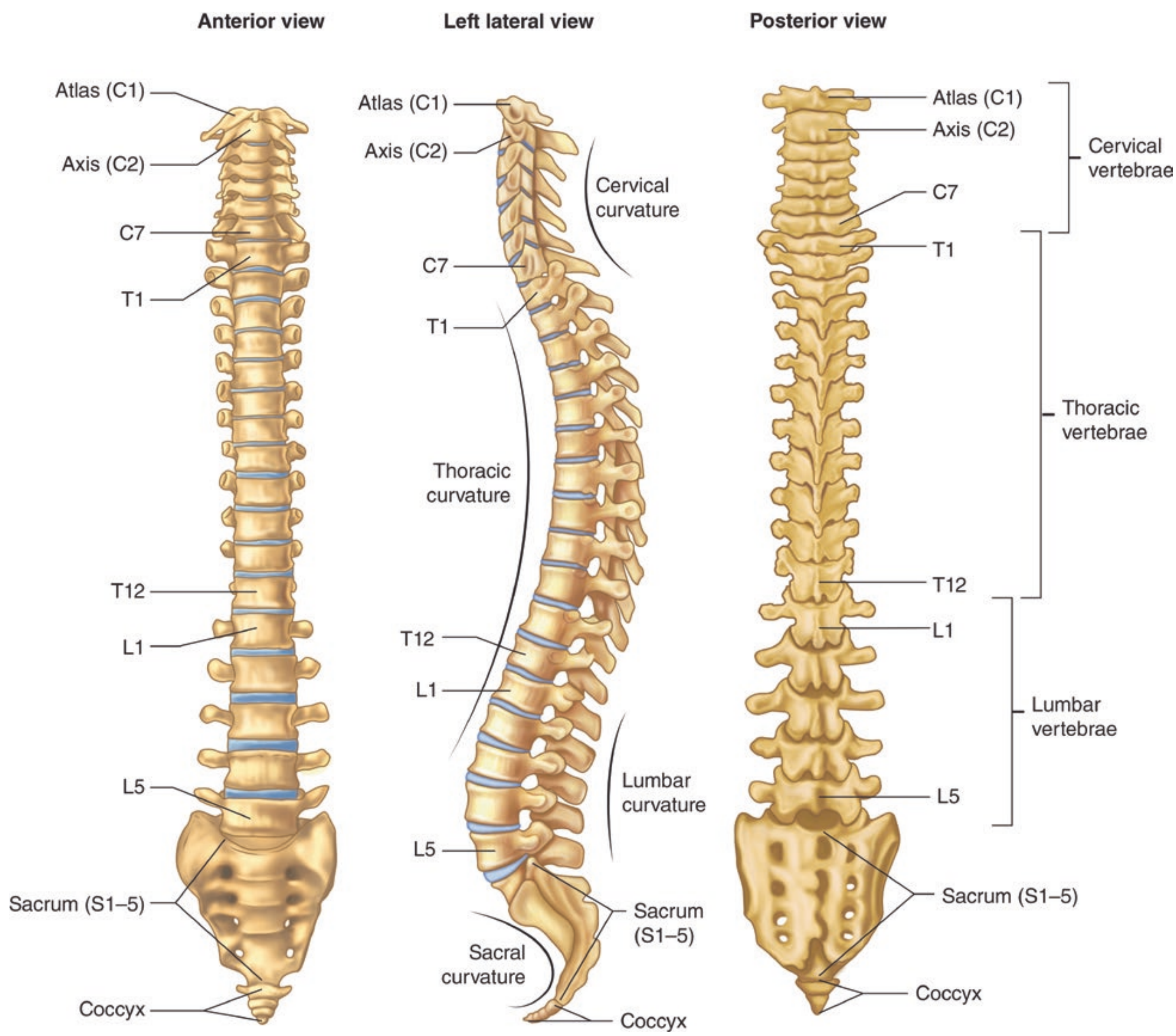

Fig. 31.1 Vertebral column anatomy

and the ventral portion of the transverse process of the corresponding vertebra. The ribs corresponding to vertebrae T1, T11, and T12 have a single articulation with their corresponding ribs.

The ribs from T1 to T7 are classified as true ribs since they attach directly to the sternum anteriorly, and ribs T8–T10 are false ribs as they attach indirectly to the sternum via fibrocartilage. The floating ribs T11 and T12 do not attach to the sternum. This increased stability of the thoracic spine, provided by the connection to the ribs and sternum, allows for better protection of the spinal cord and the organs found in the thoracic cavity [4, 5]. The thoracic spine has the smallest spinal canal-to-spinal cord ratio; therefore injuries to this area have an increased risk of resulting in spinal cord damage. However, it is the transition regions from the rigid kyphotic thoracic spine to more mobile lordotic lumbar and cervical spine regions that are the most often injured levels.

Fractures

Mechanism of Injury in Sports

Compression fractures (AO/OTA Types A1 and A2) (Figs. 31.3, 31.4, 31.5, 31.6, and 31.7) are flexion injuries and are commonly seen in sports such as snowboarding and mountain biking [3, 6, 7]. They can be seen in other sports as well but are overall more commonly seen in older patients with osteoporosis or after a fall from a height [8]. In these fractures the anterior edge of the vertebral body loses height, while the posterior border of the vertebral body maintains its integrity. Burst fractures and fracture-dislocations (AO/OTA Types A3 and A4) are the types of fracture most likely to result in neurologic sequelae but less likely to occur in sports compared to other mechanisms of injury [3] (Fig. 31.3). In burst fractures, a high-energy axial loading of the vertebral

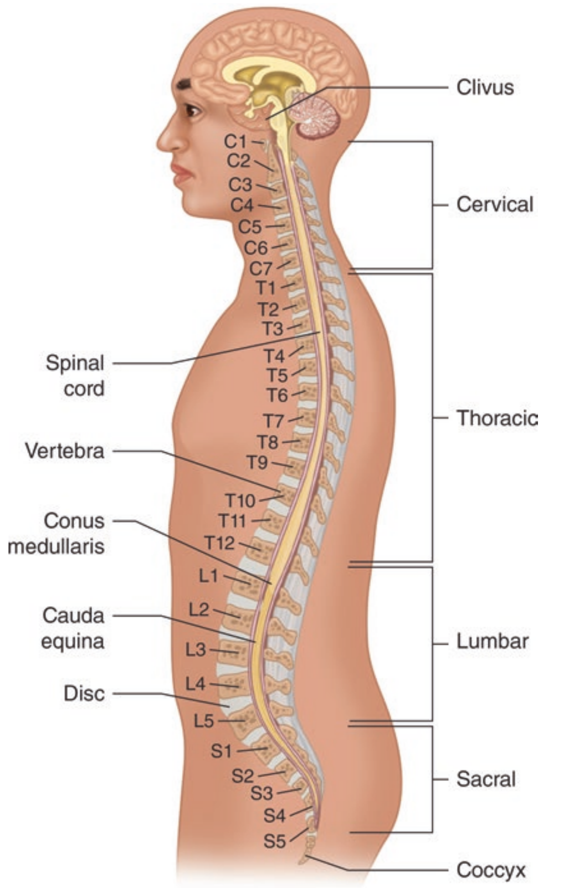

Fig. 31.2 Spinal cord anatomy in relation to the spinal canal

column results in the intervertebral disc being driven into the vertebral body below, resulting in retropulsion of the vertebral body cortex into the spinal canal. Fractures are generally seen in the lower thoracic spine (T9–T12). These are often seen in a fall from height or in motor vehicle crashes and have been reported in sports such as skiing or snowboarding. The patient will likely present with back pain or neurologic symptoms [9].

Type C injuries may be without translation (C1) or with translation (C2 and C3), which are otherwise called fracture-dislocations. These injuries are fortunately rare in sports but can occur with high-energy traumas [3, 6]. Flexion and rotational forces cause bony and ligamentous disruption and displacement of the vertebra. These fracture-dislocations frequently cause significant neurologic sequelae requiring urgent surgical stabilization [8].

Spinous process fractures (Figs. 31.3 and 31.8) are also rare events resulting from a forceful hyperextension or a direct blow to the spinal column. They have been reported in multiple sports. These injuries, also referred to as clay-shoveler's fractures, are most commonly seen in the upper thoracic spine T1–T3 [10–16].

Transverse process fractures (A0) are uncommon and often missed on plain radiography (Figs. 31.3, 31.7, and 31.9).

Epidemiology

In most studies thoracic and lumbar injuries are reported together so there is limited information about the occurrence of thoracic fractures alone. Thoracolumbar spine injuries account for about 5% of direct catastrophic injuries in high school and college sports during the 2015–2016 school year [17]. Studies in pediatrics have reported approximately 35% of fractures occurred in the thoracic spine, but these cohorts contained both sports- and non-sports-related causes [18, 19]. Injuries are more common in children older than 9, ranging from 7% to 21% of reported injuries [18, 19]. A review of thoracic and lumbar fractures in skiers and snowboarders found 40% of fractures in the thoracic spine although overall risk of spinal fracture was low, 0.009% per skier/snowboarder day – combined thoracic and lumbar fractures [3]. Football is the most commonly reported sport for spine fractures, and they can also be seen in diving, hockey, and soccer [18]. Mountain biking has a reported rate of 13% for combined thoracolumbar fractures [6]. Compression injuries are the most commonly reported [3, 18, 20]. Isolated spinous process fractures may also occur in skiing and snowboarding as well as other sports [3, 10–16]. Multilevel injuries may frequently occur as well [21, 22].

Classification

Numerous classification systems have been proposed and used for thoracic spine fractures. Denis developed a classification system in 1983 primarily based on radiographic findings [23]. With the advent of advanced CT imaging technologies, Magerl et al. introduced a complex system based upon increasing injury pattern and instability [24]. This is the Arbeitsgemeinschaft für Osteosynthesefragen (AO) spinal injury classification system that is most commonly used [24–26]. There are three main classifications under this system with several subtypes (Fig. 31.3).

With multiple subclassifications this system was found to have low reproducibility among health providers and to be inefficient for everyday usage [26]. Recently, the Spine Trauma Study Group, composed of spine surgeons from several worldwide trauma centers, developed the Thoracolumbar Injury Classification and Severity (TLICS) score which assesses injuries based on injury morphology, neurologic status of the patient, and integrity of posterior ligamentous complex (PLC) [25–27]. Each of these three variables is assigned points based upon the severity of injury with higher points indicating worse mechanism, neurological status, or integrity of PLC (Table 31.1).

Injury morphology is divided into three subtypes of increasing severity: compression, rotation/translation, and distraction injury. A burst fracture is a qualifier of a compression

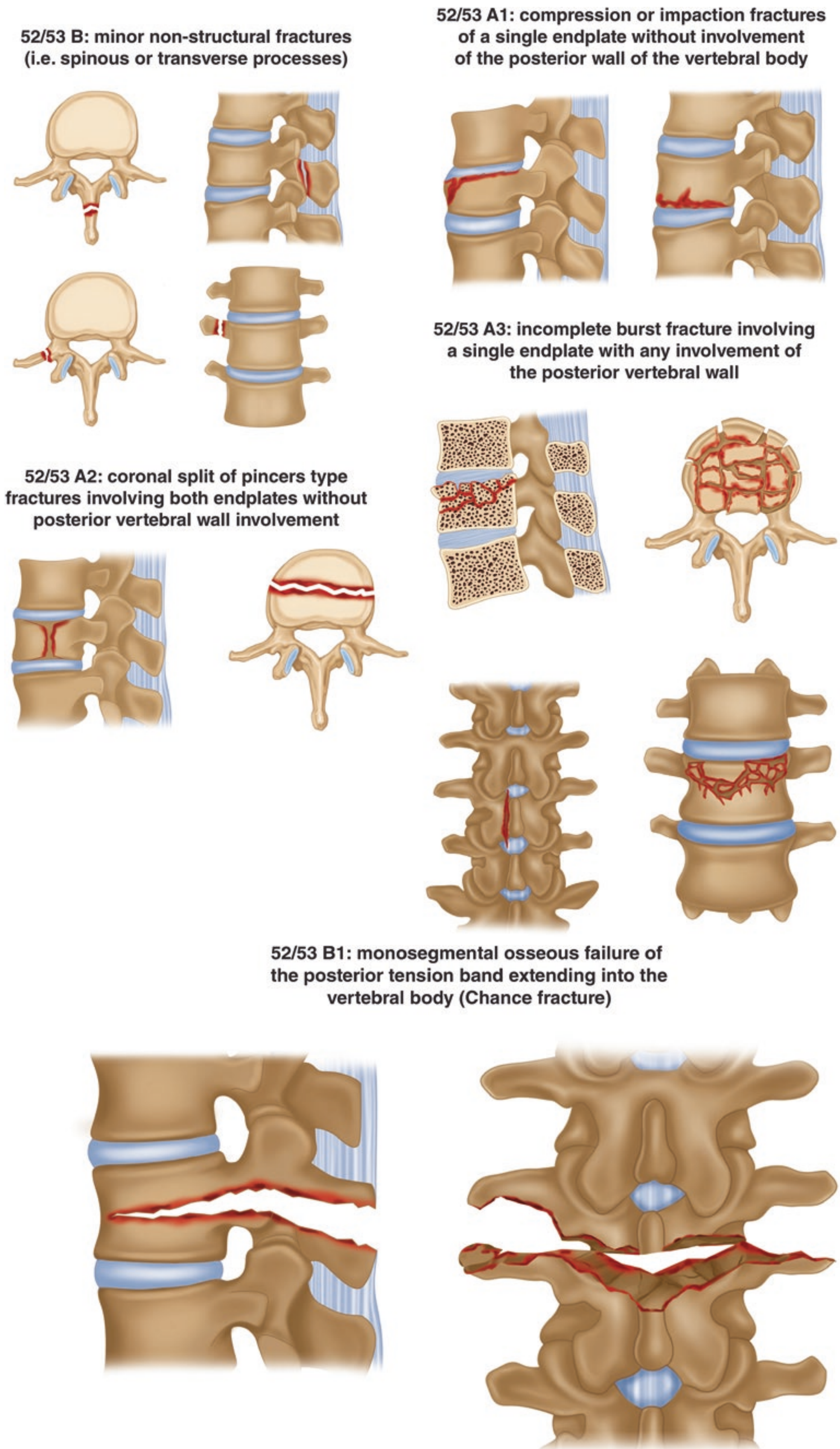

Fig. 31.3 Illustrative depiction of the examples of the AO/OTA (AO Foundation/Orthopaedic Trauma Association) classification system for thoracic or lumbar spine fractures. (Adapted from [7])

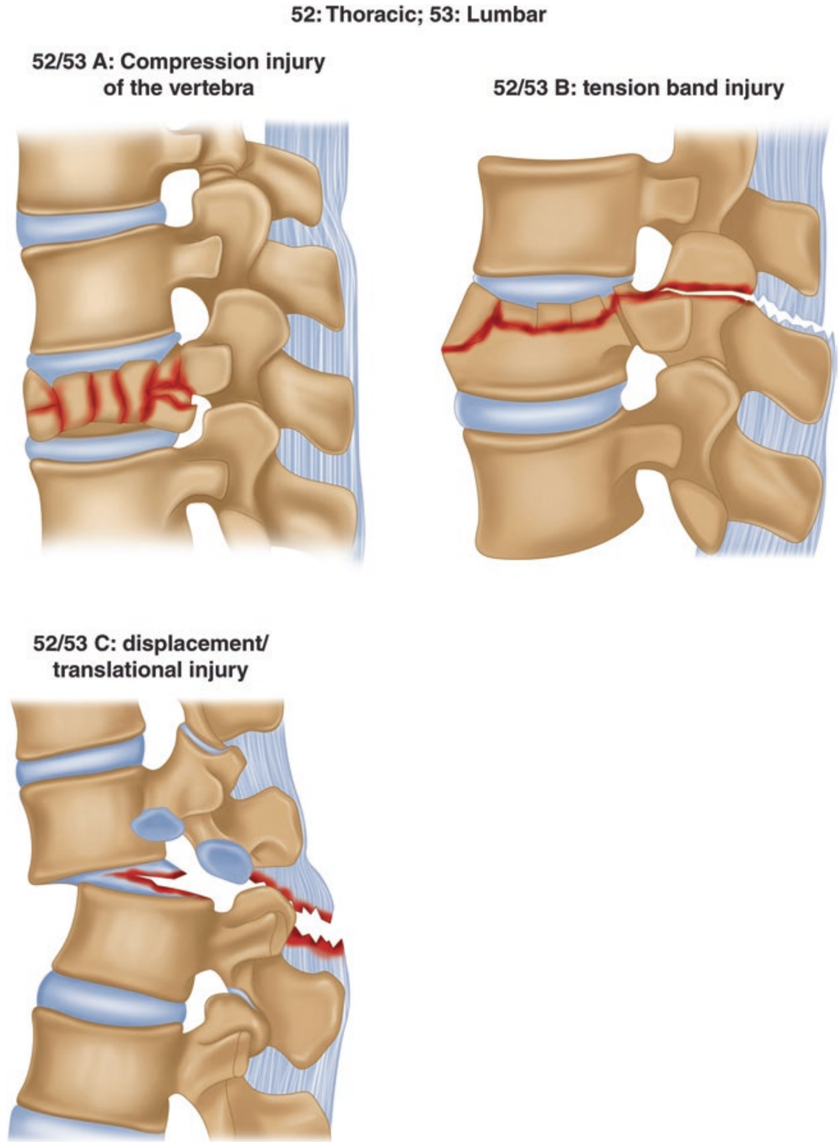

Fig. 31.3 (continued)

fracture with an additional point given for disruption of the middle column [27–29]. The patient's neurological status is a critical indicator of the degree of spinal instability. It is again described in terms of increasing urgency from neurologically intact to cauda equina or incomplete spinal cord injury. The PLC is a critical component in providing stability to the spine and protecting the spinal cord. Assessment of the integrity of the PLC can be assessed on clinical examination with a palpable gap or step-off of the spinous processes or on diagnostic imaging consisting of plain radiographs, CT, or MRI. When the evidence of disruption is subtle, the PLC integrity is either suspected or indeterminate [25–27].

A final score is calculated by adding the three variables together, which is used to help determine appropriate treatment. The higher the aggregate score of the three variables, the more unstable the fracture. A total score greater than 4 is suggestive of surgical intervention for significant fracture instability, whereas a score less than 4 is indicative of nonsurgical treatment [25–27]. Several studies have shown good reliability and reproducibility for classifying the injury and in guiding appropriate treatment in both adults and pediatric patients [27–29]. The validity of the TLICS was lower in pediatric patients when compared to adults, but this was thought to be due to differences in treatment options for the pediatric patients [29]. There is no separate classification system for spinous process injuries. These injuries are believed to occur from forceful muscle contraction or direct trauma. The most common levels seen are T1–T6 [10].

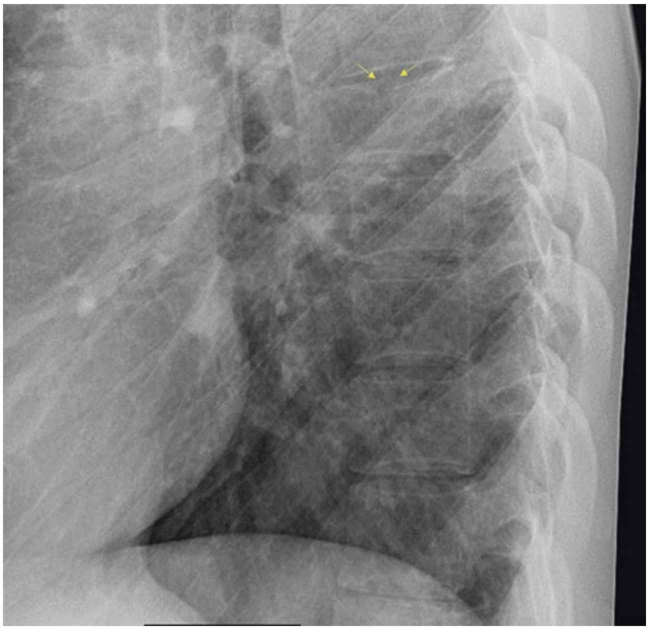

Fig. 31.4 A T7 superior endplate fracture in a 32-year-old male as a result of a mountain biking injury (arrows)

Clinical Presentation

Thoracic spine injuries usually present with pain at level of the injury and may be associated with muscle spasm. Pain with compression injuries is usually worse with flexion. Spinous process injuries are painful to palpate at the level of the injury. Associated symptoms such as shortness of breath or abdominal pain should prompt evaluation for other internal injuries. More severe injuries will present with pain and tenderness at the affected level as well as neurologic symptoms and signs specific to the level of nerve root or spinal cord involvement.

Diagnosis

Plain AP and lateral radiographs are appropriate initial screening for acute thoracic trauma [8]. If radiographs are negative, but a high clinical suspicion remains, a follow-up CT should be pursued. CT is particularly valuable for evaluating the posterior column and can detect subtle

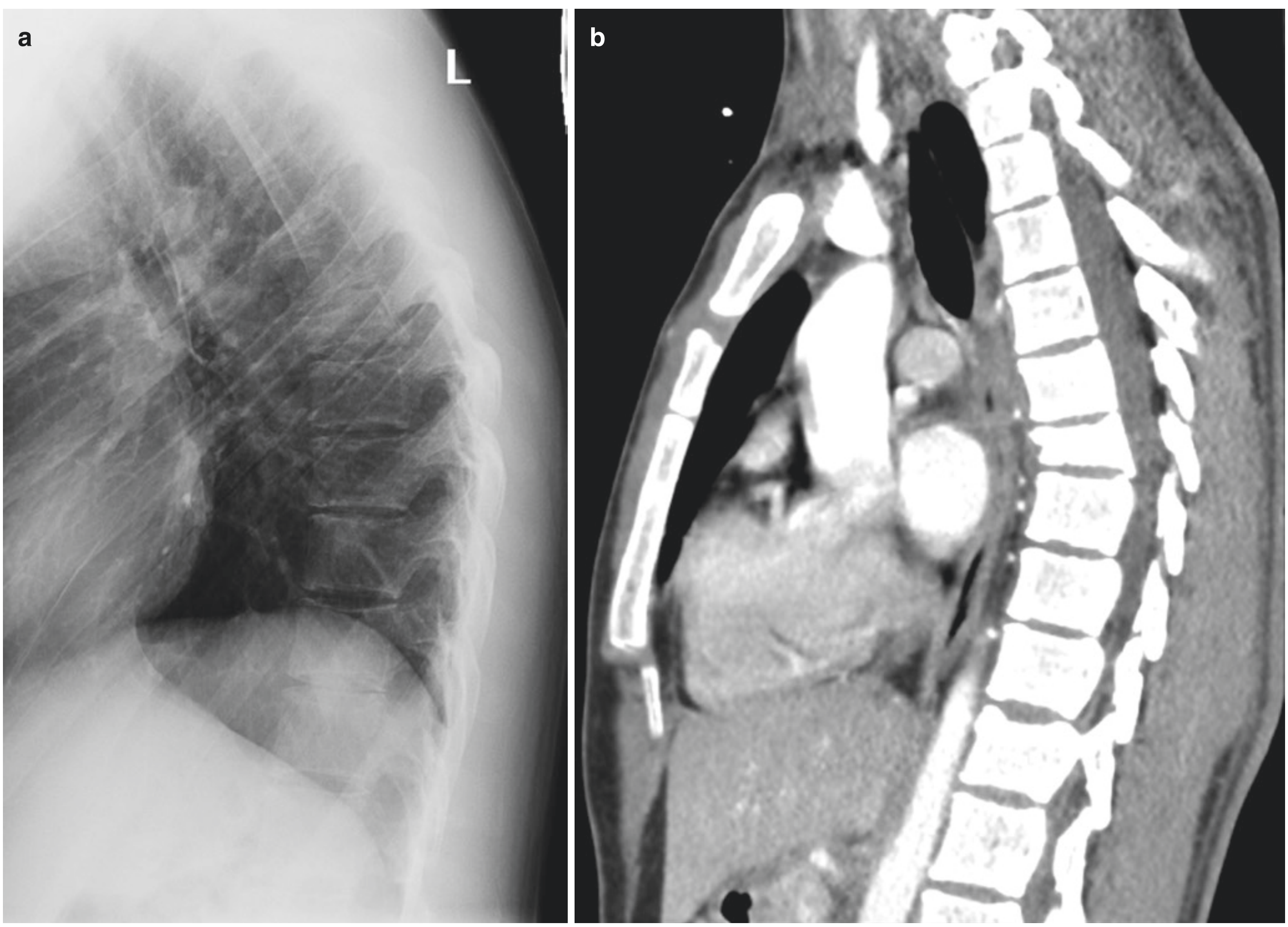

Fig. 31.5 A T7 burst fracture in an 18-year-old male as a result of a mountain biking injury apparent on plain radiography (**a**) and CT (**b**) images

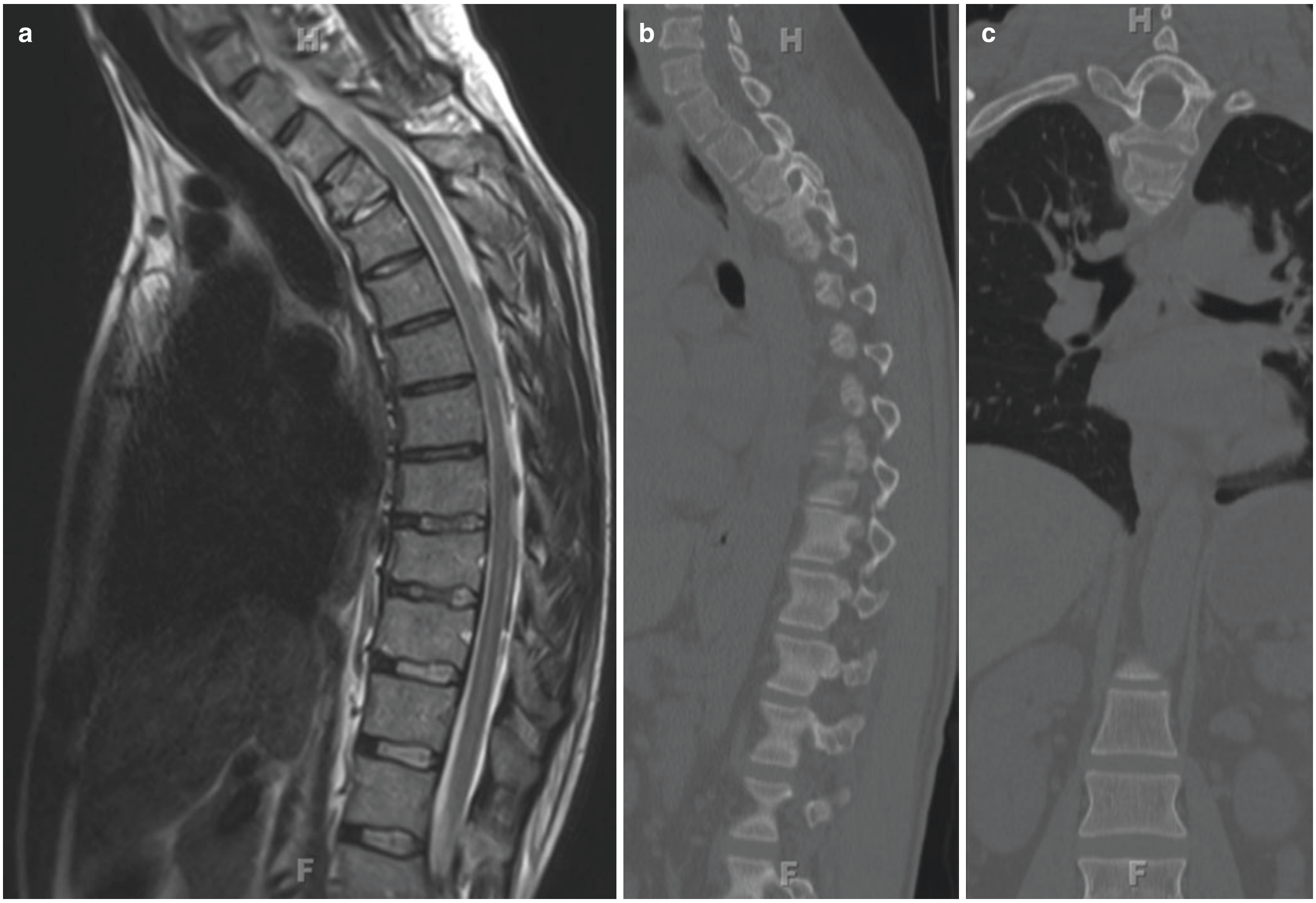

Fig. 31.6 A T3 body fracture in a 38-year-old male as a result of a mountain biking injury apparent on MR (**a**) and CT (**b**, **c**) images

Fig. 31.7 A T5–T6 burst fracture in a 28-year-old male as a result of a mountain biking injury difficult to see on plain radiography (**a**). CT (**b**, **c**) images demonstrate the fracture pattern. CT (**d**) also revealed left T3–T7 transverse process fractures (only T3–T5 fractures are visible on this cut)

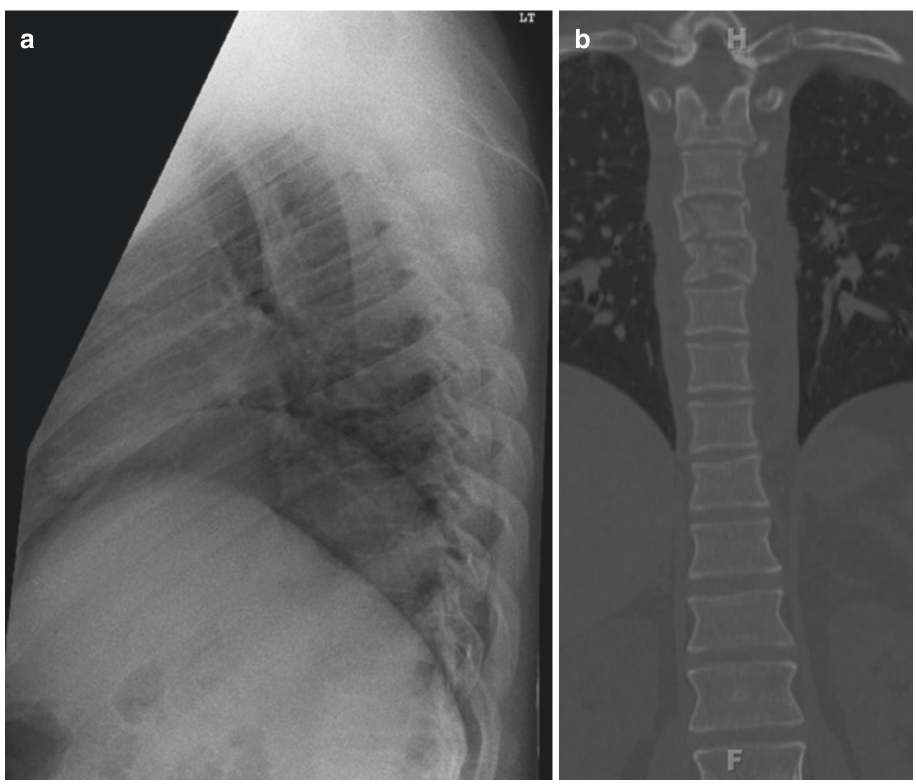

Fig. 31.7 (continued)

occult fractures. MRI is less routinely used in acute trauma, although this has changed some with the advent of the TLICS (Table 31.1) [8]. MRI in this setting should be used to detect injury to the posterior ligamentous complex (PLC) rather than other disc or soft tissue injuries as MRI has not been shown to change management in neurologically intact patients [8, 30].

Because of the associated risk of intra-abdominal/pelvic pathology in high-energy lumbar spine injuries, providers should consider imaging evaluation. Focused assessment with sonography for trauma (FAST) scanning may be used to evaluate for intra-abdominal injuries with good sensitivity and specificity [31]. CT scan is the gold standard and may be used when the FAST scan is positive or not available or indicated [31].

Initial Management

Although thoracic spine injuries with associated neurologic dysfunction are rare, providers must be prepared to evaluate and manage these injuries. There is a wide spectrum of potential fractures of the thoracic spine that can occur in the athlete, ranging from minor spinous process fractures to significant thoracic fracture-dislocation with complete spinal cord injury. A thorough history needs to be obtained with regard to mechanism of injury, location, pain, and neurologic symptoms, bowel and bladder symptoms, and any prior history of thoracic injury. A thorough physical and neurological examination needs to be performed. In high-energy injuries, there is often associated intra-abdominal pathology [8]. Secondary evaluation should also include investigation for organ and vascular injury. This emphasizes the need for a comprehensive evaluation (i.e., ATLS protocol) in a patient with concern for spinal trauma. Please see Chap. 4 for details on stabilization of the unstable patient, including equipment management.

Treatment options for thoracic spine fractures vary based on type and associated injuries. Treatment of thoracic spine fractures is summarized in Table 31.2.

When the TLICS score is <4, nonoperative treatment is recommended in most circumstances. These fractures com-

monly include isolated spinous process and compression fractures. There are several case reports in the literature of athletes sustaining spinous process fractures [10–16, 32]. All of the athletes returned to their respective sports, after a temporary period of immobilization and rest. Rarely nonunion can be seen and if symptomatic excision may be helpful [11].

Compression injuries have been traditionally treated with bracing (Jewett T6–T12 or Minerva above T6), but several recent studies have shown excellent outcomes with early mobilization without bracing in both children and adults [33–35]. Bracing however may be used for comfort in the early phases of treatment.

Other common sports fractures that are treated nonoperatively include neurologically intact burst fractures. Burst fractures involve the anterior and middle columns resulting in retropulsion of the fragments into the spinal canal. The retropulsed fragments may result in varying degrees of stenosis, which may present as a neurologic deficit. The risk of neurologic damage in the thoracic spine is greater due to the presence of the spinal cord and the decreased space available for the spinal cord from T2 to T10 [33]. More commonly thoracic burst fractures are typically encountered at the thoracic-lumbar junction, as this represents a transition region of the spinal canal from the increased stability of the thoracic spine to the more mobile lumbar spine. This area also transitions from the kyphotic thoracic spine to the lordotic lumbar spine. Burst fractures at the thoracolumbar junction lose the stability provided by the rib cage and are subjected to greater bending loads and are more likely to develop kyphosis [33].

Neurologically intact burst fractures of the thoracic spine are also traditionally treated with braces similar to compression fractures to stabilize the spine by decreasing the loads across the spine and limiting motion [33]. However, recent studies have also shown good outcomes with or without bracing [36]. Patient with a score of 4 may be considered for conservative or surgical management based on individual patient and provider [8, 32–37]. For patients with a TLICS score >4, surgery is often indicated due to the instability of the fracture pattern and possible neurologic deficit. The goal of surgical treatment for thoracic spine fractures is to restore the mechanical stability of the spine and to decompress the spinal cord and nerve roots to prevent neurological loss or deterioration [8, 32–37].

Indications for Orthopedic Referral

Patients with stable compression or spinous process injuries can generally be managed without consultation. Stable burst fractures may also be managed conservatively, and referral should be based on provider comfort and experience. Fractures with associated neurologic injury and athletes with TLICS scores ≥4 should be considered for referral to orthopedic or neurosurgery [8].

Follow-Up Care

Few randomized studies have been published comparing the efficacy of the different surgical techniques, and therefore evidence-based guidelines for the treatment of thoracic spine fractures are lacking. A recent systematic review of the literature with respect to the surgical approach for thoracic spine fractures found that none of the surgical methods maintained the initial corrected kyphosis angle [38]. However, the study did find that patient outcomes independent of surgical technique and approach were better than expected with respect to pain and future employment [38]. For stable injuries there are no evidence-based recommendations for follow-up care. Spinous process fractures are managed symptomatically and may only need follow-up if symptoms are persistent beyond 4–6 weeks. Compression fractures should be followed with clinical evaluation every few weeks until pain resolves. Repeat radiographs are generally not necessary as imaging findings will persist longer than clinical healing. Athletes with more significant injuries should be managed on an individual basis with provider experience and preference playing a large role in determining follow-up intervals and necessary imaging.

Return to Sports

No specific guidelines exist for return to play after thoracic spine injuries. All recommendations are based on expert opinion and no studies exist on risk of recurrent injury. Basic principles of resolution of pain, full motion, and strength should all be considered basic requirement for any injury. Spinous process injures can generally return to play as tolerated with other stable injuries monitored closer to ensure full healing prior to return to play. In athletes requiring surgery, single-level fusion may be considered for return to play, but multilevel fusion is generally considered a contraindication to return to contact/collision sports as is evidence of persistent instability [39]. Injuries with permanent neurological deficits or surgery need particular attention and individualized plans for appropriate sport activity after these injuries [20].

Complications

Persistent pain and residual neurologic dysfunction are the most commonly reported complications after thoracic spine

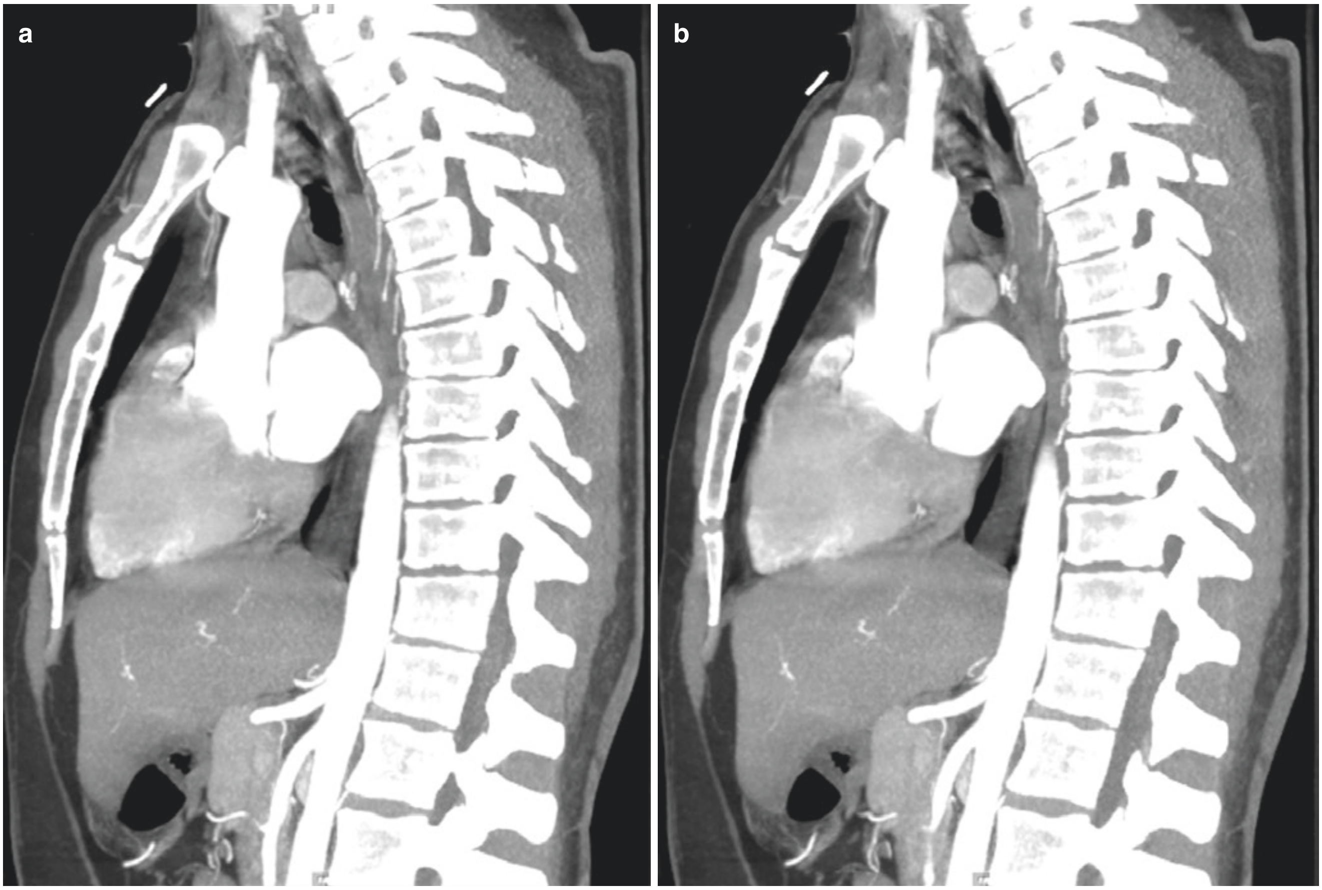

Fig. 31.8 A T3–T6 and T8 spinous process fracture in a 28-year-old male as a result of a snowboarding injury visible on CT images (**a**, **b**)

Fig. 31.9 A right T1 transverse process fracture (arrow) in an 18-year-old male as a result of a mountain biking injury

Table 31.1 Thoracolumbar Injury Classification and Severity (TLICS) score [25–27]

Morphology	Compression Burst Translation/rotation Distraction	1 1 3 4	Imaging X-ray CT
Neurological status	Intact Nerve root injury Incomplete spinal cord injury Complete spinal cord injury Cauda equina syndrome	0 2 3 2 3	
Posterior Ligamentous Complex integrity	Intact Suspected/indeterminate: some MRI signal in interspinous ligaments Disrupted: widening of the interspinous space	0 2 3	Imaging MRI

fractures [8]. Stable fractures tend to do well overall with or without surgical intervention with good reported outcomes [8]. Outcomes with unstable injuries vary greatly mostly based on degree of neurologic injury. Data on return to sports is limited but up to 70% of surgically treated unstable injuries return to previous work levels [8].

Pediatric Considerations

The pediatric thoracic spine has increased flexibility and other anatomical differences that may change patterns of injury. Vertebral bodies are normally wedge shaped in children and this should not be confused with compression inju-

Table 31.2 Lumbar spine fracture treatment based on TLICS score [8, 32–37]

Score	Treatment	Notes
<4	Nonoperative Bracing – if needed 8–12 weeks	Spinous process, transverse process, and minimal compression fractures require symptomatic treatment only
4	Nonoperative Operative considered	Individual patient and injury may determine consideration for surgical intervention
>4	Operative	Surgical intervention based on injury pattern and provider preference

ries. Children 8 years old and older tend to have more typical adultlike fracture patterns. Younger children are more prone to multilevel compression injuries. In distraction-type injuries, the vertebral physis may be injured. Both these types of injuries tend to do well with brief immobilization [18, 19]. Pediatric patients may also present with soft tissue avulsion spinous process injuries [32].

Soft Tissue Injuries

Injuries to the soft tissue of the thoracic spine include muscle injuries, contusions, strains, and ligament sprains. These injuries may occur from direct impact or from hyperflexion or hyperextension similar to whiplash injuries of the cervical spine [40]. While specific data on athletes is lacking, persistent thoracic pain after whiplash injuries is common [40, 41]. Reported injuries and dysfunction include changes in thoracic posture and mobility, myofascial pain and trigger points, and neuromuscular dysfunction of thoracic spine muscles most commonly trapezius [40]. These injuries are usually diagnosed after excluding other traumatic causes of spinal injury such as fracture and may present with similar findings of pain, limited mobility, and sometimes bruising and swelling. Imaging with both radiographs and advanced imaging (e.g., CT scan) are used to rule out underlying fractures [8]. MRI is usually limited to assessment of stability in thoracic fractures and those with neurologic symptoms and signs, although its role has been questioned [42]. The value of MRI in soft tissue injuries has not been established. Most injuries can be treated similar to other soft tissue injuries with early symptomatic treatments with ice, heat, and NSAIDs. Data specific to thoracic injuries is lacking, but in cervical whiplash the role of physical therapy, thoracic manual therapy, and other treatments beyond early mobilization is not well established, and their use can be determined by the individual athlete and provider [41]. Relative rest and gradual return to functional activity may be sufficient in many cases. No specific timeline for return to play is known, but athletes who are not progressing in the first few weeks or have significant persistent pain beyond 4–6 weeks may be considered for referral to a spine specialist.

Spinal Cord Injuries

Injuries to the thoracic spine are the second most common cause of spinal cord injuries, approximately 15–35% [43, 44]. Unlike the cervical spine where spinal cord injuries without fracture may occasionally occur, injuries in the thoracic spine almost always are associated with fractures [44]. Emergency action plans should include spinal precautions and knowledge of local and regional resources for spinal cord injury. Treatment includes stabilization of complicating factors such as hypothermia and hypotension. Adjunctive treatments such as intravenous corticosteroids and hypothermia are not indicated in most cases [45, 46]. Complete spinal cord injuries, American Spinal Injury Association (ASIA) A, to the thoracic spine have minimal chance of neurologic recovery, whereas incomplete spinal cord injuries (ASIA B, C, or D) have a better outcome [37].

The timing of surgical intervention is widely debated. Although animal studies have shown neurological improvement when decompression is performed within 6 h of injury, no human studies have demonstrated improved neurologic recovery with early surgical decompression [47]. Ideally, surgery should be performed within 24–72 h post-injury to allow for mobilization and to prevent further complications associated with immobility, such as decubitus skin ulcers and pneumonia [37]. One absolute indication for emergent surgery is a documented neurological deterioration [37]. See Chap. 30 for more details on the management of spinal cord injury.

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Lumbar Spine

32

Catherine Mygatt Naden, Stephen Huang, and Douglas Comeau

Key Points

- Low back pain in athletes is exceedingly common, approaching 80% lifetime prevalence.
- Acute lumbar spine injuries, particularly spinal cord injuries, are rare.
- Radiographs are appropriate initial screening in both acute and chronic lumbar injuries.
- CT is more commonly used as the follow-up study for acute trauma.
- MRI is indicated for some acute injuries, particularly those with neurologic deficits.
- Although there are no standardized consensus guidelines on return to play following spinal injuries, there is a general consensus that most athletes may return once they have full pain-free range of motion without neurologic or strength deficits.

Introduction

The incidence and prevalence of low back pain in athletes is variably reported in the literature, ranging between 1% and 40%, with nearly 80% lifetime prevalence [1]. Almost 30% experience acute low back pain related to sports activity [2]. The type of injury varies with the mechanism of spinal loading, and thus injury patterns vary with different sports. They range from simple ligamentous sprains or muscle strains to catastrophic injuries resulting in permanent neurologic sequelae such as disc herniation, vertebral fracture, and vertebral subluxation/dislocation. The highest risk of spinal column injury overall is associated with football, rugby, wrestling, ice hockey, skiing, snowboarding, diving, and equestrian sports [2–5]. Chronic overuse injuries are far more common than acute spinal injuries. Low-contact sports like gymnastics, baseball, and rowing have a higher incidence of chronic injuries, while high-speed high-contact sports such as football and snow sports have a higher incidence of acute spinal injuries [1]. Sport-specific injury patterns in the lumbar spine differ somewhat from the overall spinal injury trends, but there is limited comparative data.

C. M. Naden
Ryan Center for Sports Medicine, Boston University, Boston, MA, USA

S. Huang (✉)
Department of Orthopaedic Surgery, John Peter Smith Hospital Sports Medicine, Fort Worth, TX, USA
e-mail: Shuang02@jpshealth.org

D. Comeau
Department of Family Medicine, Division of Sports Medicine, Ryan Center for Sports Medicine, Boston University, Boston, MA, USA
e-mail: douglas.comeau@bmc.org

Anatomy

The spinal column can be divided into the anterior and posterior components (Chap. 31; Fig. 31.1). Anterior components include the vertebral body, each of which is cushioned by the intervertebral disc and stabilized by the anterior and posterior longitudinal ligaments (Figs. 32.1 and 32.2) [6]. The posterior segments include the spinous process, which is attached to the transverse processes via the lamina and forms an arch that is connected to the vertebral body via the pedicles. This arch and the posterior aspect of the vertebral body form the vertebral foramen that houses the spinal cord through L2 and the cauda equina distal to L2 (Chap. 31; Fig. 31.2) [6]. The pars interarticularis connects the superior articular process to the lamina, while the inferior articular process abuts the inferior edge of the lamina (Figs. 32.3 and 32.4). The posterior components of the spinal column are stabilized by the bony interlocking of the inferior articular process from one vertebra and the superior articular process of its caudal neighbor, as well as the ligaments stabilizing one spinous

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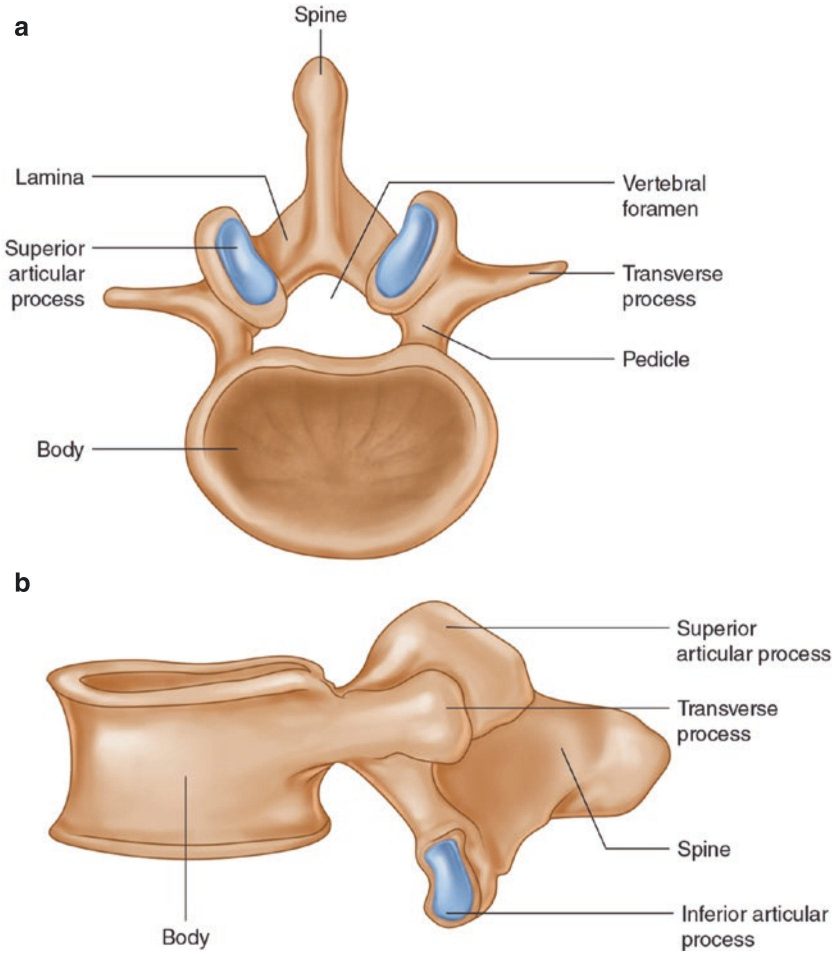


Fig. 32.1 Lumbar spine (similar to lower cervical and thoracic spine) ligamentous anatomy (**a**, **b**)

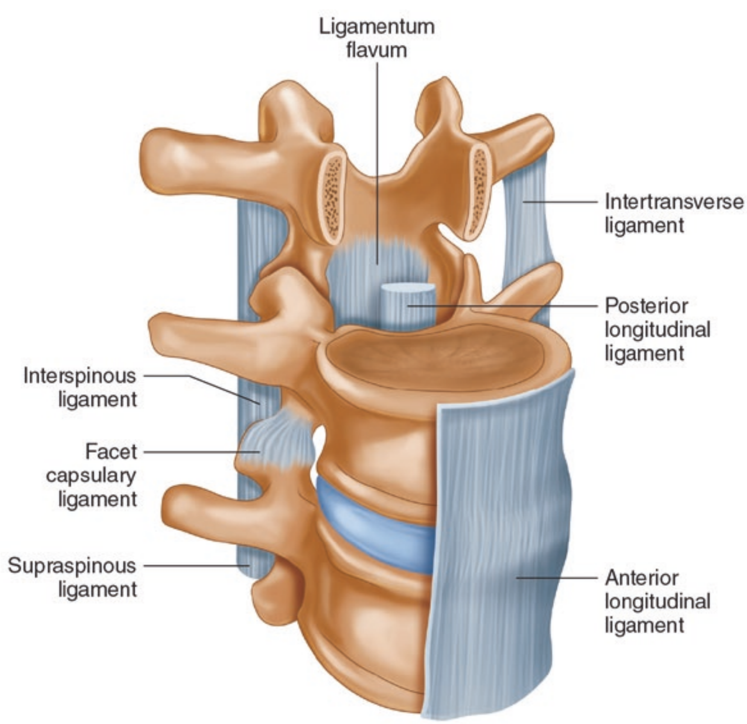
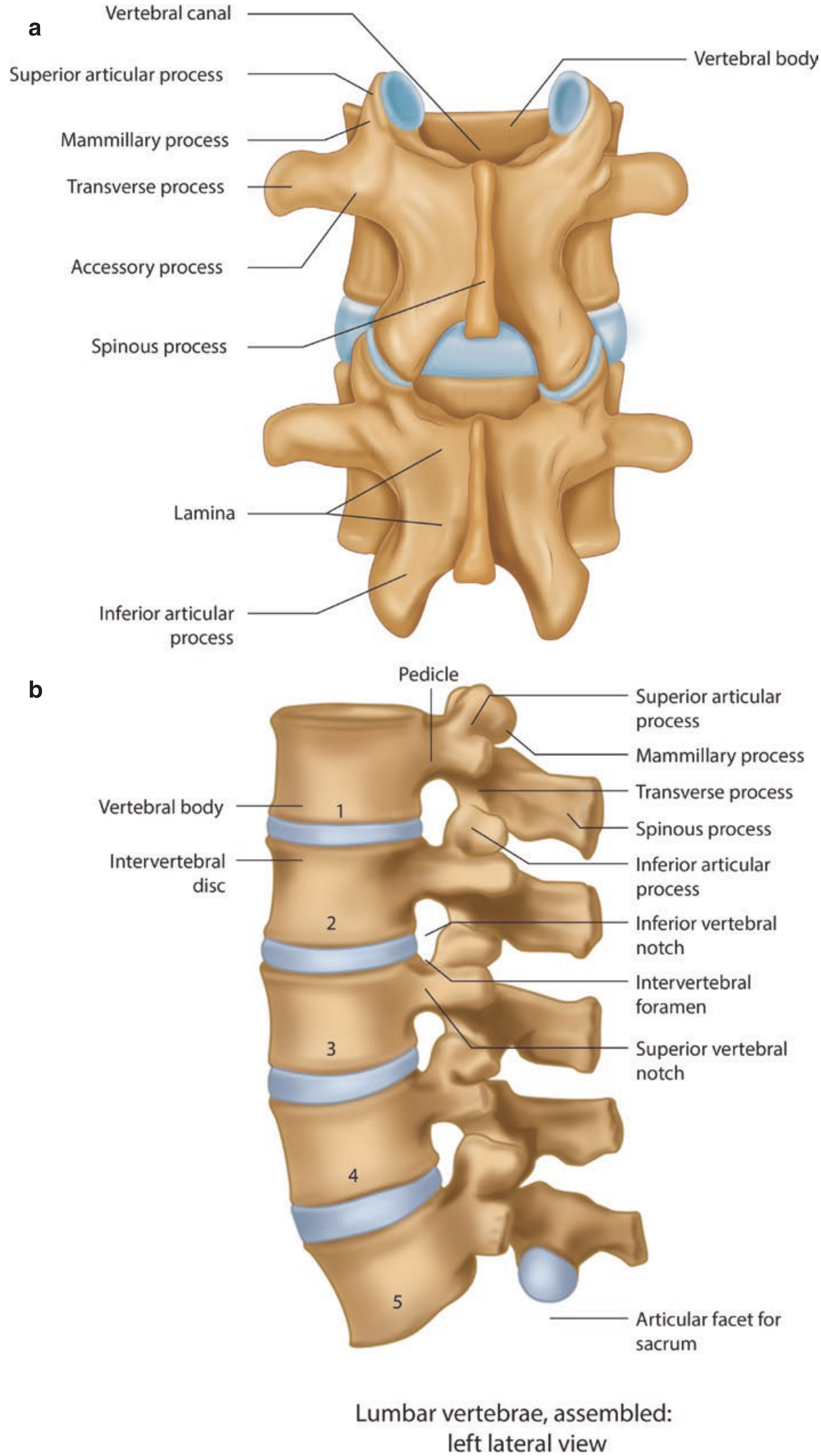


Fig. 32.2 Lumbar (similar to lower cervical and thoracic) vertebra anatomy

Fig. 32.3 Articulation of the lumbar vertebra. Posterior view of the L3–L4 articulation (**a**). Lateral view of the lumbar spine articulation (**b**)



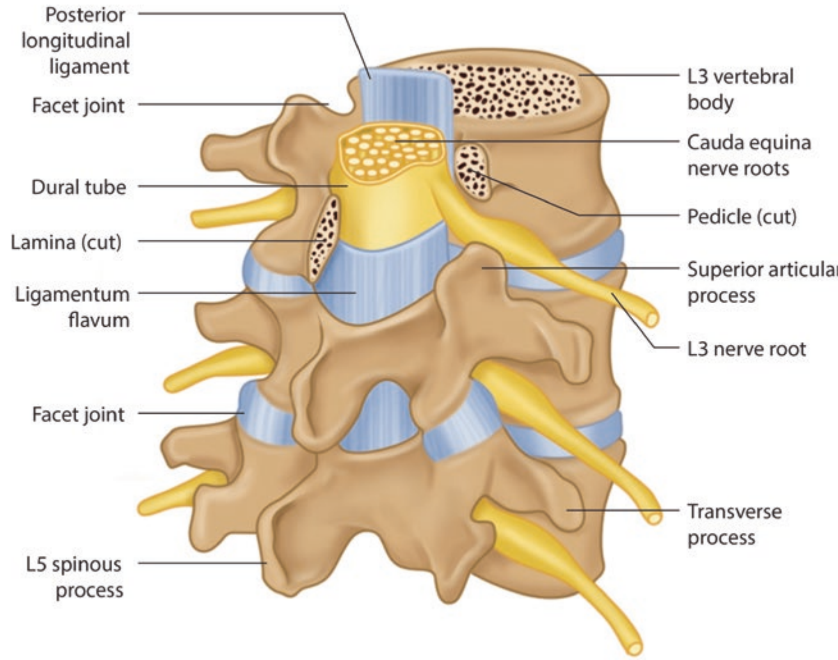


Fig. 32.4 Lumbar (similar to lower cervical and thoracic) spinal nerve anatomy

process on the next [6]. These ligaments include ligamentum flavum anteriorly (within the vertebral foramen), the interspinous ligament between the spinous processes, and the supraspinous ligament posteriorly (Chap. 30; Fig. 30.4) [4]. The lumbar spine is more stable than the cervical spine and less stable than the thoracic spine. This is reflected in the relative incidence and severity of acute pathology in each spinal region.

Fractures

Mechanism of Injury in Sport

The Arbeitsgemeinschaft für Osteosynthesefragen (AO) classification system is the most commonly used classification system for lumbar spine fractures and includes both the type of fracture and mechanism of injury (Chap. 31; Fig. 31.3). As there is a relationship between classification system and mechanism of injury, it is briefly discussed here (see "Classification" section) [4, 7]. Compression fractures (AO Types A1 and A2) (Figs. 32.5 and 32.6) occur in a flexion injury, in which the anterior edge of the vertebral body loses height, while the posterior border of the vertebral body maintains its integrity. These fractures more commonly are seen in older patients with osteoporosis, or after a fall from a height landing in a seated position, resulting in a flexion and axial loading force [8]. Hyperflexion injuries may occur in many sporting settings and have been commonly reported in snowboarding and mountain biking [9, 10].

Burst fractures and fracture-dislocations (AO Types A3 and A4) are the types of fracture most likely to result in neurologic sequelae. In burst fractures, a high-energy axial loading of the vertebral column results in the intervertebral disc being driven into the vertebral body below, resulting in retropulsion of the vertebral body cortex into the spinal canal. This most commonly occurs at L1, with the majority occurring from T9-L5. These are often seen in a fall from a great height, usually with the patient landing on his feet, or in motor vehicle crashes. The patient will likely present with back pain or neurologic symptoms [11].

Type B fractures are usually a result of a flexion-distraction injury (Chap. 31; Fig. 31.3). These fractures can extend to involve all three spinal columns and are called Chance fractures (Fig. 32.7). Chance fractures are considered unstable and have a significant association with intra-abdominal injuries. Neurological complications are relatively uncommon.

Type C injuries may be without translation (AO C1) or with translation (AO C2 and C3), which are otherwise called fracture-dislocations (Fig. 32.8). These generally occur in high-velocity situations with flexion and rotational forces, causing bony and ligamentous disruption that displaces one vertebra off another [4, 5, 7]. Given the location of the spinal cord, these fracture-dislocations frequently cause significant neurologic sequelae and almost always require urgent surgical stabilization.

Avulsion-type injuries such as transverse process fractures are usually categorized under the minor nonstructural fracture category (AO Type A0). Transverse process frac-

Fig. 32.5 A compression fracture of the L2 superior endplate without involvement of the posterior wall end plate (AO Type 53A1) in a 25-year-old male as a result of a skiing injury (**a**). CT images reveal the extent of the fracture (**b**, **c**)

tures occur in settings of extreme side bending or a direct blow to the lumbar spine (Figs. 32.9 and 32.10). They may also occur following a strong contraction of the psoas muscle, which attaches to the transverse processes of the lumbar spine. Transverse process fractures are often quite painful but usually stable. Pain with hip flexion should raise suspicion for this type of injury [12]. They have been observed in horseback riding and football, although they are more commonly seen in motor vehicle collisions and falls from >3 m [13]. They are frequently missed on radiographs and require a high index of suspicion. Transverse process fractures are rare in the general population; studies focusing on particularly high-risk sports, such as snowboarding, however, suggest a higher incidence than commonly reported. In a 2016 study of 8723 snowboarders with injuries, only 4.9% presented with spinal fractures. Of these, 33% (*N* = 143) were transverse process fractures and 25% (*N* = 108) were compression fractures [14]. Spinous process fractures are also rare events resulting from a forceful hyperextension or a direct blow to the spinal column. They are uncommon in the lumbar spine with none being reported in several series [3, 10].

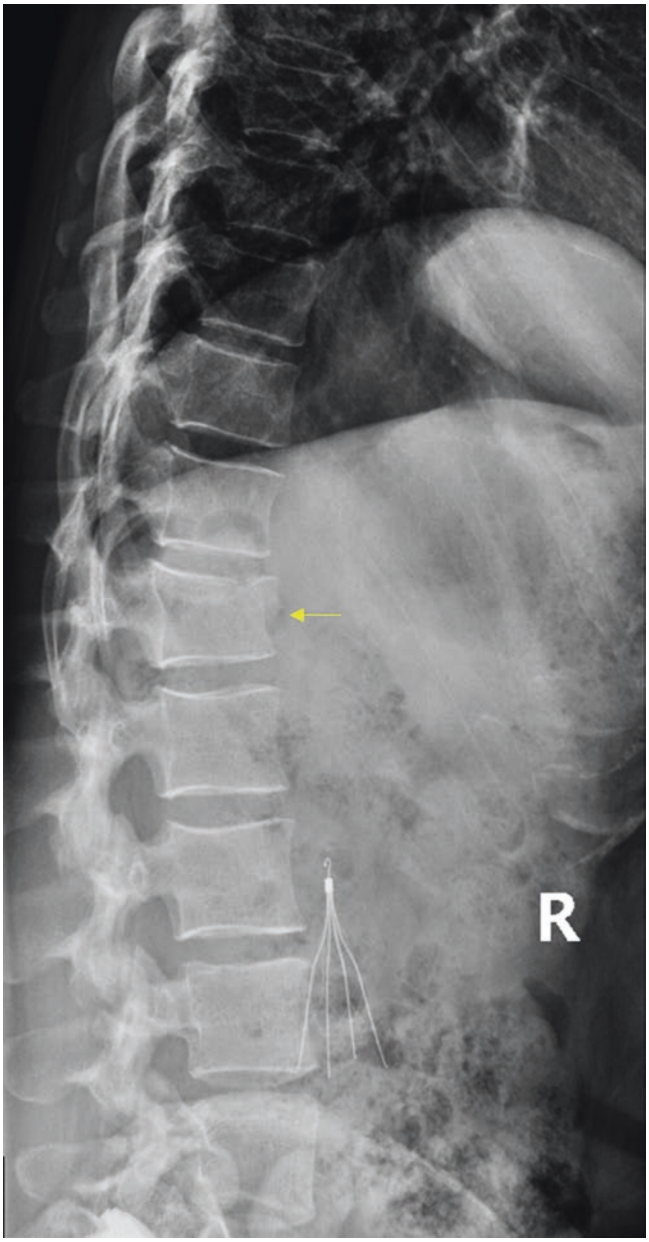


Fig. 32.6 An L1 vertebral body fracture (arrow) in a 55-year-old female. An IVC filter is also present

Epidemiology

Most studies combine thoracic and lumbar injuries resulting in a paucity of statistics on the exact epidemiology of lumbar spine injures. Most injuries occur via motor vehicle accidents (up to 80%) with sports injuries being reported less frequently [7]. Commonly implicated sports include skiing and snowboarding, American football, and rugby, but injuries have been reported in multiple other sports as well [2–5]. In a review of skiing and snowboarding injuries, approximately 60% of fractures were lumbar [9]. Compression fractures were most common followed by transverse process and spinous process fractures. Snowboarders more commonly had compression fractures, and distraction and rotation fractures (AO Types B and C) were rare and occurred only in skiers in this cohort [10]. Compression and burst fractures more commonly occur in higher-energy sporting activities, such as mountain biking [9]. Of note, in high-speed traumatic injuries to the lumbar spine, such as in skiing and snowboarding, injury at two or more levels occurs in about 50% [15, 16]. In a study of American college football, rates of lumbar fracture were reported as 1 per 1,000,000 athlete exposures, and fracture types were not specified [17].

There are no published statistics on the incidence of isolated acute lumbar trauma resulting in "catastrophic injury," defined as an injury producing severe permanent neurologic deficits with a large review not reporting any lumbar injuries [18, 19]. However, based on the following data, one can infer how rare catastrophic spinal injury in sports is. Less than 1% of injuries to the axial spine result in lumbar spine fractures [20]. Overall, according to the National Spinal Cord Injury Statistical Center, there is an annual incidence of 12,000 new cases of any spinal cord injury per year in the United States, approximately 9.2% (1100) of which are related to traumatic sports events [2]. In two of the highest-risk sports for acute lumbar pathology, snowboarding and skiing, only 5% of thoracolumbar fractures are themselves classified as unstable and therefore predispose a patient to neurologic sequelae [14]. The rates of neurological injury are in the range of 1 in 1,000,000 skier days, and complications leading to death are as low as 1 in two million skier days [15, 16]. In a study of skiers and snowboarders by Hubbard et al., isolated spinal injuries occurred in 4.6% of patients who sustained injuries severe enough to be referred to a tertiary care trauma center [21]. Skiers had only a 2.5% incidence of lumbar spine fracture or dislocation overall. Snowboarders had a 4.9% risk of lumbar spine fracture or dislocation. Only 6% of these rare lumbar spine injuries resulted in SCI. However, 55% of these lumbar injuries went on to be treated with surgical fixation, with the likelihood of surgical intervention significantly higher in those with SCI (OR 13.4, 95% CI 4.87–37.25) [21]. In the other sport most commonly cited as high risk for acute lumbar injury, American football, no catastrophic lumbar injuries were observed between 1977 and 2001 [18].

In Hubbard's study, closed head injury most commonly accompanied spinal injuries (13%), and conversely, 13% of all closed head injuries had an associated spinal injury (at any vertebral level) [21].

Disc and ligament injuries may occur in isolation or in conjunction with fractures. Lumbar disc herniation has a reported prevalence of up to 58% in the athletic population [22]. However, the clinical significance of this is unclear, as the presence of disc disease does not correlate with the rate of back pain. Care should be taken when these injuries accompany non-displaced fractures or minimal compression

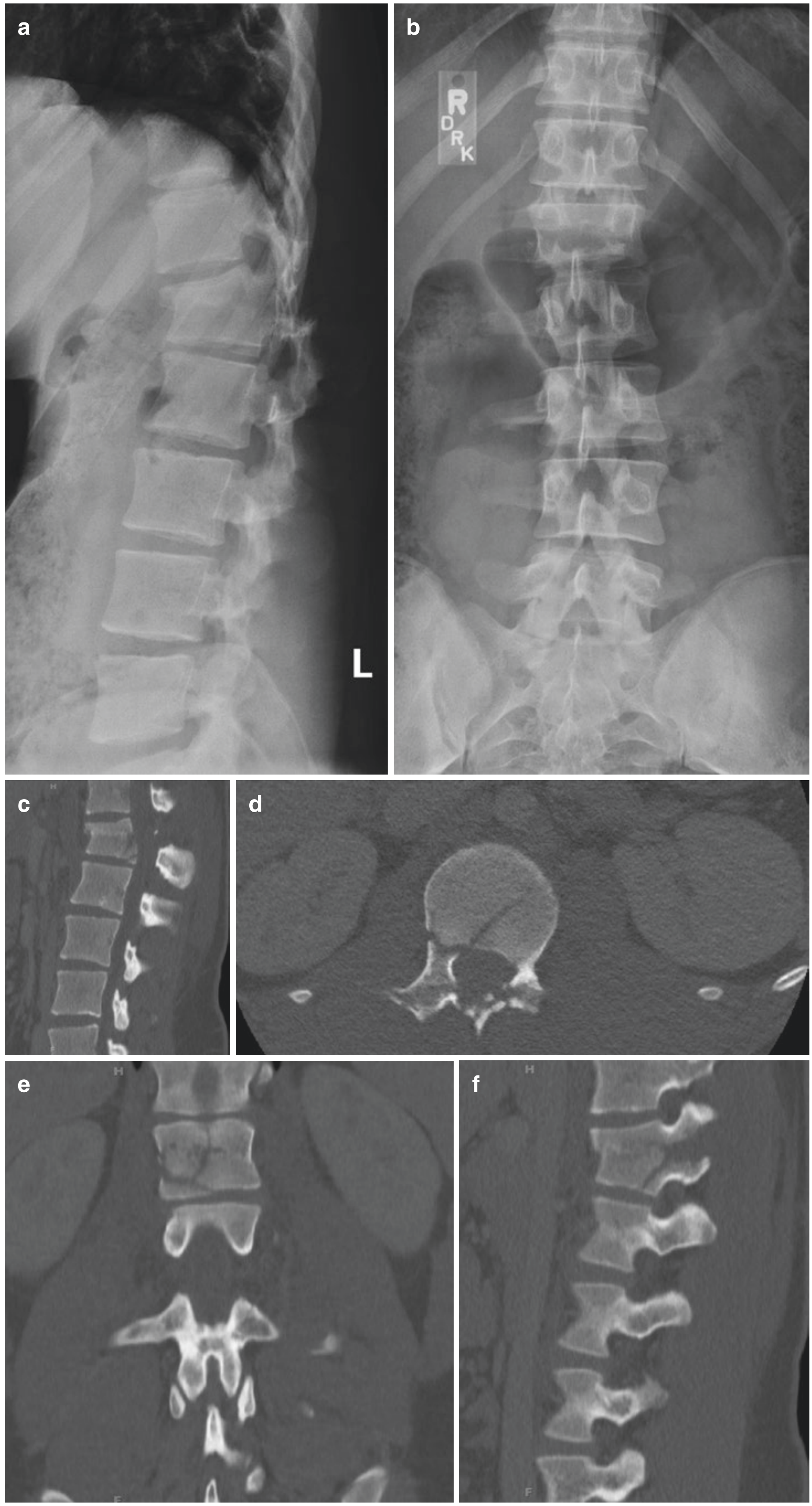


Fig. 32.7 An L1 "Chance" fracture (AO Type 53B1) in a 17-year-old male as a result of a skiing injury (**a**, **b**). CT images demonstrate the extent of the injury (**c**–**f**)

Fig. 32.8 A superior/anterior L2 vertebral body fracture with anterolisthesis of L1 over L2 (AO Type 53C) in a 25-year-old male as a result of a mountain biking injury visible on plain radiography (**a**), CT (**b**), and MRI (**c**, **d**). MRI also shows L1–L2 disc injury without significant spinal cord pressure (**c**, **d**)

injuries as they may have been present before the injury. In a 2009 study by Baranto of 71 male athletes and 21 nonathletes followed for 15 years, there was no statistically significant correlation between back pain and disc changes seen on MRI [23].

Significantly more common than acute bony injuries are stress-related injuries to the pars interarticularis (known as spondylolysis), which is at the junction of the pedicle, articular facets, and lamina. These injuries will sometimes present with sudden onset of pain or with an acute injury,

Fig. 32.9 A left L2 transverse process fracture (arrow) in a 24-year-old male

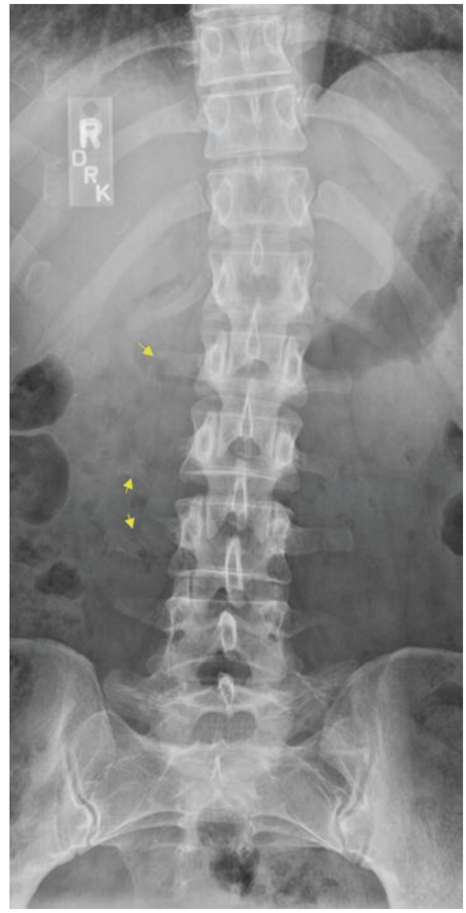


Fig. 32.10 Right L1–L3 transverse process fracture (arrows) in a 30-year-old female as a result of a mountain biking injury

creating confusion about acute injury versus stress injury, so they are briefly mentioned here. One study reported that spondylolysis accounts for 47% of low back pain in the adolescent athlete, followed by discogenic and muscle-tendon strain (20%) [24]. Of note, however, another study of 3000 athletes reported only 7.8% incidence of spondylolysis in adolescents with back pain [25]. Neither study commented on acute versus insidious onset of pain. The majority of spondylolysis (95%) occurs at L5, and the incidence decreases as you move cephalad [26]. The most probable mechanism is repetitive microtrauma resulting from recurrent extension and rotation of the lumbar spine. In a 1986 case study of 253 CT scans of patients with spondylolysis, only 16% were unilateral, but this number varies significantly in the literature [27]. In a 2013 case-control study of 50 patients, 60% of spondylolysis were unilateral [28]. Spondylolysis may result in spondylolisthesis, in which the vertebra is translated anteriorly or posteriorly. Both spondylolysis and spondylolisthesis can be asymptomatic or cause acute or insidious onset back pain and radiculopathy.

Classification

Multiple classification systems have been proposed historically; however, the Arbeitsgemeinschaft für Osteosynthesefragen (AO) spinal injury classification system is most commonly used (Chap. 31; Fig. 31.3) [4, 29, 30]. There are three main classifications under this system with several subtypes (Table 32.1).

While this classification can be useful in describing injuries, it is not specifically prognostic and has only been found to be moderately reproducible between observers [8]. The Thoracolumbar Injury Classification and Severity (TLICS) score can guide treatment options by adding points for each category (Table 32.2). This is discussed further under treatment.

Clinical Presentation

Stable injuries of the lumbar spine generally present with pain and without neurologic complaints.

Table 32.1 AO lumbar spine fracture classification [4, 29, 30]

Type A – Compression/ burst injuries	Axial compression Anterior element injury Posterior elements intact
Type B – Tension band injuries	Injury to posterior elements
Type C – Displacement injuries	Injury to anterior and posterior elements creating displacement of structures

Table 32.2 Thoracolumbar Injury Classification and Severity (TLICS) score [4, 29, 30]

Morphology	Compression Burst Translation/rotation Distraction	1 2 3 4	Imaging X-ray CT
Neurological status	Intact Nerve root injury Complete spinal cord injury Incomplete spinal cord injury Cauda equina syndrome	0 2 2 3 3	Physical Exam
Posterior Ligamentous Complex integrity	Intact Suspected/indeterminate: some MRI signal in interspinous ligaments Disrupted: widening of the interspinous space	0 2 3	Imaging MRI

Tenderness to palpation over the affected vertebrae or spinous process is common. Bruising and swelling may be seen. Compression injuries tend to hurt more with lumbar flexion. Transverse process fractures may hurt with resisted hip flexion due to the attachments of psoas or with lateral bending away from the injured side [12]. Muscle spasm often accompanies injuries with moderate to severe pain. More severe injuries will present with tenderness at the affected level as well as neurologic findings specific to the level of nerve root or spinal cord involvement.

Diagnosis

Despite its rarity, the on-field physician must be prepared to evaluate a patient who has suffered an injury that predisposes them to spinal cord trauma. Although spinal cord injury in isolated lumbar injury is rare, attention should be given to evaluation of the entire spine. The key tenets of acute management of severe spinal injury are (1) an algorithmic approach including pre-event planning, (2) a good understanding of resources available to stabilize and transport an injured athlete, and (3) the ability to stabilize and transport in a timely manner. Please see Chap. 4 for details on stabilization of the unstable patient, including equipment management. It is important to note that in a suspected lumbar spine injury, logrolling techniques should be reserved for initially rolling a prone athlete to facilitate evaluation and stabilization. In all other cases where lumbar pathology is suspected, a six-person lift and transfer or lift and slide technique is preferred as it provides greater stabilization to the lumbar spine [31].

In high-energy injuries, there is often associated intra-abdominal pathology [7]. Secondary evaluation should also include investigation for organ and vascular injury. Almost half of skiers and one third of snowboarders with lumbar injuries had multi-organ trauma [21]. This emphasizes the need for a comprehensive evaluation in a patient with concern for spinal trauma.

Plain AP and lateral radiographs are appropriate initial screening for acute lumbar trauma [7]. If radiographs are negative, but a high clinical suspicion remains, a follow-up CT should be pursued. CT is particularly valuable for evaluating the posterior column and can detect subtle occult fractures. MRI is less routinely used in acute trauma, although this has changed some with the advent of the TLICS [7] (Table 32.2).

The pitfall of MRI use for assessing spinal problems is the well-demonstrated lack of correlation between abnormalities found on MRI and clinical symptoms across all sports. Low back pain is commonly seen in some sports more than others, particularly gymnastics, dance, soccer, football, wrestling, weightlifting, rowing, cricket, ice hockey, and tennis. In a 2009 publication by Baranto, 71 male athletes (weightlifters, wrestlers, orienteers, and ice-hockey players) and 21 nonathletes were evaluated with baseline MRI and then had a follow-up study done 15 years later [23]. Thirty-two of the athletes (45%) and all nonathletes (100%) had disc height reduction at one or several disc levels. Disc degeneration was found in more than 90% of the athletes, and deterioration had occurred in 88% of the athletes, with the highest frequency in weightlifters and ice-hockey players. There was no statistically significant correlation between back pain and MRI changes. Similar findings are abundant in the literature, and caution should be used when interpreting these findings in the setting of traumatic low back pain. MRI in this setting should be used to detect injury to the posterior ligamentous complex (PLC) rather than other disc or soft tissue injuries [7].

Because of the associated risk of intra-abdominal/pelvic pathology in high-energy lumbar spine injuries, providers should consider imaging evaluation. Focused assessment with sonography for trauma (FAST) scanning may be used to evaluate for intra-abdominal injuries with good sensitivity and specificity [32]. CT scanning is the gold standard and may be used when the FAST scan is positive or not available or indicated [32].

Initial Management

Injury classification gives some input into treatment; however, the overall utility is limited.

Table 32.3 Lumbar spine fracture treatment based on TLICS score [7, 33–37]

Score	Treatment	Notes
<4	Nonoperative Bracing – if needed 8–12 weeks	Spinous process, transverse process, and minimal compression fractures require symptomatic treatment only
4	Nonoperative Operative considered	Individual patient and injury may determine consideration for surgical intervention
>4	Operative	Surgical intervention based on injury pattern and provider preference

The TLICS score can be used to guide treatment of lumbar spine injuries (Table 32.3).

The risk of neurologic damage is less in the lumbar spine compared to the thoracic spine due to the anatomy of the spinal cord and the increased space available in the lumbar spine for the distal cord and cauda equina. When the TLICS score is less than 4, nonoperative treatment is the suggested recommendation in the majority of circumstances [7, 30]. Fractures with a low TLICS score usually include isolated spinous process or transverse process fractures, compression fractures, and neurologically intact burst fractures.

Transverse process and spinous process fractures are treated with relative rest and symptomatically for pain control but generally do not require other interventions.

Compression and neurologically intact burst fractures of the lumbar spine can be treated with braces to stabilize the spine by decreasing the loads across the spine and limiting motion [7]. Braces that limit flexion (i.e., Jewett braces) may be used for upper lumbar compression injuries, and various forms of thoracolumbar sacral orthosis can be used for burst fractures. The role of bracing in these injuries has been questioned [33–35].

Lumbar burst fractures are more commonly encountered at the thoracic-lumbar junction, as this represents a transition region of the spinal canal from the relatively stable thoracic spine to the unstable lumbar spine. This area also transitions from the kyphotic thoracic spine to the lordotic lumbar spine. Burst fractures at the thoracolumbar junction lose the stability provided by the rib cage, are subjected to greater bending loads, and are more likely to develop kyphosis [33]. Recent studies have compared bracing versus no bracing for patients with compression and burst fractures. Ohana et al. found that compression fractures with up to 30% of anterior column collapse could be treated with early ambulation and hyperextension exercises and did not need bracing [33]. Similar study for thoracolumbar burst fractures from T10-L3 without neurologic injury found similar results with bracing and non-bracing protocols [34]. Although compression fractures are uncommon in children and adolescents, they do occur in the athletic population, and a recent study demonstrated similar results in the treatment of these fractures without bracing [35].

For patients with a TLICS score greater than 4, surgery is often indicated due to the instability of the fracture pattern and possible neurologic deficit. The goal of surgical treatment for lumbar spine fractures is to restore the mechanical stability of the spine and to decompress the spinal cord and nerve roots to prevent neurological loss or deterioration. The timing of surgical intervention is widely debated. Although animal studies have shown neurological improvement when decompression is performed within 6 hours of injury, no human studies have demonstrated improved neurologic recovery with early surgical decompression [36]. Ideally, surgery should be performed within 48–72 hours post-injury to allow for mobilization and to prevent further complications associated with immobility, such as decubitus skin ulcers and pneumonia [37]. One absolute indication for emergent surgery is a documented neurological deterioration [37].

Indications for Orthopedic Referral

Athletes with unstable fractures should be transported to a facility with higher level of care capable of managing these patients. Fractures with associated neurologic injury and athletes with TLICS scores equal to or greater than 4 should be considered for referral to orthopedic or neurosurgery [7].

Follow-Up Care

For stable injuries there are no evidence-based recommendations for follow-up care. Spinous process and avulsion injuries do not generally require repeat radiographs unless pain is persistent beyond 4–6 weeks. Because radiographic findings of compression injuries may be persistent well beyond clinical healing, repeat radiographs are generally not repeated unless clinical progression arrests. Individualization is recommended as some athletes may only need education and as needed follow-up, i.e., small spinous avulsion, while others may need closer follow-up care.

Follow-up for more severe injuries is based on the injury and need for surgery and is also individualized. For athletes with TLICS injury score of 4 in whom conservative treatment course is planned, there is no recommended follow-up interval. Providers should consider weekly follow-up initially to monitor neurologic status.

Return to Sports

There are no standardized consensus guidelines regarding return to play after spinal injuries including both fractures with and without neurologic injury and isolated disc and ligamentous injuries. There is general consensus that athletes should have full pain-free range of motion, with full and symmetric strength, and no neurologic deficits to be able to return to the sport. Furthermore, multilevel (two to three levels) spinal fusions are an absolute contraindication for return to contact sports [38]. Following lumbar percutaneous discectomy and microdiscectomy, return to contact sports after 2–6 months is plausible and 4–8 weeks for lighter non-contact activities. Return to play criteria after spine surgery and bony injury are provider-specific and lack evidence and consensus [38].

Complications

The most common complication after lumbar spine fractures is persistent pain [7]. Residual neurological deficits may persist as well with the degree of disability based on the individual injury. Almost three quarters of patients are able to return to work-related activities but data on athlete return to sport is limited [7].

Pediatric Considerations

Pertinent to the pediatric population is the rare apophyseal ring avulsion fracture (Fig. 32.11) [39]. This may be misdiagnosed as a lumbar disc herniation or may coincide with a disc injury. The apophyseal ring begins to ossify at 5 years and completes ossification by age 18. This area is susceptible to avulsion injuries particularly during peak growth velocity in adolescents. These fractures present like lumbar disc herniations, with low back pain and potential radicular symptoms, and may even be associated with claudication, paresis, and cauda equina syndrome [39]. Physical examination commonly reveals a restricted lumbar range of motion, with a potentially positive straight leg raise, although other frank neurological deficits are rare [5, 39]. CT images are the most sensitive modality for diagnosing this condition. Conservative management may be pursued in the absence of neurological deficits. If pain is persistent and is felt to be due to the avulsion injury, surgery can be considered. Surgery consists of a laminectomy and excision of the fractured fragments rather than fixation, while the disc should be spared if possible [5–39].

Soft Tissue Injuries

Back pain is very common in athletes and most is reported to be mechanical or nonspecific [6, 40]. Acute soft tissue injuries such as contusions, muscle strains, and ligamentous injuries, however, do occur and represent up to 10–15% of reported injuries across multiple sports [40, 41]. Injuries may occur with both contact and non-contact mechanisms with direct blows causing mainly contusions and hyperflexion or hyperextension injuries causing strains and sprains [6]. These injuries may range from minor pain and no limitations to severe symptoms limiting practice and play. Athlete may present with pain, limited motion, muscle spasm, and bruising. Diagnosis is based on physical examination with

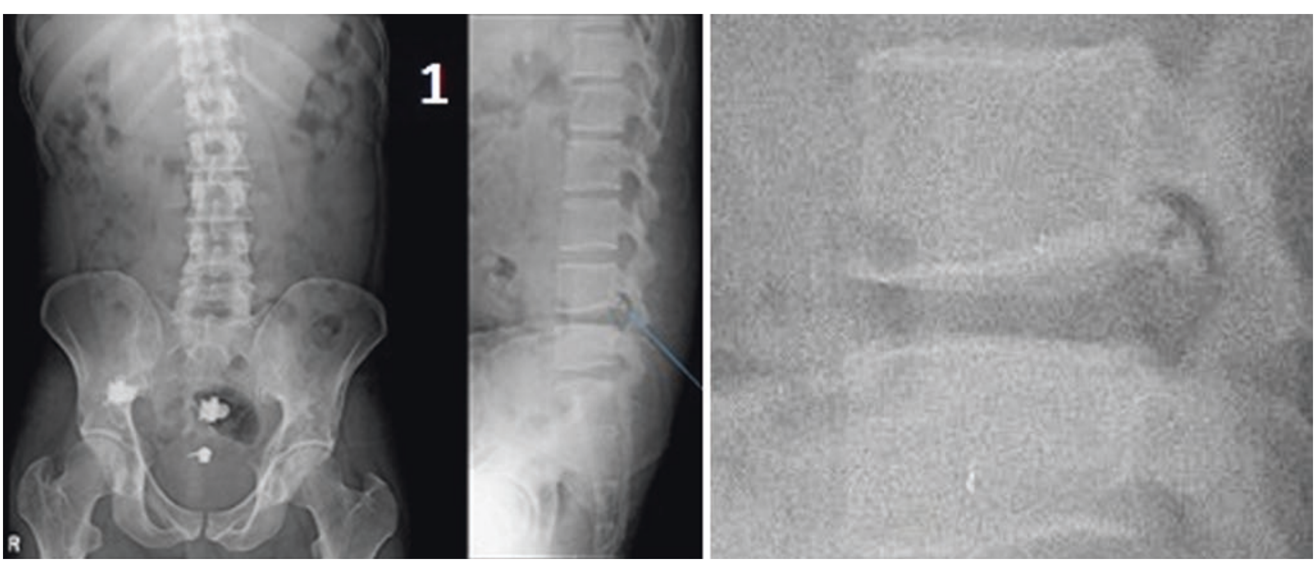


Fig. 32.11 Apophyseal lumbar ring fracture. (Used with permission from: Ravi Kumar et al. [39])

imaging used to rule out underlying fractures. Standard radiographs may be helpful, but advanced imaging with CT scan is almost always recommended if clinical concern for fracture exists [7]. MRI is usually reserved for evaluation of ligamentous stability in fractures. MRI in the acute setting for suspected soft tissue injuries is generally not recommended and has high risk of false-positive findings [6, 23]. Most injuries respond initially to symptomatic treatment with ice, heat, and NSAIDs [6]. Relative rest with early mobilization as tolerated can be helpful. The roles of other treatments such as physical therapy have not been well studied and can be used on an individual basis. Return to play is based on function and pain, and athletes who are not improving over the first 1–2 weeks or have significant pain longer than 4–6 weeks can be considered for referral to a spine specialist.

Neurological Injuries

Acute Sciatic Nerve Injuries

Sporting activity is a relatively rare cause of peripheral nerve injuries accounting for about 3% of injuries [42]. While most injuries occur to the upper extremity, cases of acute sciatic nerve injury occur with sports being the reported cause in about 6% of cases [42]. Mechanisms include direct impact causing crush injuries or traction forces. Injuries usually present with pain and varying degrees of weakness on examination. Treatment is supportive based on the severity of the injury. For injuries with persistent symptoms, electromyography can be considered after a few weeks to help classify the degree of nerve injury (Chap. 38). Athletes can gradually return to play as symptoms resolve and strength improves, but this can take months in some cases with rare instances of permanent injury.

Acute Spinal Cord Injuries

Sports are a rare cause of spinal injury accounting for approximately 8% of injuries. Injuries to the lumbar spine represent only a small portion, 11%, of these injuries [43]. Fractures most commonly occur in the upper lumbar spine, L1–L2 [7, 10, 14]. Athletes may present with varying degrees of pain and neurologic deficit. Pre-event emergency action plans should include knowledge of local and regional resources for spinal cord trauma. Initial stabilization of the spine and management of complications such as hypothermia and hypotension should be performed. Adjunctive treatments such as corticosteroids and cooling should not be used in most cases due to lack of evidence [44, 45]. The American Spinal Injury Association (ASIA) scoring system is most reliable at 72 hours but can be performed once the athlete is stabilized to classify clinical impairments and risk stratify outcomes [43]. Complete injuries, ASIA A, have worse outcomes than incomplete injuries, ASIA B–D. Surgical intervention is generally performed within 24–72 hours with some studies showing improved outcomes with 24 hours [46]. See Chap. 30 for more details on the management of acute spinal cord injuries.

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Check for updates

Stress Fractures

33

Bradley G. Changstrom

Key Points

- Stress fractures are common injuries in active athletes.
- Distance running and sports associated with leanness are the highest risk for stress fracture.
- Stress fractures associated with relative energy deficiency or the female athlete triad require additional evaluation.
- High-risk stress fractures have increased risk of complication including nonunion, delayed union, fracture with displacement, avascular necrosis, and need for surgical management.
- High-risk locations include the femoral neck, anterior mid-tibia, navicular, body of the talus, proximal second metatarsal, sesamoids, and pars interarticularis.
- Upper extremity stress fractures are uncommon; however, athletes who participate in overhead sports can develop these injuries.
- Imaging typically starts with plain radiographs; however, this method of imaging is not sensitive for stress fractures, especially early stress fractures.
- Preferred follow-up imaging includes MRI and radionuclide bone scans. CT imaging is occasionally used in confirmation of fracture lines or follow-up to assess bony healing.
- Treatment of stress fractures varies by the grade of stress fracture and location of injury, though recovery times can be prolonged compared to acute fractures.
- Acute presentations of stress fractures can occur if stress injuries are allowed to progress to complete fractures (Grade IV injuries); however, it is uncommon for these fractures to cause comminuted or displaced fractures other than tension-sided stress fractures.

B. G. Changstrom (✉)
Department of Internal Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Bradley.changstrom@ucdenver.edu

Introduction

Bone stress injuries are common overuse injuries in athletes and nonathletes alike. These injuries result from the failure of the skeleton to hold up against repetitive submaximal forces [1]. Stress fractures can occur in normal or metabolically weakened bones. Those that occur in the setting of weakened or osteoporotic bones are sometimes termed insufficiency fractures. Stress and insufficiency fractures should be differentiated from pathologic fractures that result from fractures of abnormal bone like those associated with bone tumors (malignant or nonmalignant) or Paget's disease [2]. This chapter will focus on bone stress injuries, particularly in acute symptomatic settings and sideline management in athletes.

Epidemiology

Rates of stress fractures vary by sport, age, and level of competitiveness. Due to inconsistent standards for measuring stress injuries with previous studies, it can be challenging to compare rates of injuries across populations. High school students have a rate of 2.22/100,000 athletic exposures in girls' sports and 1.27/100,000 athletic exposures in boys' sports [3]. At the collegiate level, 15% of division 1 female athletes reported a history of stress fracture on a pre-participation evaluation [4]. In a cohort study of a

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similar population of division 1 female athletes, approximately 10% suffered a bone stress injury over a 6-year study period [5].

Stress fractures occur most commonly in female athletes, distance running, and sports associated with leanness [6–11]. Women have a relative risk of 1.5–3.5 for stress fractures compared to men [3, 7]. One prospective study described an incidence of 3.9% in female adolescent athletes and demonstrated that several sports were risk factors for stress fractures, including running, basketball, and cheerleading/gymnastics [11]. Other risk factors for bone stress injuries include prior bone stress injury, BMI <19 kg/m^2, and late menarche (e.g., ≥15 years), all features of the female athlete triad [12]. Of note, several studies have demonstrated that prior fracture is the biggest risk factor for a bone stress injury [6, 13]. Clinicians should inquire about previous stress fracture history when evaluating athletes with a suspected bony stress injury.

In retrospective case studies, the most common locations for stress fractures are the tibia (24%), tarsal navicular (18%), metatarsal (16%), fibula (16%), femur (7%), pelvis (2%), and spine (1%) [14–16]; however, the location of stress injury varies by sport (Table 33.1). Stress fractures of the upper extremity are found nearly exclusively in overhead athletes and are much less common than lower extremity stress fractures [3, 17].

If left untreated, bony stress injuries can progress to complete fractures. This sometimes occurs in an acute manner as the weakened bone suddenly gives out. Athletes who continue to compete or train despite known bony stress injuries are more likely to have severe injuries as are athletes who have delays in diagnosis. These acute injuries are less likely to occur if bony stress injuries are diagnosed early. Higher-grade bony stress injuries (Grades 3 and 4) represented approximately 40% of bony stress injuries in a prospective study of college runners [18].

What Is Unique About Acute Presentation of Stress Fractures?

Stress fractures are unique overuse injuries with a broad spectrum of risk factors for injury. Risk factors for stress fractures can be divided into biological and biomechanical risk factors [1, 24]. Biological factors include female sex, medications, genetics, female athlete triad, and other dietary contributors [1]. Biomechanical factors include training patterns (e.g., mileage, inadequate recovery, type of exercise (e.g., running, basketball, gymnastics), training surface, and footwear), bone characteristics, and anatomic considerations (e.g., leg length discrepancy, foot type, small calf cross-sectional area) [1, 24].

What Do the Practitioners Need to Know While Managing Acute Presentation of Stress Fractures?

Stress fractures should be suspected when athletes complain of pain with increasing activity or repeated excessive activity and limited rest. Pain with ambulation, focal tenderness, and edema are the most common history and exam findings [16]. Athletes with acute symptoms at the site of a previous stress fracture have a high risk of recurrent injury in this area; athletes should be questioned for previous stress injuries.

The differential diagnosis of stress fractures certainly depends on location of injury, but includes muscle strains, tendinopathies, periostitis, exertional compartment syndrome, nerve or artery entrapment, and pathologic fractures [7, 16]. Exam findings include the "hop" test for tibial stress fractures (and other lower extremity stress fractures), the "fulcrum" test for femoral stress fractures, and a spinal extension or "stork" test for pars interarticularis stress fractures (spondylolysis) [7, 16]. Direct palpation with reproduction of pain, where feasible, is probably the best diagnostic test maneuver [7].

Table 33.1 Most common locations of stress fractures in athletes

	First	Second	Third
Cross country/track			
Women [3, 6, 19]	Tibia/lower leg	Metatarsal/foot	Tarsal/foot
Men [3, 6, 19]	Metatarsal/foot	Tibia/lower leg	Tarsal/foot
Basketball			
Women [3, 19, 20]	Tibia/lower leg	Metatarsal/foot	–
Men [3, 19, 20]	Tibia/lower leg	Metatarsal/foot	–
Tennis [21]	Tarsal navicular	Pars	Metatarsals
Football [3]	Foot	Lumbar spine/pelvis	Lower leg
Soccer [3, 22]	Metatarsals/foot	Lower leg	Lumbar spine/pelvis
Baseball [3]	Lumbar spine/pelvis	Olecranon	Metatarsals/lower leg
Volleyball [3]	Foot	Lumbar spine/pelvis	Lower leg
Gymnastics [3]	Lower leg	Lumbar spine/pelvis	–
Rowing [23]	Lumbar spine	Rib	–

Plain radiographs are the recommended initial step in diagnosis of a suspected stress fracture [16]; however, plain radiographs may miss early stress injuries as they only have a sensitivity between 10% and 70% depending on the timing of imaging and onset of symptoms [7, 25]. Radionuclide bone scans are more sensitive than MRI, but limited by specificity and need for radiation. MRI is generally the preferred follow-up imaging for suspected stress fractures [7]. On MRI, a Grade 1 injury shows periosteal edema on fat-suppressed images. Grade 2 injury shows abnormal increased signal within the marrow cavity or along the endosteal surface on fat-suppressed images. Grade 3 injuries show abnormal signal also present on T1 images and T2 images. Finally, Grade 4 injuries show a fracture line on both T1 and T2 images [7]. Fracture lines visible on plain radiographs are consistent with Grade 4 injuries (Fig. 33.1).

High-risk stress fractures are located in areas of poor vascular supply or are tension-sided stress injuries. High-risk stress fractures therefore have an increased risk of complication including nonunion, delayed union, fracture with displacement, avascular necrosis, and need for surgical management [7]. High-risk locations include the femoral neck, anterior mid-tibia, navicular, body of the talus, proximal second metatarsal, sesamoids, and pars interarticularis [7, 18, 24, 26]. Stress fractures located in trabecular bone (e.g., femoral neck, sacrum, and pelvis) are more common in athletes with low bone mineral density and should prompt additional bone health evaluation [18]. Although most bony stress injuries do not require urgent or emergent management, approximately 1% may progress to needing surgical intervention [3].

Fig. 33.1 Anterior tibia stress fracture. This 18-year-old female athlete developed bilateral anterior tibial stress fractures. This postoperative radiograph of the left leg demonstrated the “dreaded black line” consistent with cortical disruption of the anterior tibia (tension sided) due to a stress fracture (white arrows) and the surgically implemented intramedullary rods. (Photo Courtesy of Dr. Michelle Wolcott)

Stress fracture management should take into consideration the location of injury, the grade of injury, and the individual’s competitive situation [27]. Clinicians should consider the location of injury and risk in determining the urgency for imaging when considering a possible stress fracture as high-risk locations should be imaged more urgently. Ultimately, early detection of stress fractures is important as delay in diagnosis and treatment can result in prolonged return to activity and progression of injury [16, 17].

Managing suspected acute stress fractures on the sideline is relatively straightforward. Athletes should be removed from competition or training. Lower extremity injuries should be made non-weight bearing until the injury is better characterized with imaging. Urgent or emergent imaging should be considered if there is concern for a complete stress fracture, a displaced stress fracture, or especially if there is a concern for a stress fracture in a high-risk location. Overall, however, the risk of displaced fractures and comminuted fractures is small.

Return to Play

Return to play for stress fractures can vary between 4 and 12 weeks or longer once activity is restricted [14, 17]. The degree of severity found on MRI correlates to return to play in athletes [18]. On average, those with triad risks have higher-grade bone stress injuries on MRI and longer return to play [18].

Low-risk injuries respond well to nonoperative management and treatment is guided by the patient’s symptoms. High-risk injuries (by location and/or grade of injury) should be treated more aggressively with absolute rest, immobilization, or surgical fixation. The goal of management is to allow the stress injuries to heal with avoiding excessive deconditioning to the athlete [27].

Finally, for athletes with stress fractures, clinicians need to evaluate for relative energy deficiency in sport [28]. As stress fractures are the result of complex interactions between bone health, energy availability, hormonal states, and activity levels [28], failure to identify these athletes can lead to long-term health consequences outside of re-injury including osteoporosis, menstrual dysfunction, and abnormalities in

immunity, and cardiovascular and psychological health [28]. For example, peak bone mineral density, a major determinant of long-term risk of osteoporosis risk, is attained by early adulthood [11, 29]. Ongoing bone loss or failure to obtain adequate bone stores in young athletes is a major risk factor for osteoporosis. Identifying those at risk for stress injuries can help clinicians implement strategies to prevent stress fractures and sequelae of low energy availability states [12].

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Part VI

Acute Soft Tissue Injuries in Sports

Anna L. Watrerbrook

Tendons

34

Sagir Bera, Stephen R. Paul, and David Millward

Key Points

- Both full- and partial-thickness tears can be treated with nonsurgical and surgical techniques.
- Proximal biceps tears often involve chronic changes to the rotator cuff.
- Complete distal triceps tears are preferably treated by surgical repair.
- Smooth glide of the flexor tendons within the pulley system is important; therefore, referral should to be made to a hand surgeon promptly for primary repair of ruptured tendons.
- Extensor tendons are superficial and more amenable to direct repair in the emergency department.
- Ultrasound is useful in diagnosis of tendon injury and tears, offering dynamic visualization of the tendon and surrounding structures, although MRI remains the gold standard for precise diagnosis and prognostication.
- Early identification of central slip tear may prevent development of chronic boutonniere deformity.
- Patellar tendon tears occur more frequently at the patellar insertion.
- Complete ruptures of the patellar or quadriceps tendon can be treated both conservatively and surgically.
- The myotendinous junction of the hamstring experiences the highest eccentric loads and is the most susceptible site to injury.
- Patients with distal hamstring tendon injuries may benefit from early surgical intervention, while proximal injuries may be treated with conservative therapy.
- Achilles tendon ruptures are predominantly found in physically active middle-aged males.
- Surgical management of Achilles tendon ruptures can speed recovery back to work, though has higher risk of complications.
- Conservative and operative management of Achilles tendon ruptures have similar outcomes on return to preinjury levels of play.
- Patients with peroneal tendon subluxations and dislocations benefit from early surgical intervention, due to recurrent nature of symptoms.

Introduction

Tendons are load-bearing structures that transfer forces from muscle to bone [1]. Loading forces help modulate tendon structure, causing remodeling and degeneration. A strain is defined as a stretch or microtear of the underlying tendinous or muscular structure. Tendinopathy refers to a wide spectrum of disorders ranging from tendinitis (acute tendon inflammation) to tendinosis (chronic tendon degeneration) to tenosynovitis (tendon sheath inflammation) to partial and complete tendon ruptures. Ruptures are defined as complete tendon tears [4].

S. Bera (✉)
David Geffen School of Medicine at UCLA, Department of Family Medicine and Sports Medicine, Burbank, CA, USA

S. R. Paul
Department of Family and Community Medicine, Campus Health Service, University of Arizona, Tucson, AZ, USA
e-mail: spaul@email.arizona.edu

D. Millward
Primary Care Sports Medicine, Campus Health Services, The University of Arizona, Tucson, AZ, USA
e-mail: millward@email.arizona.edu

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Shoulder and Elbow

Rotator Cuff Tendons

The rotator cuff is made up of four muscles that run across the glenohumeral joint to help support and stabilize the dynamic movements of the shoulder. Due the multiplanar movements of the shoulder joint, the rotator cuff muscles and tendons can be exposed to chronic and repetitive stresses placing them at risk of injury. The biceps and triceps have tendons that cross the shoulder and elbow joint. As such, these tendons are often associated with shoulder injuries.

Mechanism of Injury in Sports

The shoulder joint is a highly dynamic joint that is partially stabilized by the four muscles (supraspinatus, infraspinatus, subscapularis, and teres minor) that comprise the rotator cuff (Fig. 34.1). Tears can be traumatic or atraumatic in nature. Traumatic tears are usually the result of a fall on an outstretched arm, direct blow, or forceful hyperextension and external and/or internal rotation of an abducted arm. More commonly, atraumatic tears result from erosion of the rotator cuff in overhead athletes, such as pitchers, swimmers, and volleyball players [5, 6].

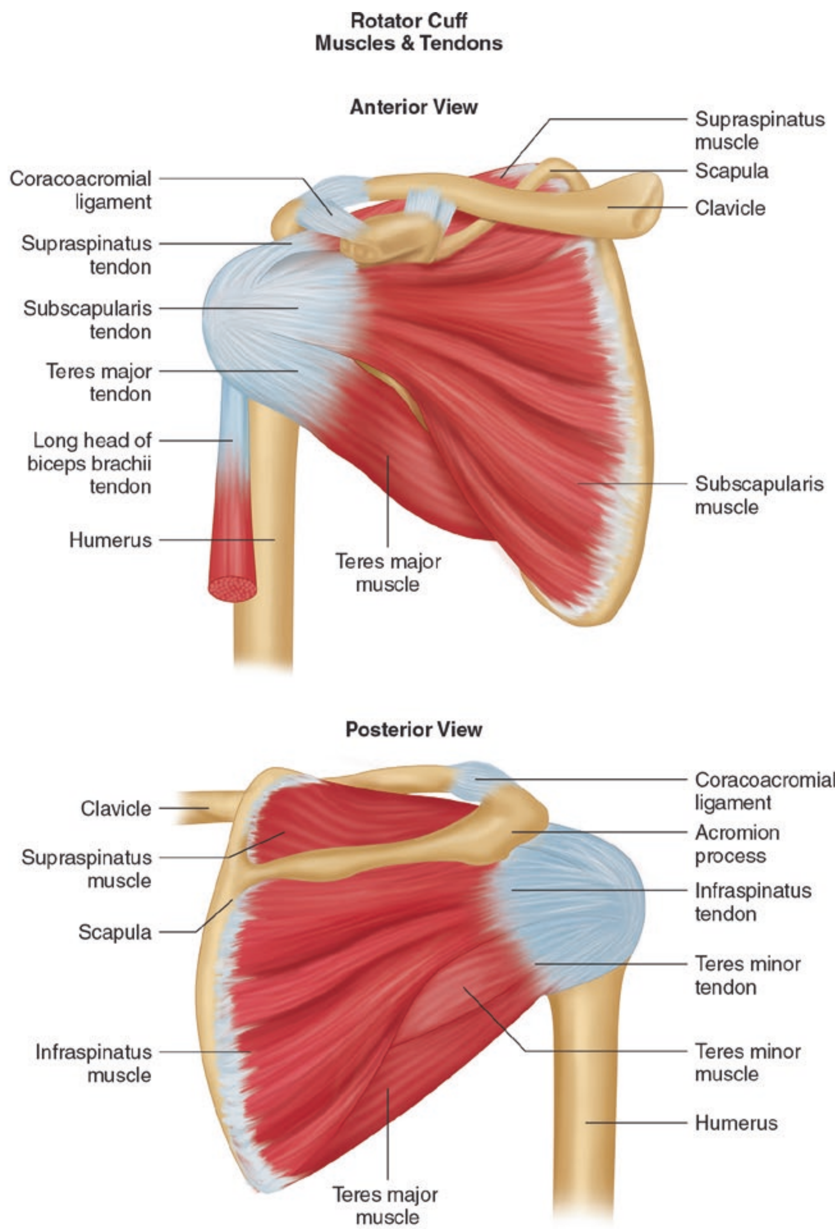

Fig. 34.1 Anatomy of rotator cuff

Epidemiology

Rotator cuff tears are multifactorial; some nonphysical activity-related risk factors include age, smoking, hypercholesterolemia, and family history [3]. However, rotator cuff disorders are the most common form of disability in the elderly with nearly 25% of individuals older than 60 and 50% of individuals older than 80 being disabled by rotator cuff disorders [3, 7]. Many can go on for years remaining asymptomatic. The shoulder joint is one of the few joints where the soft tissue can wear away quicker than the underlying bone. Atraumatic injuries may result from chronic degeneration of the rotator cuff.

Contact sports (e.g., football, rugby) or sports with repetitive overhead activities (e.g., baseball, volleyball) are more prone to shoulder injuries. Overhead athletes are at highest risk of atraumatic rotator cuff tears, due to the repetitive shearing forces along the soft tissue. Tears can affect the articular and/or bursal aspects of the rotator cuff tendon. The supraspinatus (84%) and subscapularis (78%) were most commonly involved in traumatic rotator cuff tears [6]. Partial-thickness tears are more commonly seen than full-thickness tears, with partial-thickness tears (<1 cm) progressing at a slower rate (25% at 2 years) than full-thickness tears [7]. Progression of tear size is often associated with worsening symptoms, in particular pain.

Clinical Presentation

As many rotator cuff tears are asymptomatic, they often go undetected for years before experiencing a traumatic or atraumatic event. Many times, patients may present with vague nocturnal pain or pain with sleeping on the affected side, which may radiate to the elbow. In an athletic setting, atraumatic rotator cuff tears are often associated with an identifiable event, such as a particular pitch or spike of the volleyball. Individuals with traumatic injuries will frequently present with fall on an abducted arm, a direct blow to the shoulder, or dislocation of the shoulder.

Diagnosis

As with most musculoskeletal injuries, rotator cuff tears can be diagnosed clinically, though imaging can help determine the extent of the injury. Patients may have limited range of motion or a painful arc. The empty can test (Jobe sign) is the most sensitive (68.4%) test and the drop-arm and lift-off tests are the most specific (100%) for detecting rotator cuff tears [8]. Numerous other tests, such as the external lag sign, can assist in diagnosing rotator cuff tears. Sensitivity and specificity of these tests improve with increasing thickness of the tear.

Plain radiographs are not specific for diagnosing rotator cuff tears but can help identify potential underlying etiology, such as calcifications from repetitive injury or subacromial osteophytes predisposing to injury. Ultrasound has good sensitivity (84%) and specificity (89%) for detecting partial- and full-thickness rotator cuff tears, though this is largely dependent on operator competence [8]. MRI has also shown a high sensitivity for detecting rotator cuff tears, with better sensitivity detecting full-thickness tears [9].

Initial Management

Acute treatment consists of rest, ice, compression, and pain management. An arm sling can be used for comfort and partial immobilization for a short time. It is important to rule out neurologic etiology of symptoms from the cervical spine.

Indications for Orthopedic Referral

There is much controversy among the medical community on which patients are the best candidates for surgical management. Multiple studies have shown an improvement in short-term outcomes of surgically treated partial-thickness tears; however, there were no significant differences in long-term outcomes with those undergoing physical therapy alone [6, 10, 11]. Those with massive tears and pseudoparalysis (reduced active range of motion) can benefit from early surgical intervention [12]. Professional athletes often choose early surgical intervention. For instance, one study found that only 8% of NFL athletes with full-thickness rotator cuff tears were treated nonoperatively [5]. Failure to improve with conservative management is also an indication for surgical evaluation. Depending on the type and extent of injury, surgical techniques include open repairs (often used in large or complex tears), arthroscopic repair, or mini-open repair (mixing components of arthroscopic and open techniques).

Follow-Up Care

Whether surgical or nonsurgical treatment is selected, the patient needs to go through a proper rehabilitation program. Physical therapy has shown good outcomes for both partial- and full-thickness tears [10]. Corticosteroid injections have also been found to improve pain and relieve symptoms following rotator cuff tears [8, 13]. Rehabilitation should focus on strength, range of motion, and return back to functional activity.

Return to Sports

Surgically treated patients can require between 4 and 6 months for full recovery. As with return to play with most musculoskeletal injuries, patients should have full, symmetric, pain-free range of motion. Strength should ideally be back to baseline or at least within 85% of the unaffected side. Return to play can be quite variable, ranging from a few weeks to a few months, dependent on the extent of injury and requirements for return to play.

Complications

Improperly treated and rehabilitated rotator cuff tears can lead to further progression of tears, in turn, leading to worsening of symptoms such as pain and loss of range of motion (e.g., adhesive capsulitis). For professional athletes, an improperly treated rotator cuff injury can lead to improper recovery and incomplete return back to prior level of sporting activity.

Biceps Tendon

Mechanism of Injury in Sports

Distal biceps ruptures are rare but most commonly occur at the tendon insertion along the radius, because of sudden excessive eccentric extension load to a flexed elbow. Proximal biceps ruptures are much more common than distal ruptures, with partial ruptures being uncommon. Proximal ruptures usually occur at the long head of the biceps tendon (LHBT) either in older populations with a history of underlying rotator cuff pathology or more acutely in younger populations in the context of lifting activities or bracing a fall with an eccentric load.

Epidemiology

There is a paucity of epidemiological information about biceps ruptures, especially sports related. The overall incidence of biceps ruptures in an epidemiological study in the UK reported total incidence of 0.53/100,00 patients/year. The incidence for distal biceps rupture has been reported to be 1.2 per 100,000 patients/year in a small cohort [14]. A more recent, larger epidemiological study reported the incidence to be higher: 2.55–5.35 per 100,000 patient-years [15]. 90–97% of biceps ruptures involve the long head of the biceps. Most acute ruptures occur in the older (>50) population doing routine activities and lifting. Injuries in the younger population may be associated with falls or heavy resistance loads.

Fig. 34.2 Popeye deformity found with proximal biceps rupture. Right side is acute, left is 10-year-old injury. (Courtesy of Dr. Morteza Khodaee)

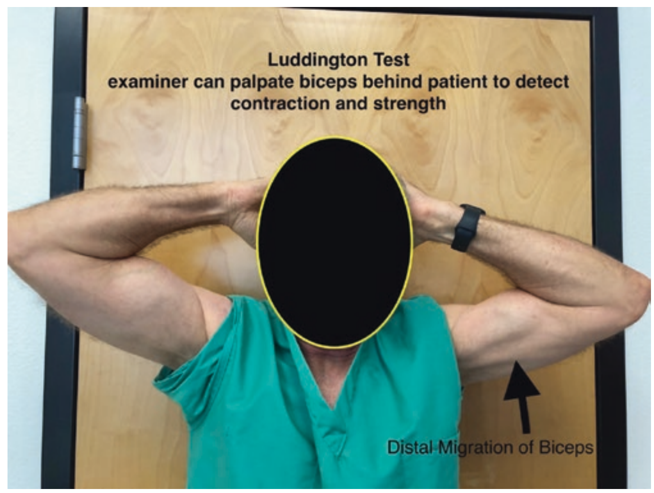

Fig. 34.3 Ludington test to help evaluate proximal biceps tendon injuries. (Credit Stephen Paul)

Clinical Presentation

Patients with distal biceps tendon ruptures may present with a sensation of a "pop" and pain with resisted eccentric load to contracted biceps. Pain will be felt along the cubital fossa, while having weakness and pain with supination and flexion of the elbow. Individuals with proximal biceps tendon ruptures present with acute pain to the anterior shoulder or upper humerus. Often a "Popeye" deformity from a retracted biceps muscle is visible (Fig. 34.2).

Diagnosis

Injuries may demonstrate weakness against resistance in supination and flexion of the elbow. A palpable defect may be present with complete rupture, including the "Popeye" sign with proximal tears and a "reverse Popeye" sign with a distal biceps tear. Additionally, a palpable gap may be noted. Partial tears are subtler and may have crepitus along the radial tuberosity with strength testing. The hook test (patient flexes elbow to 90° with supination—examiner is able to hook the distal tendon from lateral aspect of an intact tendon) can assist in diagnosing distal tendon tears, while Ludington test (distal migration of proximal muscle belly) can assist in diagnosing proximal tears (Fig. 34.3).

Imaging can be helpful in diagnosing biceps ruptures. Arthroscopy is the gold standard for diagnosis. But, ultrasound can be an inexpensive imaging modality to dynamically visualize the biceps tendon and functionally very

effective. The FABS view (flexion, abduction, supination) of the MRI helps identify biceps injuries.

Initial Management
As with most musculoskeletal injuries, initial management includes rest, ice, compression, and elevation. Patients may be placed in posterior elbow splint for immobilization, with sling for comfort.

Indications for Orthopedic Referral
Distal biceps rupture involving more than 50% of the tendon benefits from surgical intervention. Prolonged pain following proximal biceps rupture is an indication for tenotomy of the long head of the biceps. In younger patients, or where symmetry is required, tenodesis can be helpful.

Follow-Up Care
Partial tears involving less than 50% can undergo conservative management as initial approach. Physical therapy can be helpful in recovery. Passive range of motion should be started early in the rehabilitation process. Slower progression may be beneficial in older population.

Return to Sports
Strengthening usually begins in 4–5 weeks or after resolution of pain. Complete recovery usually takes on average 3 months.

Complications
Patients may experience reduced strength in elbow flexion and supination, as well as loss of range of motion. Additionally, patients may experience a cramping sensation with prolonged supination. Nerve damage can be seen in as high as 30% of patients after surgical repair. Permanent dysmorphia may be present following conservative management. Shoulder pain may persist if associated rotator cuff pathology is not addressed.

Triceps Tendon

Mechanism of Injury in Sports
Triceps tendon tears are rare. Strong, acute eccentric loads against a contracted triceps muscle can put a patient at risk of a triceps tendon rupture. This is often seen with a fall on an outstretched arm or with a blocking motion (e.g., offensive lineman). Direct blows to the region of the triceps tendon can also cause a rupture.

Epidemiology
Triceps tendon ruptures are rare in sports, accounting for less than 1% of all tendon injuries in the upper extremity [16]. Males are more frequently affected than females. There is an equal predominance bilaterally, with hand dominance not playing a role. Steroid and quinolone antibiotic use are extrinsic risk factors for triceps tendon ruptures, as is chronic tendinosis [17].

Clinical Presentation
Patients usually present with preceding injury, such as fall on outstretched arm, direct blow, or forcible flexion of the elbow joint. Pain, swelling, and ecchymosis may be present at the insertion of triceps, along with weakness in elbow extension.

Diagnosis
Diagnosis of triceps tendon rupture is mostly clinical. Physical examination may demonstrate posterior elbow pain, swelling, ecchymosis, and palpable defect. Weakness of elbow extension may have extensor lag of up to 30°. A modified Thompson test (performed with arm hanging over back or chair or table with elbow flexed, with compression of triceps muscle body causing elbow extension) can also aid in diagnosis (Fig. 34.4).

Imaging, such as MRI and ultrasound, can be helpful in determining extent of injury, whether partial or complete. Ultrasound also allows for dynamic visualization of the tendon. Radiographs may demonstrate associated bony avulsion along the olecranon.

Initial Management
Initial management consists of rest, ice, compression, and elevation (RICE) therapy with appropriate analgesic management. Patient should be immobilized with sling or posterior splint with 20–30° of flexion.

Indications for Orthopedic Referral
Complete tears of the triceps tendon should be referred for surgical repair. Some also recommend partial tears be treated operatively due to the high success rate and low morbidity of such procedures. Surgical repair is best performed within 1–2 weeks of initial injury.

Follow-Up Care
If conservative management is chosen, patients may be immobilized in posterior splint for up to 6 weeks. Passive elbow range of motion can be started within 2–3 weeks of immobilization, with active starting after immobilization is complete. A dedicated physical therapy rehabilitation program should be undertaken with emphasis on range of motion, flexibility, and eccentric strength.

Return to Sports
Some studies have shown return to play in as little as 5 days (one player in the NFL), while wearing a brace for partial triceps tendon ruptures, but they are at risk of converting to complete ruptures [18]. More commonly, the average return

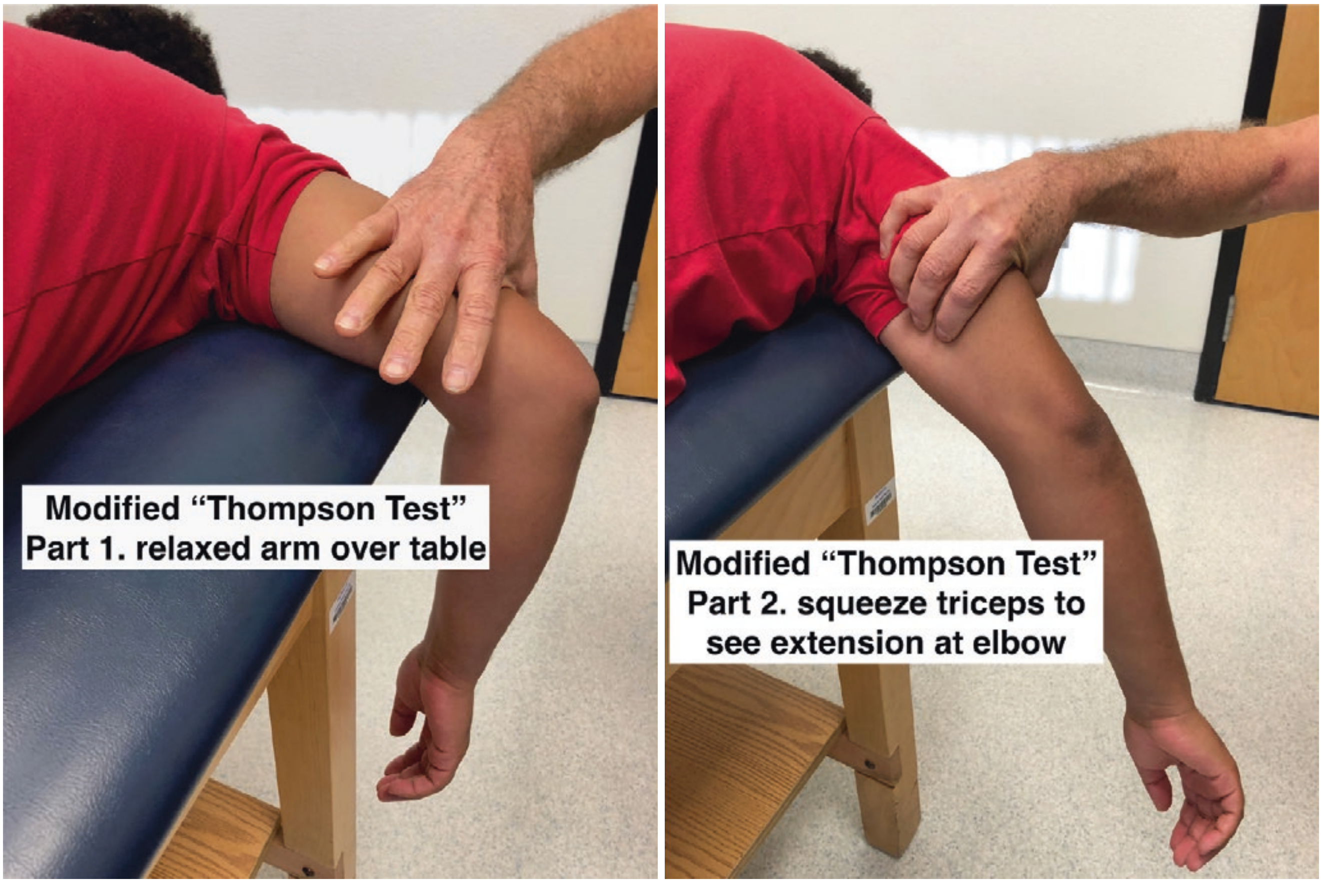

Fig. 34.4 A Modified Thompson test can be used to assess triceps tendon integrity. (Credit Stephen Paul)

to play following partial rupture is 5 weeks. The criterion is full range of motion and strength. Recovery after surgical repair can take anywhere from 3–4 months to 1 year, which usually means loss of season for professional athletes [18].

Complications

Generally, most individuals regain their strength and full functional ability. However, recovery can be a slow process. Individuals treated conservatively are at risk of repeat tendon rupture.

Hand and Wrist

Wrist and hand injuries are seen across different sports. Two of the more common injuries, hand extensor tendons and mallet finger, have prevalences of 18/100,000 per year and 9.9/100,000 per year, respectively [2]. The pulley-lever system formed by slips over the flexor tendons of the finger, along with the relative lack of protection to the extensor tendons, places the fingers at risk of a variety of injuries.

Extensor Digitorum Tendon (Mallet Finger)

Mechanism of Injury in Sports

Mallet finger, or extensor tendon avulsion from the distal phalanx, results from forced flexion of an extended distal interphalangeal (DIP) joint, such as being hit on the tip of a finger by a ball or hand to hand combat activities. This injury can result in rupture of the tendon or avulsion of the tendon insertion.

Epidemiology

The incidence of mallet finger is 10/100,000 [19], with the majority occurring during sports participation. These injuries are less frequently encountered in work or household settings. The third finger is the most frequently injured due to the anatomical prominence of the finger.

Clinical Presentation

Patients usually complain of axial loading or hyperflexion of the finger that is held in extension. They may complain of pain and swelling along the DIP joint and inability to extend the DIP joint.

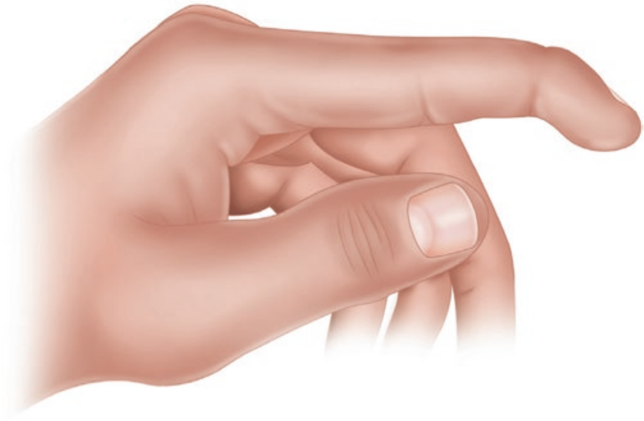

Fig. 34.5 Picture of extensor lag of DIP joint following extensor tendon rupture

Fig. 34.6 Stack-type splint holding DIP in extension. (Credit Stephen Paul)

Diagnosis

The patient may have swelling, tenderness to palpation, and pain over the DIP joint with inability to actively extend finger. To appropriately assess injury, hold the finger in flexion at proximal interphalangeal (PIP) joint and have patient try to actively extend at DIP joint. Extensor lag may be present (Fig. 34.5).

Plain radiographs are important for evaluation, specifically looking for avulsion fracture or joint subluxation. Repeat radiographs following splinting can help to evaluate bony healing.

Initial Management

The DIP joint should be continuously immobilized with a splint in mild hyperextension for a minimum of 6–8 weeks [20]. RICE therapy should be utilized, along with analgesics as required (Fig. 34.6).

Indications for Orthopedic Referral

In general, large avulsion fractures, involving greater than 40% of the joint, should be referred for operative management. However, there is no significant evidence on the exact indication for surgery. Surgery can also be considered for those that have failed 10 weeks of continuous immobilization.

Follow-Up Care

Splint should remain in place continuously for 6–8 weeks, followed by 2 additional weeks of nighttime splinting. Avulsion fractures are splinted for 6 weeks, while tendon ruptures are splinted for 8 weeks in full extension. Regular follow-up is recommended to follow compliance. There is still controversy on the optimal splinting and immobilization technique. If deformity (>20°) persists, splinting must continue. Slowly begin ROM exercises and proper strengthening. Consider splinting in extension during athletic activities for 2 additional months.

Return to Play

If able to protect the DIP joint in extension with a splint, the patient may participate in activity after 6 weeks. Otherwise, patient will have to wait until complete resolution of immobilization period, including nighttime immobilization. Regardless, the athlete should wear a splint during the activity for the next 1–2 months for protection.

Complications

Extensor lag of up to 10° is common, though this should not result in functional deficits. Most complications result from continuous compressive splinting, including skin maceration or neurovascular compromise. Volar subluxations of the distal phalanx can lead to risk of swan neck deformity or degenerative changes of the DIP joint.

Flexor Digitorum Profundus Tendon (Jersey Finger)

Mechanism of Injury in Sports
This injury is primarily seen in sporting activities that involve tackling or direct/indirect grabbing of a jersey, such as in football, hockey, soccer, and rugby. The flexed finger is subsequently forced into extension at the distal interphalangeal (DIP) joint, resulting in avulsion of the flexor digitorum profundus.

Epidemiology
The fourth (ring) finger is the most commonly injured, accounting for nearly 75% of cases. The injury is frequently misdiagnosed as a finger sprain. The injury may have an isolated avulsion tear or avulsion fracture along the base of distal phalanx. Tendon and bony fragment retraction may occur proximally, becoming entrapped at the A3 or A4 pulley (Fig. 34.7).

Clinical Presentation
The patient may recall the specific injury mechanism, such as grabbing or getting the finger caught on an opponent's jersey. Patient may have local pain and swelling along volar distal finger, with inability to fully flex with isolated flexion at DIP joint without pain.

Diagnosis
There may be swelling and ecchymosis along site of injury at the distal phalanx. If retraction is present, a palpable lump or deformity may be felt along the A3 or A4 pulley or further proximally. Loss of flexion at the DIP joint is the most reliable finding. Examination should focus on isolation of flexion at the DIP joint which can be accomplished by holding the finger in full extension at proximal interphalangeal and metacarpophalangeal (MCP) joints. A thorough neurovascular exam should be performed to rule out further compromise. Plain radiographs can help in assessing avulsion injury or fracture, as well as possible retraction, though MRI or ultrasound may be better modalities to assess extent of tendon damage and location of tendon following retraction.

Initial Management
Initial management should focus on immobilization with finger splint (applied dorsally with maintenance of slight flexion) with early referral for orthopedic surgical evaluation (Fig. 34.8). NSAIDs and ice therapy can also be helpful for swelling and pain management.

Indications for Orthopedic Referral
Most flexor digitorum profundus avulsion injuries require operative management and fixation, which are best performed within 1–2 weeks of injury. Injuries that are associated with neurovascular compromise should be evaluated emergently. Failed conservative management is also an indication for surgical repair.

Follow-Up Care
Soon after the period of immobilization, patients can begin passive flexion exercises of PIP and DIP joints. Strengthening activities under the guidance of a hand therapist can expedite recover [21]. Strength and functional ability should return prior to free return to play.

Return to Play
Return to play is 8–12 weeks after surgical repair. For those individuals that forego surgery, they may return to sports

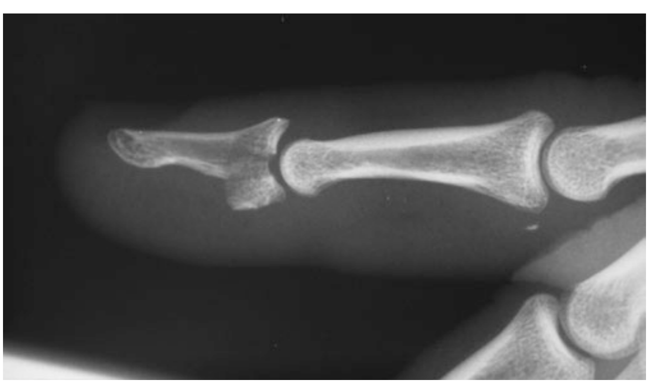

Fig. 34.7 Plain radiograph showing avulsion fracture of distal phalanx often seen with jersey finger injuries

Fig. 34.8 Dorsal splint holding DIP in flexion following jersey finger injury. (Credit Stephen Paul)

immediately, though there is the risk of having permanent complications or incomplete and improper healing of injury. Many athletes choose not to get surgical repair immediately, to allow completion of sports season.

Complications

Improperly treated injuries can have permanent functional limitations and pain.

Central Slip Tear (Boutonniere Deformity)

Mechanism of Injury in Sports

This injury results from forced flexion of an extended proximal interphalangeal (PIP) joint or a volar dislocation of the middle phalanx. Either will involve avulsion of the triangular ligament of the central slip, allowing lateral bands to migrate volarly to the joint. This is most frequently seen in ball-handling athletes or volleyball players.

Epidemiology

The exact incidence is not well known, but this injury is thought to be very rare among all tendon injuries.

Clinical Presentation

Patients typically present with exact injury mechanism, as specified above, or complaint of "jammed finger." They may complain of pain and swelling at the PIP joint, as well as difficulty moving finger.

Diagnosis

Early physical examination findings consist of tenderness to palpation and swollen PIP joint, with weak active extension but full passive extension. Stereotypical deformity usually presents later, consisting of flexion deformity at PIP and hyperextension at DIP (Fig. 34.9). A pseudoboutonniere deformity (fixed flexion contracture with inability to extend at PIP joint) can be mistaken for a true deformity. Elson's test (PIP bent over table edge at 90° resisted extension of middle phalanx, checking for DIP flexibility; positive test if DIP is rigid with weakness of PIP extension) can help diagnose this injury [22]. Plain radiographs can be helpful in evaluating fractures, dislocations, and volar plate injuries.

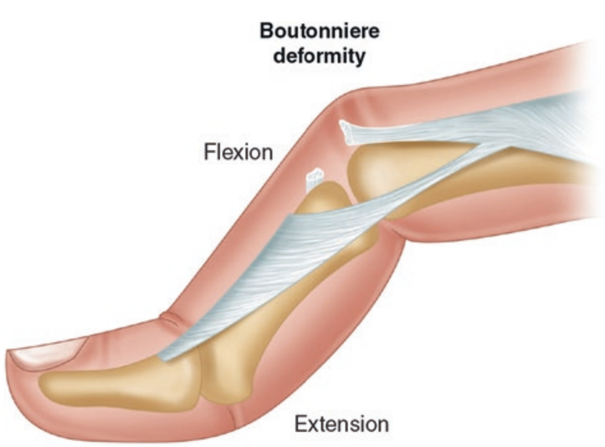

Fig. 34.9 Boutonniere deformity demonstrating hypertension at DIP and flexion at PIP joints

Initial Management

Initial management should include splinting at PIP joint in full extension for 4–6 weeks [19], while allowing full active flexion and extension at the PIP joint. Pain management and RICE therapy may be recommended for additional relief.

Indications for Orthopedic Referral

Immediate surgical referral should be made for irreducible volar PIP dislocations or any injuries with neurovascular compromise. Large or displaced intra-articular fractures should also be referred. Individuals that have flexion contractures, have failed conservative management, or have symptomatic chronic deformities may benefit from surgical intervention.

Follow-Up Care

Finger should be immobilized in strict extension at the PIP joint for 4–6 weeks, with another 2 weeks of nighttime splinting [19]. The DIP joint should be allowed to move freely. Begin gentle passive and active range of motion exercise after splinting. Physical therapy with a hand specialist can be helpful.

Return to Play

There are no consistent recommendations on return to play, and recommendations are dependent on sporting activity and requirements, as well as ability to properly splint the joint. Individualized return is advised as necessitated by athlete, sport, and position played.

Complications

True boutonniere deformity can develop several weeks after improperly treated injury. Improperly treated injuries may have chronic flexion contractures, pain, or decrease in functional ability.

Flexor Tendons of Hand (Pulley System Tears)

Mechanism of Injury in Sports

This injury results from a sudden slip while in a gripping position causing a strain of the flexor tendon and a subsequent tear in the pulley system from forced extension on a flexed finger. Most often the second (A2) and fourth (A4) pulleys of the ring and middle fingers are involved. This

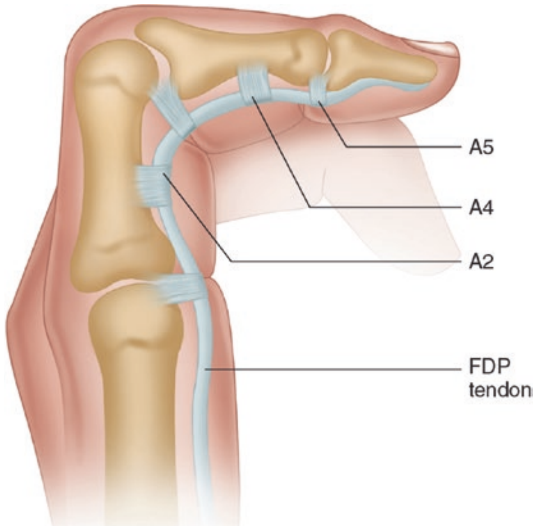

Fig. 34.10 Pulleys of the finger

injury is most frequently seen in climbers and baseball players, with the former involving the A2 and/or A4 pulleys and the latter involving the A4 pulley (Fig. 34.10).

Epidemiology

The exact incidence is unknown, as these injuries are rare. As mentioned above, the second (A2) and fourth (A4) pulleys of the middle and ring fingers are most frequently affected.

Clinical Presentation

Patient may complain of a painful pop after slipping from a gripping motion. They may also have pain along the flexor tendon and may have contracture noted at the proximal interphalangeal joint.

Diagnosis

Diagnosis is primarily made clinically. Patient may complain of pain at pulley, with or without bowstringing. If two pulleys are involved, the patient may demonstrate significant bowstringing along with difficulty making a fist (Fig. 34.11). Diagnostic ultrasound may reveal the torn pulley and slippage of volar tendon. MRI can also assist in identifying extent of injury.

Initial Management

As long as there is no significant bowstringing, initial management consists of pain control, icing, and immobilization for 10–14 days [23].

Indications for Orthopedic Referral

Surgical management should be considered if more than one pulley or bowstringing occurs. This can also be considered for those that fail conservative management.

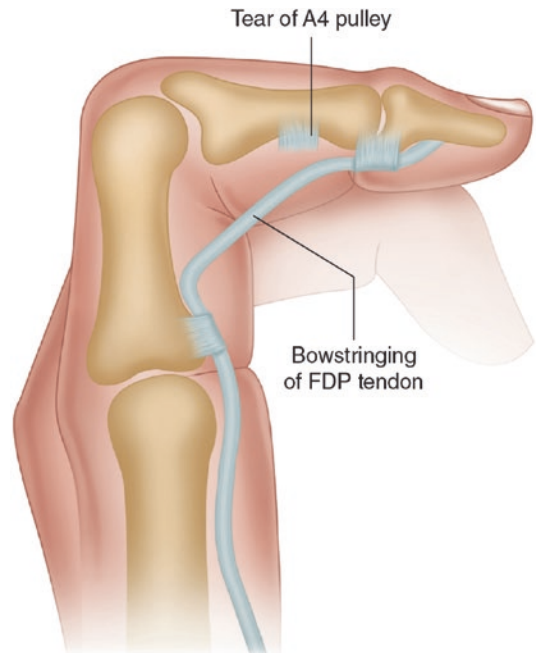

Fig. 34.11 Bowstringing of the flexor digitorum profundus following A4 pulley tear

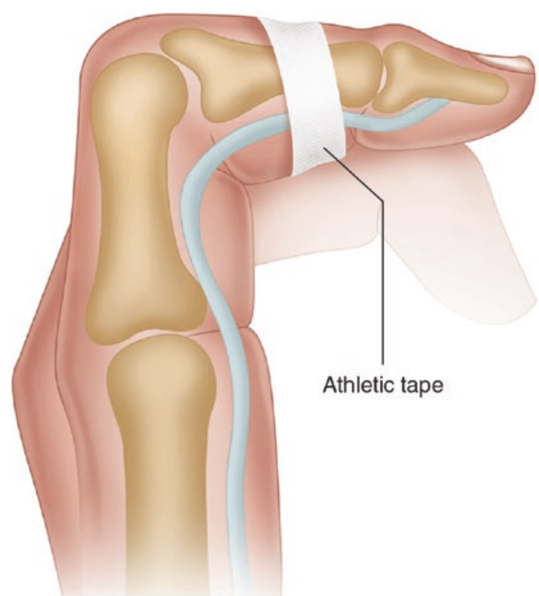

Fig. 34.12 Athletic tape to support flexor digitorum profundus following A4 pulley tear

Follow-Up Care

Following a period of immobilization, area should be protected by taping over the torn pulley to contain the tendon in close proximity to the finger as the normal, physiological pulley would (Fig. 34.12). A dedicated physical therapy program preferably with a hand specialist should be started.

Return to Play

Patient may return to activity once pulley system has painless flexor activation with protection. Otherwise, the patient may require protection of the pulley system with medical taping for 4–6 weeks or use of pulley ring for up to 6 months.

Complications

Most individuals recover completely. Rare complications include continued bowstringing, pain, and stiffness.

Extensor Carpi Ulnaris Tendon

Mechanism of Injury in Sports

The extensor carpi ulnaris tendon may sublux or dislocate with combined wrist flexion, ulnar deviation, and supination. This is most frequently seen in racquet- or stick-bearing sports, such as hockey, tennis, or lacrosse.

Epidemiology

The exact incidence of injury is unknown, as this is a rare injury.

Clinical Presentation

Patient may experience a snapping sensation, while wielding a stick or racquet with sudden deceleration. Patient may complain of pain along the dorsal-ulnar aspect of the wrist, usually in wrist extension, ulnar deviation, and supination.

Diagnosis

The patient may present with tenderness to palpation and swelling along the extensor carpi ulnaris tendon. Wrist flexion, ulnar deviation, and supination may cause palpable subluxation. The synergy test can help differentiate extensor carpi ulnaris involvement from intra-articular ulnar-sided pathology [24]. This test can be performed by having the patient rest their elbow on the table in supination, with a neutral wrist; the examiner than grasps the long finger and thumb, while patient actively abducts the thumb, causing the extensor carpi ulnaris (along with flexor carpi ulnaris) to synergistically activate with prominent pain, indicating tendon pathology. It is important to rule out intra-articular pathology including triangular fibrocartilage complex (TFCC) tears, distal radioulnar joint pathology, or lunotriquetral ligament tear. Radiographs can help rule out bony pathology. Ultrasound can help visualize dynamic function, while MRI helps visualize tendinosis or other pathology.

Initial Management

Initial management consists of pain control and RICE. Underlying tenosynovitis should be appropriately treated; then to address the subluxation, immobilize with the wrist in radial deviation and the forearm in pronation. This may be needed for up to 6 weeks [25].

Indications for Orthopedic Referral

Surgical management may be considered for dislocations or for those that fail conservative management.

Follow-Up Care

If immobilizing, wrist should be placed in radial ulnar deviation and pronation of the forearm for several weeks for a subluxation or for up to 4 months for dislocation. After a period of immobilization, isometric strength training can begin, progressing to eccentric strengthening exercises [25].

Return to Play

Typical return to play is 4–6 weeks after immobilization and proper rehabilitation for conservatively treated subluxation or dislocation of the extensor carpi ulnaris tendon, compared to 3 months for surgically repaired injury.

Complications

Improperly treated injuries may have continued instability, recurrent subluxation, and/or ongoing tenosynovitis.

Thigh and Knee

Lower extremity muscle injuries commonly occur within sporting events and training activities. Thirty percent of all injuries recorded during a 5-year study of European soccer players were muscle-related injuries, 32% involving the quadriceps, and 28% involving the hamstrings [26]. Injuries to the tendons commonly occur at the origin, the myotendinous junction, or the distal insertion. The myotendinous junction is referred to as the focal point at either the proximal or distal aspect of a muscle before tapering to the insertion [26]. The most common type of quadriceps injury is an intramuscular strain of the musculotendinous junction [26]. In addition to these three sites, it is important to remember that the hamstring and quadriceps tendons have large intramuscular or central tendons, a site where injury commonly occurs [27, 28].

Quadriceps and Patellar Tendons

Mechanism of injury in sports: The quadriceps muscle group crosses two major joints, the hip and the knee (Fig. 34.13). Most commonly, the quadriceps muscle group is injured by an eccentric load [29]. Muscles often contract eccentrically to absorb kinetic energy and protect joints; however, eccentric muscle activation can produce more

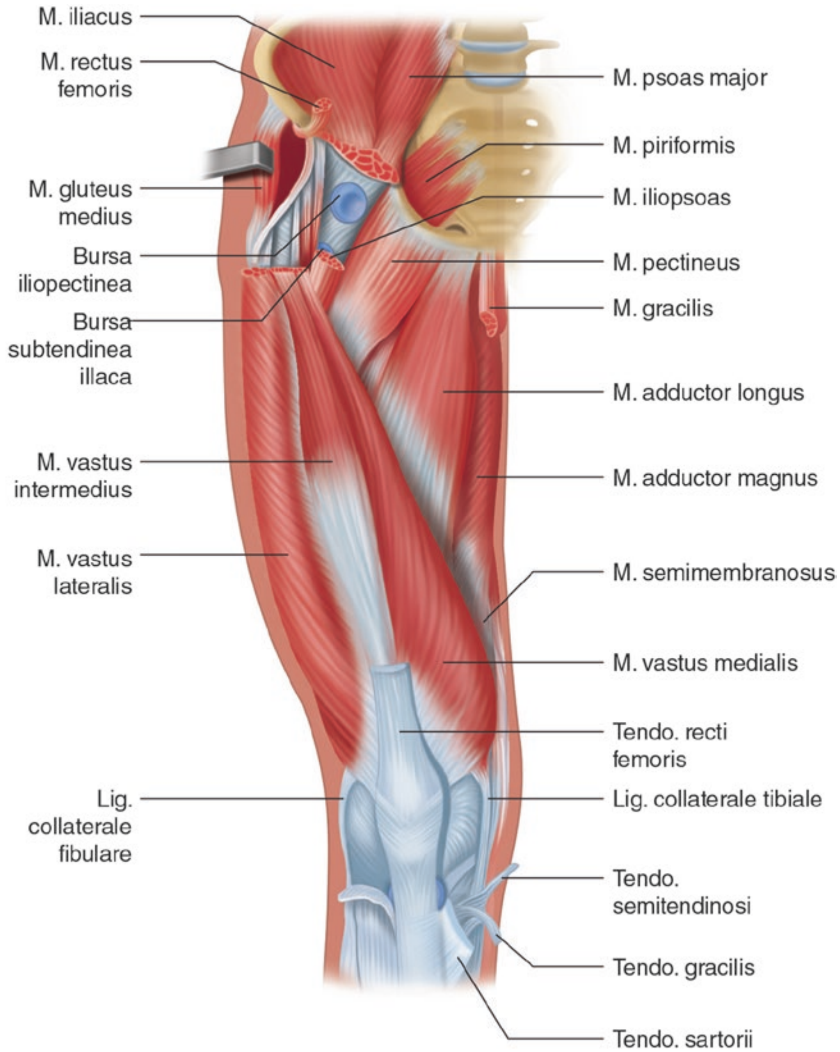

Fig. 34.13 Anatomy of quadriceps musculature

force or tension within the muscle than when it is activated concentrically, making it more susceptible to rupture or tearing [30]. The direct head of the rectus femoris originates at the anterior inferior iliac spine (AIIS) and the reflected head in the upper region of the acetabulum. Its tendon stretches from the distal third of the quadriceps muscle forming an extensive myotendinous structure down to the tendinous insertion at the upper pole of the patella [29]. The quadriceps tendons merge together prior to inserting onto the patella in a layered fashion. The superficial layer represents the rectus femoris, the deep layer represents the vastus intermedius, and the middle layer consists of the vastus lateralis and the medialis [31]. This tendon continues inferiorly to the anterior aspect of patella inserting onto the tibial tuberosity. The portion from the inferior patella to the tibial tuberosity is referred to as the patellar tendon as it histologically resembles a tendon rather than a ligament.

Kicking athletes are particularly vulnerable to rectus tendon avulsion during a forceful contraction of the quadriceps with the hip extended and the knee in a flexed position moving to the hip flexed and the knee in an extended position. If this motion is suddenly stopped or blocked by another player or object, a rectus avulsion may occur. The primary function of the quadriceps muscle during running is a deceleration of knee flexion when the heel strikes the ground [32]. The injury can occur with sudden deceleration during running, as well as during hip hyperextension with the knee in a flexed position [33].

Proximal avulsion injuries of the rectus femoris tendon are not common. The injury has been described both at the direct head of the rectus femoris where it attaches to the anterior inferior iliac spine (AIIS) [34] and at the myotendinous junction [35]. Injury at the patellar insertion can occur as a result of direct trauma but also eccentric contraction as previously mentioned [36]. Patellar tendon ruptures occur more often than quadriceps tendon ruptures [37].

Epidemiology

Multiple studies have examined various intrinsic and extrinsic risk factors for these injuries. Studies have

shown that increased age, career duration, and previous injury increase the risk of injury [38]. Mechanical instability of the ankles or knees, general joint laxity, and functional instability have shown a predisposition to injury [38]. Extrinsic risk factors that appear to increase the injury risk include lack of training, low training to match ratio, and playing on a hard surface with high friction [38].

Clinical Presentation

Initially, the patient may report a sudden pain during a sporting activity. If a complete tear has occurred, the patient may hear or feel a sudden pop, followed by severe pain, swelling, and difficulty ambulating. With complete tendon tears, the provider should consider other contributing causes such as anabolic steroid abuse, renal disease, metabolic bone disease (hyperparathyroidism), and medications associated with tendon rupture (fluoroquinolones) [39].

Diagnosis

The initial physical examination begins with observing the athlete's gait. These individuals may be unable to ambulate with complete tears or have an antalgic gait with partial tears. A neurovascular examination should be performed to assess for neurologic injury. Ecchymosis and edema may be observed on inspection (Fig. 34.14) Palpating along the complete course of the tendon and muscle group can reveal a gap if the tendon or muscle fibers have retracted (Fig. 11.4). Comparing to the opposite side for symmetry is beneficial. When a quadriceps tendon rupture occurs, there may be a palpable defect just proximal to the superior pole of the patella [39]. When assessing strength, the ability to extend the knee is limited with partial tears and absent with complete tears. The patient with a complete tear cannot maintain a straight leg and cannot raise the leg against gravity while supine [39]. MRI and ultrasound can be beneficial in detecting the extent of tendon damage to help better guide appropriate treatment (Fig. 34.15).

Fig. 34.14 (**a**) Gap in the left quadriceps tendon just proximal to the superior border of the patellae in a 64-year-old male with quadriceps tendon rupture. (Courtesy of Dr. Morteza Khodaee). (**b**) A 24-year-old male with complete left patellar tendon rupture due to a basketball injury. (Courtesy of Dr. Morteza Khodaee)

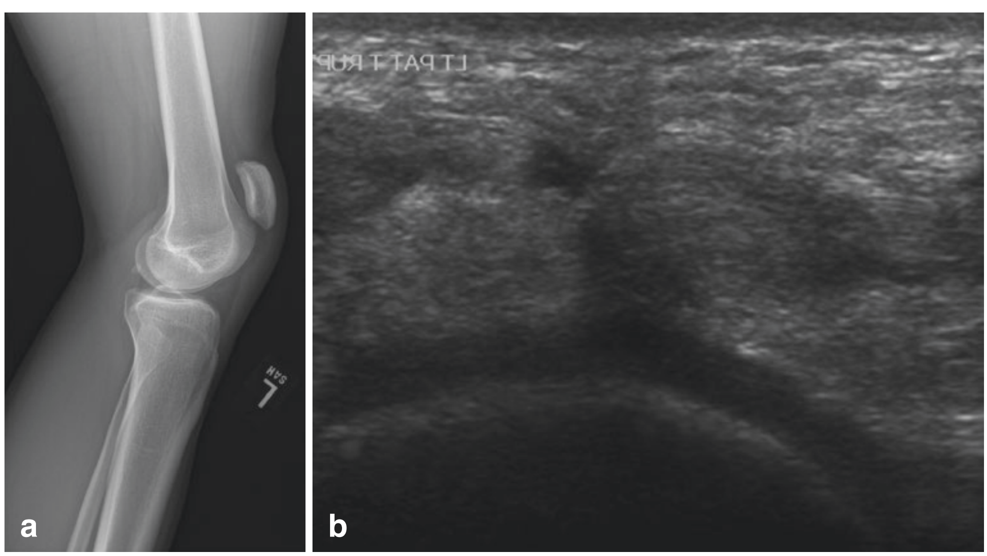

Fig. 34.15 (**a**) Lateral plain radiography of the same patient (Fig. 34.14b) reveals superior patellar migration and knee joint effusion. (Courtesy of Dr. Morteza Khodaee). (**b**) Long axis ultrasound reveals a gap in the patella tendon. (Courtesy of Dr. Morteza Khodaee)

Initial Management

The initial care of an athlete with partial or complete tear of the quadriceps tendon consists of RICE therapy and pain management. With a significant or complete tear, non-weight-bearing status with crutches is recommended. The knee can be protected with a straight leg immobilizer for a short period.

Indications for Orthopedic Referral

Orthopedic referral should occur immediately if a complete quadriceps or patellar tendon tear is suspected. Early surgical reconstruction of quadriceps or patellar tendon ruptures is necessary to restore the extensor mechanism and optimize a functional recovery. With regard to rectus femoris avulsions, there is no consensus on the appropriate treatment in high-level athletes [29]. The proximal avulsion of the rectus femoris in adult patients is not commonly reported, and the treatment of this injury remains controversial. In a study involving professional American football players, it was shown that players could be managed without surgery and successfully return to play in 6–12 weeks [33]. In another small case series, surgical treatment was chosen with some athletes returning to professional play 15.8 ± 2.6 weeks after surgery [29].

Follow-Up Care

As pain subsides following the acute injury, rehabilitation of the quadriceps tendon injury begins. If a complete tear is suspected, orthopedic referral should occur immediately. Orthopedic referral should be considered if there is persistent or worsening pain, progressive weakness, or a failure to improve with rehabilitation.

Return to play

Return to play follows the same general principles as with other injuries. The athlete should have regained symmetric, pain-free range of motion (ROM). Ideally, full strength should be regained to reduce the likelihood of reinjury. Rehabilitation should include a functional progression of sports-specific activities in a controlled setting prior to return to competitive sport.

Complications

Partial and complete ruptures of the quadriceps tendon can result in weakness of the muscle, which can be minimized with appropriate rehabilitation. This is especially true for professional athletes.

Hamstring Tendon

Mechanism of Injury

Injuries to the hamstring muscle group have been classified into two mechanisms, high and low energy. The high-energy mechanism involves a quick change in muscle length combined with a change in magnitude and rate of force [40]. It is most often associated with sports involving sprinting. During the last 25% of the swing phase of running, the hamstrings assist in proximal hip extension, while decelerating knee extension distally. Research evaluating sprint mechanics suggests that strain injury risk is greatest near the end of the swing phase, when the hamstrings reach maximal length and undergo eccentric contraction just before heel strike [41, 42]. The low-energy mechanism is a stretching-type injury occurring at the extremes of muscle length [40]. This mechanism of injury occurs with the hip flexed and the knee extended, while exceeding the muscle's end range of motion [40].

Complete rupture of the proximal complex has been defined as the tearing of all three tendons with or without retraction [43]. Complete ruptures are rare and tend to occur with preexisting tendinopathy [44]. The myotendinous junction experiences the highest eccentric loads and is the most common location of injury [44].

An understanding of the hamstring anatomy will assist with the diagnosis of this injury. The hamstring complex consists of three muscles (Fig. 34.16) [45]:

1. The biceps femoris long head arises from the medial aspect of the posterior ischial tuberosity and inserts on the fibular head. The short head arises from the linea aspera below the gluteal tuberosity and inserts on the fibular head.
2. The semimembranosus originates from the ischial tuberosity anterior to the conjoined tendon and inserts primarily into the posterior medial tibia plateau but also the medial collateral ligament and popliteus.
3. Semitendinosus arises from the long head of the biceps femoris via the conjoined tendon and inserts on the proximal medial tibia posterior to the sartorius.

Epidemiology

Hamstring injuries, both muscular and tendinous, commonly occur in athletes, accounting for up to 29% of all injuries across various sports [44]. The hamstring muscle group spans both the hip and knee joints, producing potential for rapid and extreme muscle lengthening. In a review of 179 cases of injury to the hamstring muscle group, there were 21 injuries involving the proximal origin of the ischial tuberosity with 16 avulsions, 154 injuries of the muscle belly, and 4 injuries involving the distal insertion site [26].

Clinical Presentation

Most patients will present with acute pain in the posterior thigh region of the leg. Athletes may report an audible pop and the onset of hamstring tightness [46, 47]. The presentation can be acute, but often times, there is a history of recurrent injury.

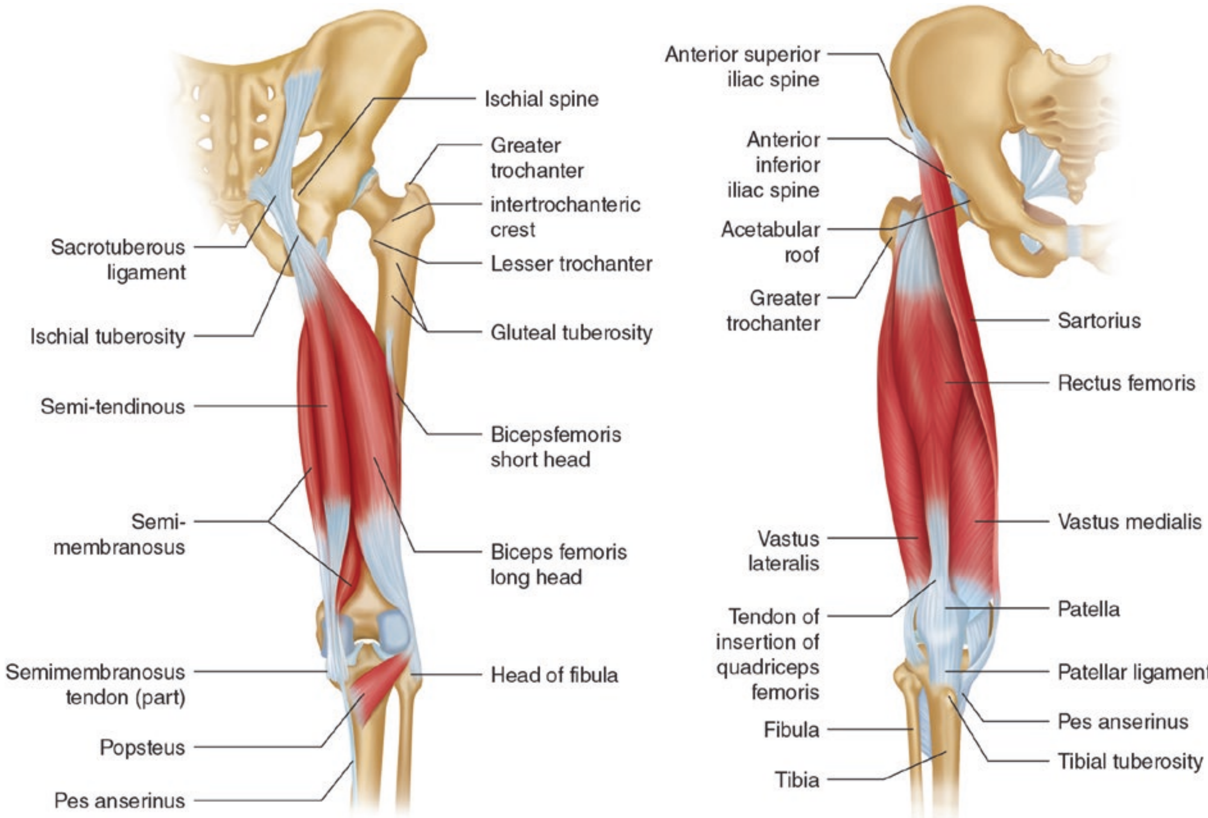

Fig. 34.16 Anatomy of hamstring

Diagnosis

Observing the athlete's gait can provide the first clue as they may walk "stiff-legged" in an attempt to avoid hip flexion and knee extension [48, 49]. A thorough neurovascular examination should be performed to assess for sensory or muscle weakness. Careful inspection and palpation of the hamstring muscles should be performed to localize the injury and assess for a defect in the muscle or tendon. There may be obvious swelling and ecchymosis in the posterior thigh (Fig. 34.17). Tenderness at either the origin or insertion should alert the provider to the possibility of an avulsion. A thickened area of subcutaneous tissue can occur with proximal and distal avulsion injuries [49–51]. As with other injuries, comparison to the opposite side for symmetry is helpful.

Range of motion is assessed with the patient lying supine. The popliteal angle is determined by flexing the hip and knee to 90°, followed by passive extension of the knee. The knee flexion angle at which posterior thigh pain and guarding occur is compared with that in the contralateral, uninjured leg [44]. An increased angle on the affected side suggests a hamstring injury [46].

Assessing hamstring strength can be done with the patient in the prone position. While resisting active knee flexion, the severity and location of the injury can be assessed. Active knee flexion while the examiner extends the knee to 30° reproduces the common eccentric load mechanism and also aids in diagnosis [44]. While in the prone position, hip extension strength can be assessed and compared to the opposite side.

Imaging can help confirm the diagnosis and predict prognosis. MSK ultrasound and MRI are the two modalities most commonly used. Plain radiographs can also be helpful as an avulsion off the site of origin may sometimes be seen. Ultrasound is relatively inexpensive and convenient as it can be done at the point of care. The contrast resolution, however, is not as good as MR, and evaluation of deeper structures can be limited when faced with bulky musculature [26].

Initial Management

The initial care of an athlete with partial or complete tear of a hamstring tendon consists of RICE therapy and pain management. With a significant or complete tear, non-weight-bearing status with crutches is recommended.

Indications for Orthopedic Referral

Distal injuries can occur at any of the hamstring insertion sites. Early referral to orthopedics to assess the need for surgery is recommended. While evidence to guide appropriate therapy in

Fig. 34.17 Right semitendinosus partial rupture in a 42-year-old male day #3 post-injury. (Courtesy of Dr. Morteza Khodaee)

this area is lacking, the limited data that is available suggests early surgery may be beneficial [45]. In a case series of 25 distal semitendinosus tendon ruptures in high-level athletes, early treatment involved nonoperative care including rest, modalities, and rehabilitation exercises, followed by functional progression [51]. Forty-two percent of the patients failed initial nonoperative treatment and subsequently had surgery with resection of the torn tendon and adjacent scar tissue, with an average recovery time of 12.8 weeks. Another group of patients was treated with acute surgery that resected the torn tendon and adjacent scar tissue within 4 weeks of injury. The acute-phase group had an average recovery of 6.8 weeks after surgery. The authors concluded that distal semitendinosus ruptures frequently do not recover when treated nonoperatively. Therefore, acute surgery may be considered in elite athletes for whom time to recovery is very important for more favorable time frames to return to participation [44].

Follow-Up Care

Injuries to the hamstring origin are best managed when recognized early. In general, conservative treatment is considered for proximal hamstring injuries that involve a single-tendon and/or multiple-tendon injuries with less than 2 cm of retraction [44]. Surgical indications for proximal hamstring injuries include those involving 2 tendons with >2 cm of retraction and 3-tendon tears. Recognition and treatment of proximal hamstring injuries within 4 weeks have been shown to possibly improve outcomes as later recognition provides for a more difficult repair, leading to increased surgical complications [50].

Conservative management consists of activity modification, NSAIDs, and physical therapy. Other modalities include ultrasound, shockwave therapy, electrical stimulation, and cryotherapy. Overall, with regard to rehabilitation, there is limited, high-level evidence to support or refute a specific protocol. Various rehabilitation protocols have been used to facilitate return to play with mixed results. In a study by Askling et al. [52], loading the hamstring during extensive lengthening reduced recovery time as compared to conventional hamstring exercises with less focus on lengthening. In a separate study, four daily sessions of static hamstring stretching shortened recovery as compared to one session per day [53]. There is preliminary evidence from another small study of mixed ability athletes to suggest that exercise to correct movement dysfunction could reduce time to return to full activity and the risk of reinjury [54]. Further studies are needed to help guide rehabilitation from this injury.

Return to Play

Return to play follows the same general principles as with other injuries. The athlete should have regained symmetric, pain-free range of motion (ROM). Ideally, full strength should be achieved or fall within 85% of the unaffected side. Rehabilitation should include a functional progression of sports-specific activities in a controlled setting prior to return to competitive sport.

Recovery time and return to play with hamstring tendon injuries can be prolonged. In terms of the clinical assessment, the following factors have been associated with a recovery time exceeding 40 days: VAS pain score greater than 6 out of 10, popping sound at the time of injury, pain during everyday activities for more than 3 days, ecchymosis, and a loss of at least 15° of motion [55].

The following factors noted with MRI studies correlated with a longer recovery time. Findings on MRI associated with a higher grade of injury include muscle involvement >75%, complete transection, retraction, central tendon disruption of the biceps femoris, proximal tendon involvement, shorter distance to the ischial tuberosity, length of the hamstring injury, depth, volume, and large cross-sectional area. With respect to ultrasound studies, the following factors

were associated with a poor prognosis: large cross-sectional area, injury outside the musculotendinous junction, hematoma, structural injury, and injury involving the biceps femoris.

Complications

Knee flexion and hip extension weakness can occur as a result of this injury. Appropriate physical therapy may help to minimize weakness. In complete tears or those that require surgery, the sciatic nerve can be involved and include neuropraxia or a stretch of the nerve during surgery that eventually resolves [44]. This complication can lead to burning pain radiating down the leg and weakness of the affected extremity.

Foot and Ankle

The Achilles tendon is formed from the congruence of the gastrocnemius and soleus musculature, inserting with a common tendon along the calcaneus. The Achilles tendon is the strongest tendon in the body [56]. Chronic degeneration is thought to play an integral role in leading to Achilles tendon rupture. Rupture of the Achilles tendon is usually related to involvement to physical activity. Similarly, injuries to the peroneal tendons (brevis and longus) are thought to be related to physical activity. The tendons arise from the fibular shaft and run posterior to the lateral malleolus before inserting along the base of the fifth metatarsal (brevis) and cuboid (longus). The peroneal tendons are one of the few tendons following a nonlinear pathway in the body, thereby having a dynamic range of actions. The tendons are held in place by a superior peroneal retinaculum.

Achilles Tendon

Mechanism of Injury in Sports

Despite being the strongest tendon in the body, the Achilles tendon is the most frequently injured [3, 56]. The true etiology of tendon ruptures is unknown but is thought to fall under one of two theories, the degenerative and mechanical theories [57]. Under the mechanical theory, the Achilles tendon ruptures due to different movements and forces exerted on the tendon. Conversely, the degenerative theory postulates that minimal forces applied to chronically degenerated soft tissue lead to failure of the tendon. Athletes that require repetitive jumping or running (e.g., basketball, football, running) are most vulnerable to this injury. Approximately 9% of recreational runners are affected by Achilles tendon ruptures [56]. Furthermore, nearly 5% of professional athletes have their career ended by rupture of the Achilles.

Epidemiology

Nearly three quarters of Achilles tendon ruptures are experienced by men aged 30–49 participating in sporting/physical activity [3]. The lifetime prevalence of acute tendon rupture and chronic tendinopathy in athletes is 8.3% and 23.9%, respectively, compared to 5.9% and 2.1% in the general population [58]. Long-distance runners are particularly at risk of tendon ruptures. Injuries are a result of forced dorsiflexion or pushing off motion. The Achilles receives its blood supply at the musculotendinous and osteotendinous junction. Accessory blood supply to the middle portion of the Achilles tendon is via the thin layer of tissue encasing the Achilles tendon known as the paratenon. This zone, approximately 2–6 cm proximal the insertion of the Achilles tendon into the calcaneus, is the least vascularized and most prone to rupture and poor healing [56, 59].

Athletics participation and male predominance places individuals at risk of Achilles tendon ruptures [56, 57]. Additional intrinsic risk factors include leg length discrepancy, pes cavus, limited mobility at subtalar joint, inflammatory arthropathies, obesity, diabetes, and hypertension [56]. Corticosteroid and quinolone antibiotic use are additional risk factors associated with Achilles tendon rupture [59]. Overtraining and chronic tendinopathy can also place individuals at risk of rupture.

Clinical Presentation

Patients typically present sudden onset of posterior heel pain with sensation of a "kick" to the back of the leg, with or without an audible "pop" [58]. Patients are typically unable to bear weight due to pain in the area and have weakness or inability to plantarflex. As Achilles tendon ruptures are most often associated with athletic or physical activities, patients may recall a specific incident, such as jump stop or getting foot caught in pothole.

Diagnosis

Diagnosis of Achilles tendon ruptures is largely clinical. There may be a palpable defect, with pain and ecchymosis, along the Achilles tendon (Fig. 34.18). The American Academy of Orthopedic Surgeons (AAOS) guidelines note that diagnosis of an Achilles tendon rupture can be made if two or more exam findings are positive: decreased plantar flexion strength, palpable defect proximal to the insertion site of the Achilles tendon, positive Matles test (increased passive ankle dorsiflexion at rest), and positive Thompson test (lack of passive plantar flex with compression of calf in prone position) [57]. The Matles and Thompson test were found to have high sensitivities, 88% and 96%, respectively [60].

There is conflicting evidence on the utility of MRI in the evaluation of Achilles tendon ruptures, given the high sensitivity and positive predictive value of physical examination tests. The sensitivity for Achilles tendon rupture is 100% for patients found to have a positive Thompson test, palpable

defect, and decreased resting ankle tension versus 90.9% on MR imaging [61]. Ultrasound allows for inexpensive, dynamic visualization of the Achilles tendon to help better determine if surgical repair is required [57] (Fig. 34.19).

Initial Management

Initial focus of acute treatment should consist of RICE therapy, while providing appropriate analgesia therapy. Crutches can assist with maintaining non-weight-bearing status. The ankle can be temporarily immobilized in a cast or walking boot in slight plantar flexion.

Indications for Orthopedic Referral

At this time, there is little consensus on the timing and need for operative management of Achilles tendon tears. Failed nonoperative management for 4–6 months resulting in persisting impairment or those with high functional demands are indicators for surgical intervention [58]. Surgery (Fig. 34.20) has been shown to decrease re-rupture rates and speed up recovery back to work but also has higher risk of surgical complications, including wound necrosis, superficial infection, or sural nerve injury [57, 62].

Follow-Up Care

A functional dorsal block splint, which maintains the patient in slight plantar flexion, is gradually decreased back to neutral. Previous studies recommended prolonged immobilization, but more recent literature recommends early range of motion (after 10–14 days) to reduce risk of re-rupture [57,

Fig. 34.18 Left Achilles tendon rupture. (Courtesy of Dr. Morteza Khodaee)

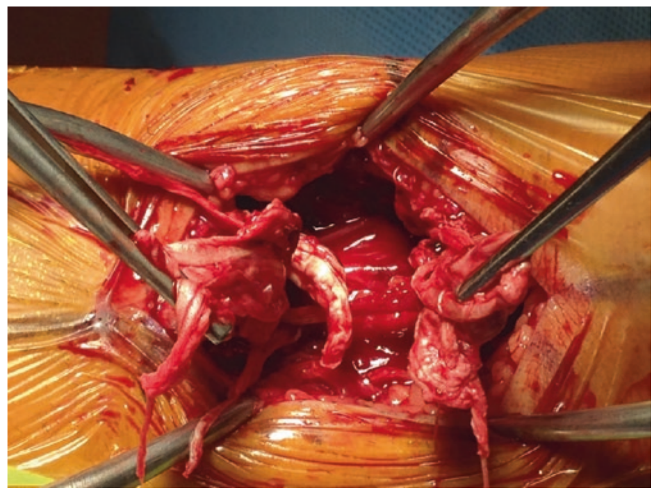

Fig. 34.20 Intraoperative image of an Achilles tendon rupture 1 day after injury. (Courtesy of Dr. Morteza Khodaee)

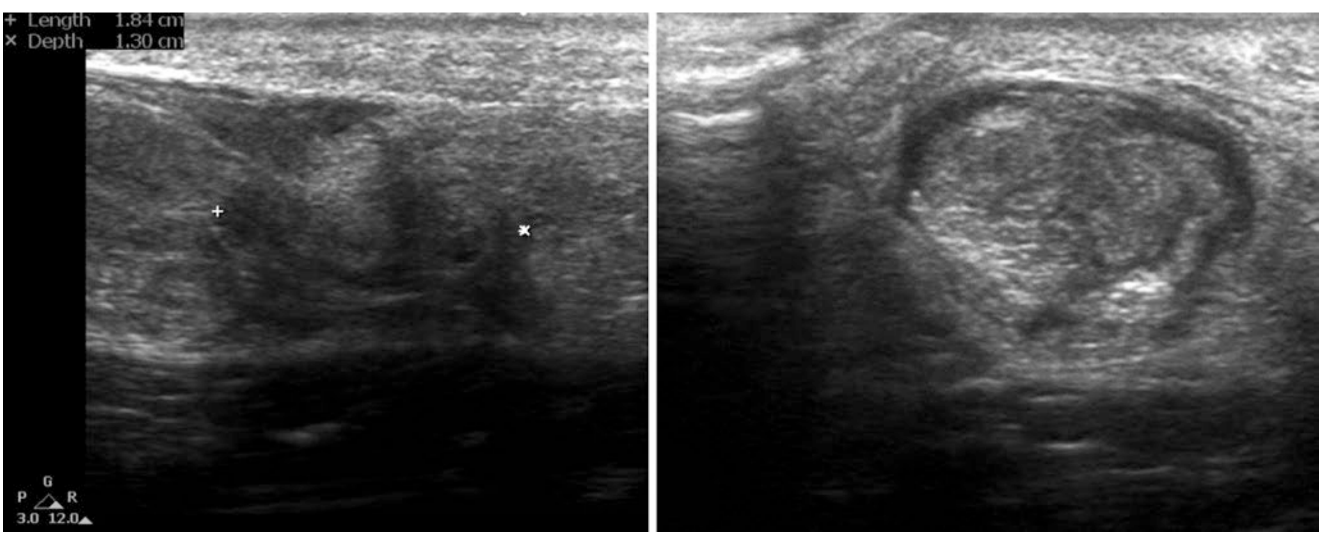

Fig. 34.19 Long and short axis ultrasound images of the right Achilles tendon rupture in a 45-year-old male injured playing soccer. (Courtesy of Dr. Morteza Khodaee)

58]. These individuals should be monitored closely as they are at high risk of deep vein thromboses. Each patient should undergo a dedicated strengthening program, focused on regaining range of motion, functional activity, and eccentric strengthening.

Return to Sports

There is little consensus on the most appropriate time for return to play, with the mean time of 6 months postoperatively [57]. Return to sports following conservative management also follows a similar timeline, as strength, range of motion, and functional activity return.

Complications

While average patients and amateur athletes may return to preinjury levels of performance, elite athletes have difficulty returning to preinjury levels [63]. Despite appropriate physical therapy, some individuals may continue to have functional deficits and decreased physical activity.

Peroneal Tendons

Mechanism of Injury in Sports

The action of the peroneal tendons (brevis and longus) is plantar flexion and eversion, while providing dynamic stability to the lateral ankle. They arise from the fibular shaft forming the lateral compartment of the leg. After running posterior to the lateral malleolus, the peroneus brevis inserts at the base of the fifth metatarsal, and the peroneus longus inserts medially at the base of the first metatarsal and first cuneiform. The peroneal tendons pass through a single-tendon sheath which is held in place by the superior peroneal retinaculum.

Peroneal tendon injuries are most frequently seen in the athletic population [64]. Specifically, these injuries are seen in sports that require extensive cutting or jumping, including soccer, basketball, ice skating, skiing, hockey, and gymnastics. Most frequently the injury results from an eccentric contraction of the peroneal tendons in ankle dorsiflexion, with or without inversion. This can be seen with quick stop or cutting motions.

Epidemiology

Due to the dynamic, nonlinear pathway of the peroneal tendons, injuries to the tendons can include chronic tendonitis, rupture, subluxation, or dislocation (Fig. 34.20). Isolated ruptures of peroneal tendons are extremely rare but are often associated with inversion ankle injuries or fractures [65]. Peroneal tenosynovitis and chronic tendinopathy can predispose to injury. The peroneus brevis is most frequently ruptured at the level of the malleolus, while the peroneus longus rupture is most frequently seen along the cuboidal fossa or tunnel. Dislocations occur in 0.3–0.5% of all traumatic ankle events but are often misdiagnosed and thought to be underreported [64]. Dislocations do not regain their anatomical position, while subluxations spontaneously reduce back into anatomical position.

Clinical Presentation

Acute injuries are often very difficult to distinguish from lateral ankle sprains, due to pain and swelling. Intense pain may be palpable along the pathway of the peroneal tendons, posterior and inferior to the lateral malleolus. The patient may recall an injury mechanism involving dorsiflexion, with or without inversion. Additionally, the patient may complain of a popping or snapping sensation over the lateral malleolus [66]. Often, these patients are unable to bear weight or continue playing, while having a sensation of ankle instability [59].

Diagnosis

As mentioned, peroneal tendon injuries are often misdiagnosed as lateral ankle injuries due to the similarities in presentation, as well as the higher prevalence of such injuries. The patient may have pain and swelling along the pathway of the peroneal tendons. A positive peroneal tunnel compression test, pain with active dorsiflexion and eversion of the foot against resistance along the posterior ridge of the fibula, is indicative of entrapment of the superficial peroneal nerve [59]. A subluxation of the peroneal tendons may be palpable if there is a tear of the superior peroneal retinaculum. Injuries to the superior peroneal retinaculum are graded according to Oden's classification: periosteal elevation along the fibular (type 1), tear of the fibular attachment (type 2), avulsion fracture at the fibular attachment (type 3), and tear of its posterior attachment (type 4) [65]. Type 1 and type 3 injuries are the most common.

Imaging modalities can be very helpful in diagnosing peroneal tendon injuries and differentiating from more common lateral ankle injuries. Plain radiographs may demonstrate avulsed cortical bone along the lateral malleolus. MR imaging is helpful in visualizing the peroneal tendons, especially in the axial plane views. However, ultrasound has been shown to have more accuracy than MRI (94.4% compared to 65.7%) in evaluating tendons around the ankle [65] (Fig. 34.21). Ultrasound can be utilized for dynamic visualization of the peroneal tendon, including frank subluxation with foot in resisted eversion and dorsiflexion [65].

Initial Management

Acute treatment consists of RICE therapy and analgesics as required for pain management. Crutches can be used for non-weight-bearing, as initial injury can be painful. If symptoms are minor, an ankle brace can be used for comfort. A non-weight-bearing cast can be utilized to completely immobilize the ankle in mid plantar flexion to allow the injury to heal [59].

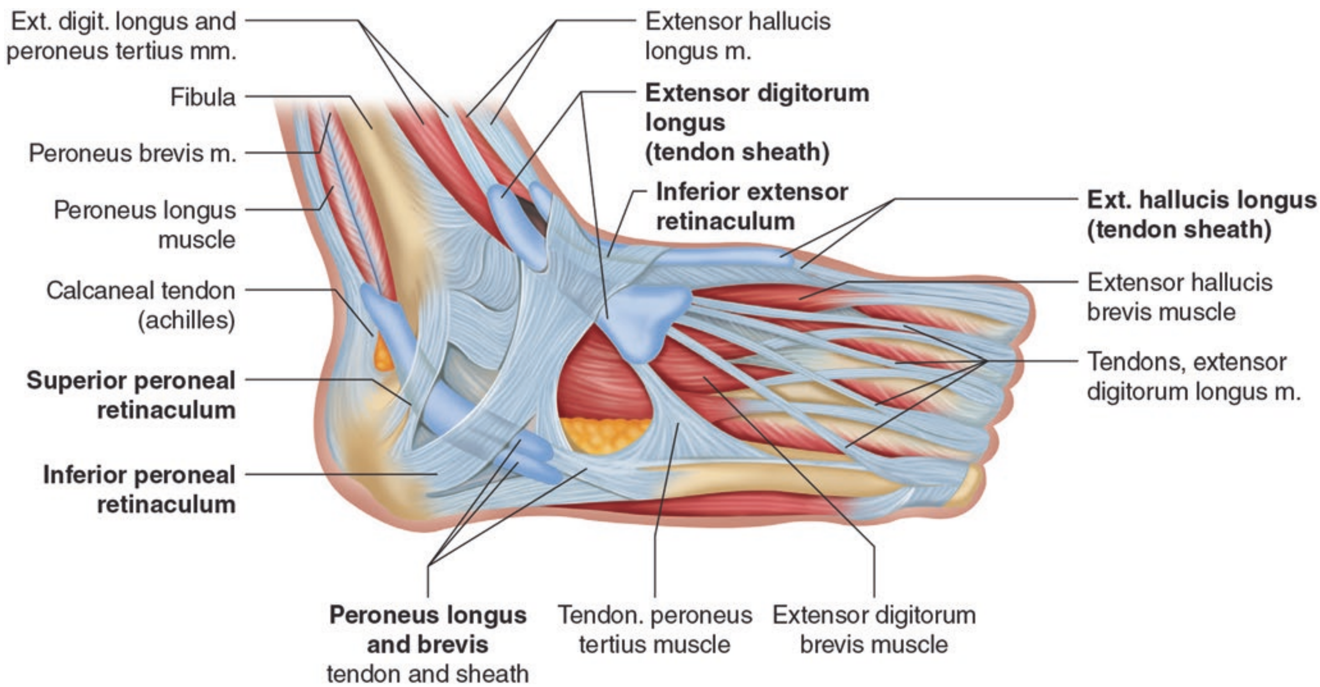

Fig. 34.21 Peroneal tendon anatomy

Indications for Orthopedic Referral

Though conservative treatment is usually advised primarily, surgical repair has been shown in some studies to have better long-term results for acute or recurrent subluxations [65]. Surgical repair allows for quicker return to normal activity and athletics with no instability. Operative management should be considered in high-level athletes due to recurrent nature of conservatively treated injury. Several viable techniques are currently being utilized by orthopedic surgeons.

Follow-Up Care

Following surgical or conservative management, physical therapy should begin, with an emphasis on eccentric training to promote new collagen formation [59]. Lateral heel wedges and ankle taping can help unload stresses on the peroneal tendon, but there is little evidence that this speeds healing from peroneal tendon injury. Surgical referral should be made in cases of failing conservative therapy, recurrent instability, or rupture of peroneal tendon.

Return to Sports

Mean time for return to play is 9 months following surgery, with postoperative immobilization lasting on average 6 weeks [67]. There is little consensus on the exact duration of immobilization and progression of therapy. However, there is consensus that dedicated physical therapy can improve recovery. Return to play following conservative treatment requires a minimum of 5–6 weeks of recovery, which may be prolonged if extensive immobilization is required.

Complications

Failed conservative therapy can lead to recurrent subluxation or dislocations of peroneal tendons. Patients may also have chronic pain and disability. Approximately half of nonsurgically treated cases fail conservative management [67].

Tibialis Anterior and Posterior Tendons

Mechanism of Injury in Sports

The tibialis posterior tendon runs vertically posterior to the medial malleolus and then has a horizontal path along the plantar aspect of the foot, assisting in plantar flexion, as well as adduction and supination, respectively (Fig. 34.22). The tendon also assists in maintaining the medial longitudinal arch of the foot [59, 68]. Patients with injuries to the tibialis posterior often cannot recall an acute trauma, though there usually is a precursor event (e.g., twisting foot, slipping, stepping into hole) in the history [59].

The tibialis anterior originate along the lateral aspect of the tibia crossing over and inserting on the middle cuneiform and first metatarsal, serving as the main dorsiflexor of the foot, along with assisting in adduction and inversion. Most injuries are from chronic overuse, though acute injuries can occur in activities that can cause forced dorsiflexion of a foot being held in plantar flexion, such as in football and soccer [59].

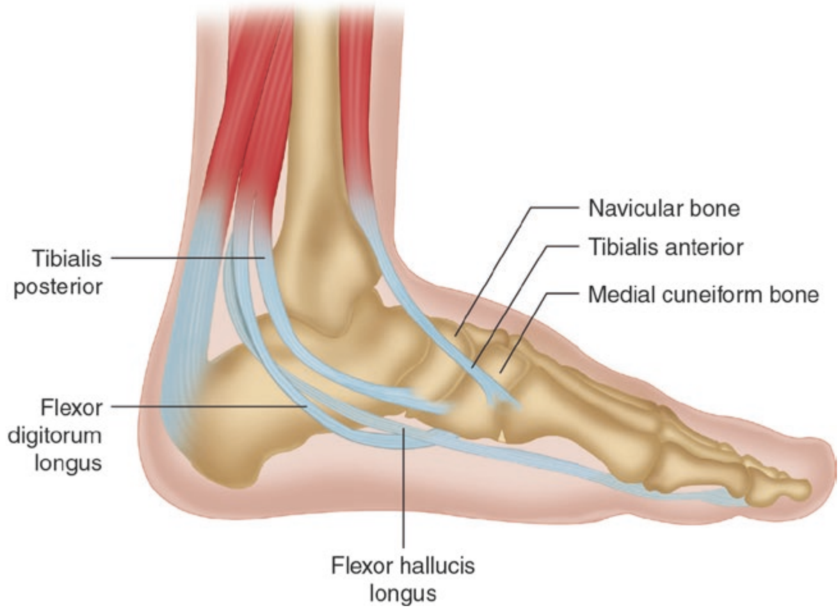

Fig. 34.22 Anatomy of tibialis anterior and posterior

Epidemiology

Ruptures of the tibialis posterior result from progression of chronic dysfunction of the tendon, usually affecting women more than men, with patients often being older than 40 years old [59]. Ruptures of the tibialis anterior are relatively rare with only case reports in the literature [69]. Most patients are usually in their 50–60s, if not older [59, 69].

Clinical Presentation

Patients with injury to the tibialis posterior usually present without specific recall of a mechanism of injury. Often times these patients are diagnosed as having a medial ankle sprain. Symptoms consist of pain and swelling along the course of the tendon, often progression over months to years. Ruptures can result in decrease in plantar flexion and inversion [59].

Injury to the tibialis anterior can present as pain with active or resisted dorsiflexion, with pain and swelling along the course of tendon or muscle body. In cases of ruptures, patients may present with a flat-footed, slapping gait [59, 69].

Diagnosis

Usually diagnosis can be made from clinical evaluation. For both tibialis tendons, pain and swelling may be found along the course of tendon and muscle body. Pain and limitations may also be found with active or resisted movements of each respective tendon. Dysfunction of the tibialis posterior tendon can be further broken down into stages [59, 68]:

1. Pain and swelling of tendon, able to perform single-leg heel raise, and no foot deformity.
2. Pain and swelling of tendon, unable to perform single-leg heel raise, pes planus, midfoot abduction, and subtalar joint is flexible.
3. Same as stage 2, but subtalar joint is fixed, arthritic.

Plain radiographs are not required for evaluation, though can help with finding any associated dysfunction. Ultrasonography can aid in dynamic visualization of the tendons but is largely operator dependent.

Initial Management

Initial management consists of pain control and RICE therapy. If suspecting rupture of either tibialis posterior or anterior, place in short leg cast. Tendinopathy of the tibialis posterior can benefit from foot orthosis to decrease pronation [59].

Indications for Orthopedic Referral

Ruptures of all tendons should be referred to orthopedic surgery for evaluation [59, 68, 69]. Also consider referral of stage 2 or 3 tibialis posterior dysfunction [59, 68] or failed improvement in symptoms and function after 3 months of conservative management.

Follow-Up Care

Physical therapy, strengthening, and flexibility are the treatments of choice of chronic tendinopathies. Anti-inflammatory medications and treatments can also be helpful. Patients may benefit from supportive foot orthoses. If symptoms persist, patients can consider being placed in a walking boot for 2–3 weeks, progressing to only wearing boot with ambulation for an additional 2–3 weeks [59]. Steroid injections can also be helpful in tibialis anterior tendon sheath but are avoided in the tibialis posterior tendon sheath due to high risk of rupture [59]. Postoperative management can vary depending on type and extent of surgical repair.

Return to Play

Patient can progressively return to play after achieving improvements in pain and function or recovering appropriately from operative management.

Complications

Improperly treated injuries can result in progression of pain, loss of function, or increased risk of rupture or re-rupture.

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Muscles

35

Alicia Gustafson and Cory A. Newman

Key Points

- Muscle injuries represent over 30% of all sports-related injuries.
- Sickle cell trait is a risk factor for muscle cramps, compartment syndrome, and rhabdomyolysis.
- There is no clear return-to-play protocol after a muscle strain, so clinical judgment is warranted.
- A quadriceps hematoma should be immobilized in at least 120 degrees of flexion for the first 24 hours after injury.

Introduction

Muscle injuries are common in all ages and different types of sports. They represent over 30% of all sports-related injuries and are a major reason for missed playing time [19–21]. This chapter discusses the diagnosis, acute and long-term management, and prevention of some of the more common muscle injuries encountered in sports medicine. It also addresses rehabilitation and return-to-play guidelines following a muscular injury (Table 35.1).

Table 35.1 Types of muscle injuries and the commonly associated sport

Injury	Most common sport
Hamstring strain	Sprinting, football, rugby
Adductor strain	Soccer, hockey
Gastrocnemius strain	Tennis, football, basketball
Chronic exertional compartment syndrome	Running
Hematoma	Football, rugby, hockey
Rhabdomyolysis	Weight lifting, endurance sports

Muscle Strain

- Acute muscle strain is one of the most common problems encountered in athletics. A muscle strain is the overstretching or tearing of muscle fibers that usually occurs near the myotendinous junction [16, 27]. The degree of tearing can range from partial to full thickness and is given a grade I–III. A grade I strain is considered mild and consists of stretching of the muscle fibers. Grade II strains are moderate strains with partial tearing of muscle fibers. Grade III strains, which are the most severe, are a full-thickness tear or complete muscle rupture [16, 17, 19]. The mechanism of injury is often either due to trauma, such as a fall or direct blow, or due to a sudden, forceful eccentric contraction as seen with the quick starts in track and field events [1, 27].
- *Risk factors:* Reduced cross-sectional area of the multifidus at L5 predicted 85% of muscular injuries of the hip, groin, and thigh in 83% of elite Australian football participants [56]. Other factors include poor lumbopelvic control, difference in muscle size when comparing right to left extremities, repetitive use of the dominant limb during sports, asymmetries in flexibility and strength between agonists and antagonist muscle groups, previous history of injury, improper training ramp up between off-season and preseason, and insufficient warm-up prior to practice or game.
- *Clinical presentation:* The clinical presentation varies depending on the grade of the strain. The athlete will sometimes feel a pop or snap at the area of injury. The presentation for each grade of strain is listed below [16, 19, 27].
  – Grade I – pain with localized tenderness, possible mild swelling and bruising. The nerve and blood vessels are still intact; therefore, healing is accelerated. Examples include blunt trauma, minor surgical procedures, and

A. Gustafson (✉)
Northwest Allied Physicians, Tucson, AZ, USA

C. A. Newman
Proactive Physical Therapy, Tucson, AZ, USA

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eccentric/concentric muscle injuries. The athlete may be able to continue playing.

  – Grade II – more severe pain and tenderness with hemorrhage, swelling, weakness, and impaired function. Injury includes the soft tissue and nerve, but vasculature is intact. The extracellular matrix is also intact, so healing is accelerated in these instances as well.
  – Grade III – severe and diffuse pain often causing the athlete to collapse after injury. There will likely be a palpable defect and significant loss of strength and function. The muscle, extracellular matrix, nerve, and blood vessels are damaged, thus prolonging the healing time and recovery. In grade III strains, the muscle has lost the ability to regenerate; thus, histological findings demonstrate a predominance of collagenous scar formation rather than muscle tissue.
- *Diagnosis:* A muscle strain is a clinical diagnosis determined by a thorough history of the injury and complete physical examination. Inspection, palpation, range of motion (ROM), and strength testing can often give you a sense of the severity [21]. Grade I strains do not require imaging unless there is no improvement. Imaging is helpful in grade II and III strains to assess the severity of the damage and the degree of tearing. Ultrasound is a very efficient and effective means to acquire post-injury information either on the sideline or in the training room. Direct visualization of edema, hemorrhage, and muscle fiber disruption can determine the grade and dictate management. Ultrasound can also be used to monitor resolution of the injury and functional dynamics. An MRI may also be obtained for finer details and better structural assessment of the involved muscle(s), tendons, and bone [21, 27].
- *Management:* The timing, speed, and intensity for applying movement and resistance exercises are individualized and dependent on age, severity of injury, and pain tolerance. In the acute stage, the acronym PRICEM "(P for protect, prevent, and promote healing; R for relative rest; I for ice or cryotherapy; C for compression; E for elevation; and M for modalities, medication, massage, mobilization, and movement)" should be followed [31]. There is conflicting evidence about the use of anti-inflammatory medications due to the possibility of slowing the healing process; therefore, the use of NSAIDs is situational and clinician-dependent [16, 18, 22, 27]. Restricted movement of the injured muscle is encouraged, as excessive stretching of injured muscles has been proven to result in excessive scar tissue formation and impaired muscle fiber regeneration [35]. Initial exercises performed should not elicit pain [27], but as pain and muscle function improve, rehabilitation can progress accordingly. Autologous blood product injections like platelet-rich plasma (PRP) are another possible management option. Results of PRP injections are anecdotal as there is insufficient human data to support the use at this time [23]. Most strains can be managed conservatively; however, surgical intervention is considered for cases that fail conservative management, most notably hamstring tears and complete ruptures [28, 29].
- *Return to play:* There is no clear consensus for when a player can return to play safely after a muscle strain. Ideally, an athlete would return to sports once they are pain-free and have regained full muscle strength and function [1, 16, 18, 27], but this is often not realistic as complete healing can take several months following grade II strains and possibly up to 1 year in complete tears. Therefore, the timing of when an athlete is safe to return to play is a group decision between the athlete, athletic trainers, therapists, and physicians. Muscles strains have a high rate of recurrence, and the exact cause is unclear due to the multifactorial nature of the injury. Some possible contributing factors are a reduction of muscle strength secondary to the injury [55], inappropriate rehabilitation methods such as not emphasizing eccentric strengthening and/or noncompliance of the patient, scar tissue reducing the extensibility of the musculotendinous unit resulting in decreased flexibility of the injured muscle, and possible alteration in biomechanics and neuromotor firing patterns [18, 27]. Also worthy of consideration is the athlete not completing a full rehabilitation program due to external pressures to return to the field prematurely.
- Some of the more common muscle strains, the rationale for increased occurrence, diagnosis, and appropriate rehabilitation methods for recovery, are described below.
  – *Hamstring* – Hamstring strains are some of the most common soft tissue injuries in athletes and the third most common hip injury in the sports medicine population, behind only gluteus medius tendinitis and trochanteric bursitis [31]. The hamstring is a two-joint muscle originating at the ischial tuberosity and inserting distal to the knee joint on both the tibia and fibula (Fig. 35.1). The hamstring is responsible for eccentrically controlling knee extension and hip flexion. The hamstring is comprised of three muscles: biceps femoris, semitendinosus, and semimembranosus, the former being the most commonly injured of the three due to the varying nerve supply. The short head of the biceps femoris is innervated by the peroneal division of the sciatic nerve, while the long head of the biceps femoris is innervated by the tibial division of the sciatic nerve. This varying nerve supply results in poor neuromuscular control ultimately leading to increased injury occurrence [27, 31]. Clinical presentation typically demonstrates a sub-optimal length-tension relationship due to postural asymmetries whereby the

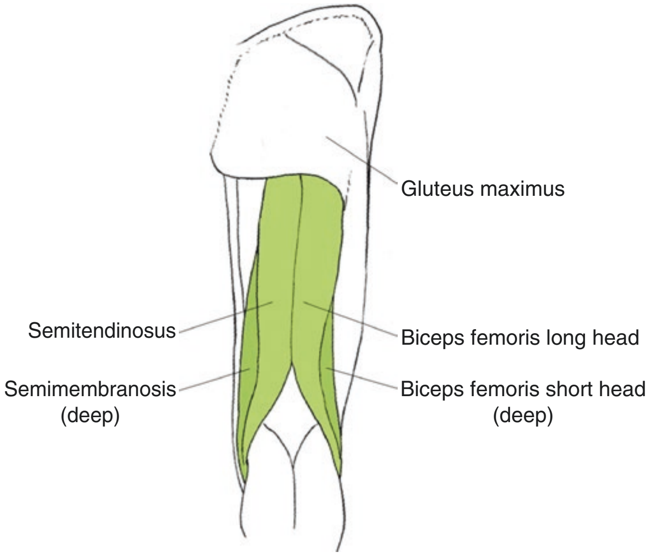

Fig. 35.1 Anatomy of the hamstring

hamstring is overly shortened and the quadriceps, antagonist, is overly lengthened. As a result of the shortened position, the hamstring cannot attenuate the eccentric forces of decelerating the leg during the terminal swing phase of gait, as is required during quick powerful movements (i.e., sprinting track events), and is consequently strained or torn.

Diagnosis – As noted, the biarticular nature of the hamstring requires multiple leg positions when testing strength and performing pain provocative tests. With the patient prone, and in 0 degrees of hip extension, knee flexion resistance can be applied at both 15 and 90 degrees of knee flexion. This resistance can be applied, while the leg is externally rotated to isolate the biceps femoris and internally rotated to bias the semimembranosus and semitendinosus muscles.

Rehabilitation guidelines are divided into three phases with specific goals and criteria in place before advancement to the next phase is indicated:

- During phase I (days 1–5), the goal is to minimize pain and edema and allow appropriate soft tissue healing with progression to phase II beginning when the following criteria are met: (1) non-antalgic walking pattern, (2) non-painful slow jog, and (3) pain-free submaximal isometric contraction (50–70%) with knee flexion at 90 degrees [27].
- During phase II gentle stretching in a pain-free range is incrementally increased, while also introducing higher-level core, lumbopelvic strengthening, and sports-specific agility drills that can be performed at higher speeds and intensities once neuromotor control is observed. Progression to phase III is achieved once the following criteria are met: (1) no pain during manual muscle testing of the injured muscle during maximal isometric contraction performed at 90 degrees knee flexion with a score of 5/5 and (2) able to run forward and backward at 50% maximum speed [27].
- Phase III, sports-specific drills incorporating functional movement patterns are emphasized in this stage. Explosive plyometric, ballistic, and quick change of direction activities should be avoided until the athlete attains back to sports criteria.

Return to sports criteria:

- Time frame for recovery is multifactorial and is dependent on the severity of the injury based upon MRI findings and clinical presentation (bruising, ROM, strength). Askling et al.. demonstrated that proximity of the injury to the ischial tuberosity is correlated to prolonged recovery time [34].
- The athlete should have full ROM and strength and be able to perform running, cutting, twisting, and jumping activities without pain or tightness. If isokinetic testing is available, there should be a less than 5% difference when comparing the involved and uninvolved sides [27].
- Brooks et al. have found there may be a link between lower incidences and severities of hamstring injury in athletes during training and competition if compliant with the Nordic hamstring protocol [36] (Table 35.2).

– *Gastrocnemius* – Strains of this muscle are referred to as "tennis leg" and frequently seen in sports that require rapid quick cutting movements in all directions. The most common mechanism of injury is forced ankle dorsiflexion while the knee is extended [31]. The triceps surae, i.e., calf muscle, is responsible for deceleration of the tibia and fibula in terminal swing phase of gait and plantar flexion of the ankle. The triceps surae is composed of the gastrocnemius and soleus muscles. The gastrocnemius, like the hamstring, is a two-joint muscle comprised of a medial and lateral head, the former originating on the medial condyle of the femur, the latter with an origin on the lateral condyle of the femur. Both medial and lateral heads conjoin to form the gastroc-soleus complex which inserts into the calcaneus via the Achilles tendon. The gastrocnemius is innervated by the tibial nerve.

Table 35.2 Nordic hamstring protocol

State of injury	Duration	Intervention	Progression criteria
Acute	1–3 days	Protect injured tissue Rest in lengthened position Non-antalgic gait pattern Ice, compression, and elevation	Passive SLR to 80 degrees with <3/10 pain
Subacute	3 days to >3 weeks	Gentle static stretching Isometric and concentric exercise begin 30% 1 rep max (RM) of uninjured leg, 3–4 sets × 10 reps Massage Cardio (UBE, stationary bike, etc.)	Perform 60% 1 RM of non-injured leg
Remodeling phase	1–6 weeks	Begin more aggressive concentric (seated hamstring curls 60–80% 1 RM) Eccentric hamstring curls and/or isokinetics from high to low speeds in pain-free range of motion Nordic hamstring exercise Plyometric progression (high knees, butt kicks, leg swings, box jumps)	Completion of all activities with <3/10 pain Perform 10 pain-free Nordic hamstring exercises Complete running progression
Return to activity	2 weeks to 6 months	17-level progression from walking to jogging to running and finally sprinting with presence or absence of soreness being indicator for regression or progression to next level High-intensity plyometrics Agility and sports-/position-specific drills	

Diagnosis – There is conflicting evidence concerning the ideal knee position when differentiating which muscles of the triceps surae are injured. Hebert-Loiser et al. demonstrate that the standing heel raise test should be performed for at least 40 times with the knee positioned between 0 and 5 degrees of knee extension to assess the strength of the gastrocnemius [45]. However, the same author concluded that heel raise testing at 0 degrees and 45 degrees of knee flexion was not enough to differentiate the varying muscles of the triceps surae [46]. Anecdotally, it has been observed that having the patient perform a single-leg heel raise in standing at 0–5 degrees of knee extension is sufficient to isolate the gastrocnemius. Resistive testing of ankle plantar flexion while seated with the knee flexed at 90 degrees is sufficient for isolating the soleus muscle. During the test, the clinician needs to be aware of signs for fatigue such as change in knee angle, reduced heel height, pace of performing a heel raise, loss of balance, or forward weight shift.

Return-to-play guidelines:

- The circumferential measurement 10 cm distal to the tibial tubercle is within 10% of the uninjured leg.
- The maximum number of single-leg heel raises is within 10% of the uninjured leg.

– *Adductor strain* – Adductor injuries are common among athletes due to the physical nature of the sports they participate in, especially contact sports requiring excessive cutting, twisting, and explosive movement patterns that involve sudden stopping and starting, as seen in football, hockey, and soccer. Also, sports that involve repetitive rotational forces such as ballet and martial arts have a high incidence of injury. Factors that contribute to hip injuries include female gender, previous injury, age, and generalized hip weakness. Arnanson et al.. demonstrated that athletes with increased fat content and reduced adductor flexibility were at risk for injury and those suffering from previous groin injury were 5× more likely to injure the same area [60].

The hip adductors are composed of five muscles that originate along the pubic symphysis, pubic crest, and inferior pubic ramus. The pectineus inserts on the posterior-medial surface of the femur just distal to the lesser trochanter and is innervated by the femoral nerve. The other four muscles are innervated by the obturator nerve: adductor brevis, adductor magnus, gracilis, and adductor longus, the latter being the most commonly injured [59]. The adductor brevis, longus, and magnus have insertion points along the linea aspera of the femur. The gracilis is a two-joint muscle inserting on the pes anserine (Fig. 35.2).

Diagnosis – The anatomical nature of the pelvis and the biomechanical relationship between the abdomen and hip make differential diagnosis challenging. Athletic pubalgia needs to be in the differential when diagnosing a groin strain. Kachingwe et al. have identified five signs for athletic pubalgia [61]:

1. Subjective complaint of deep groin pain.
2. Pain is exacerbated with exertion and relieved with rest.
3. Tenderness to palpation along the pubic ramus at site of rectus abdominis insertion.
4. Pain with resisted hip adduction at 0, 45, and 90 degrees of hip flexion.

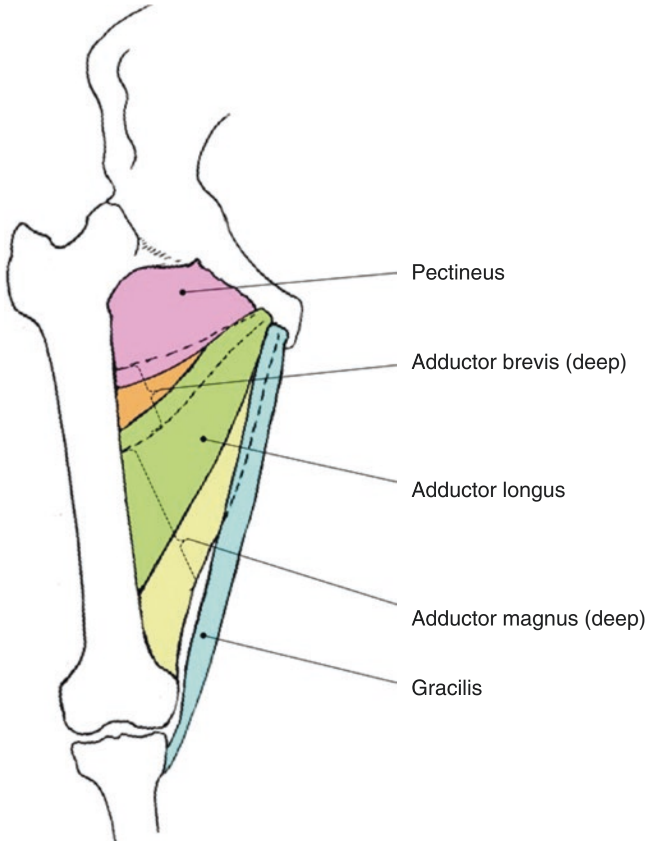

Fig. 35.2 Anatomy of the hip adductors

5. Pain with resisted abdominal crunch.

A dull aching pain is more indicative of an adductor strain compared to a deep-seated groin pain being a more reflective sign of athletic pubalgia.

Rehabilitation – Generalized rehabilitation methods mentioned previously can be implemented in the instances of an adductor strain. A broad range of return to sports time frames exist in the literature ranging from 5 weeks for surgical repair of the adductor longus in an elite athlete [62] to 10–52 weeks for adductor longus rupture managed conservatively in a college soccer player [63]. Basic biomechanical principles should be implemented when designing a return to activity progression. Each activity must be performed pain-free prior to initiating the next stage of higher-intensity activity. The progression follows the "walk before you can run" mantra and begins with pain-free walking and then progresses to the following activities in order of intensity: jog, run, sprint, cutting/pivoting, kicking, and finally return to normal practice session.

Other rehabilitation milestones that should be considered with return to field readiness include hip adduction strength deficits no greater than 10% compared to the uninvolved leg [64, 65] and hip adduction to abduction strength ratio of at least 90% [65]. These measures are best obtained with a handheld dynamometer as it has been indicated that even small changes in strength can be monitored [57].

Return to play – There are three return-to-play indicators [8]:

1. The athlete completes a running distance based on average running performed in a game.
2. Five minutes of skating on a 3 m slide board.
3. Three sets of 12 repetitions of hip adduction with 6 kg of resistance.

During the rehabilitation process, it is imperative to bear in mind the continued assessment of lumbar spine mobility, hip flexibility, ability to maintain appropriate posture during static and dynamic sports movements, and especially overall core strength as a delayed activation in the transverse abdominis muscle was frequently observed in athletes suffering from adductor strains [56].

Contusion/Hematoma

- A contusion is a bruise that is the result of a direct force or a repetitive force often seen in contact sports. The underlying blood vessels and muscle fibers at the area of impact are disrupted without damage to the overlying skin. Most contusions are subcutaneous. If significant damage to the deep vessels and muscle fibers occurs, an intramuscular hematoma can develop. A hematoma is an abnormal collection of coagulated blood outside of a blood vessel [2]. Another diagnosis to consider if there is persistent or worsening subcutaneous fluid collection at the site of injury is a Morel-Lavallee lesion which is often mistaken for a hematoma or contusion. A Morel-Lavallee lesion is generally due to a shearing force that causes the skin and cutaneous tissue to separate from the underlying fascia which leads to disruption of the lymphatics and blood vessels. It is also referred to as internal degloving [24–26]. Clinical presentation of a Morel-Lavallee lesion is a palpable, soft, fluctuant mass at the site of injury. Ultrasound will show hypoechoic/anechoic fluid with blood, lymph, and fat [24–26]. Management consists of aspiration, compression, and surgical intervention.
- *Risk factors:* The major risk factor for obtaining a contusion or hematoma is participation in a contact sport. Athletes who have a bleeding disorder such as hemophilia or are taking a blood thinner are also at a much higher risk.
- *Clinical presentation:* Contusions present with pain, tenderness, discoloration, and possible swelling at the site of injury. A hematoma will present similarly but may have

redness and warmth at the site of trauma [2]. Signs and symptoms can range from mild to severe. Mild contusions will have minimal pain and normal to slightly diminished range of motion [54]. If the lower extremity is mildly injured, that athlete's gait will still be normal. A severe contusion would be a hematoma with extreme pain and significant loss of range of motion [54]. Again, if the injury was to the leg, the athlete would have a severely antalgic gait. Moderate contusions fall in between [54].

- *Diagnosis:* Contusions and hematomas are a clinical diagnosis based on the mechanism of injury and the athlete's physical exam findings. Imaging generally is not needed in the acute setting unless an underlying fracture is suspected. In the subacute setting, ultrasound can be useful to further diagnose a hematoma and determine the size. A hematoma in the early phase will show hypoechoic fluid throughout the injured muscle that is generally well demarcated. Subacute and chronic hematomas will show more lobulations within the fluid as the blood coagulates. Ultrasound will also likely show calcifications if heterotopic bone formation is occurring within the hematoma. An MRI may be warranted to rule out other muscle pathology such as an avulsion or strain [54].
- *Management:* The initial management of contusions and hematomas is the same as for most other injuries: cryotherapy, rest, and compression. Resting the injured muscle helps to limit the size of the hematoma which will prevent further gaping within the muscle and reduce surrounding scar tissue [51]. Compression reduces intramuscular blood flow [51]. In the case of a quadriceps hematoma, the injured extremity should also be immobilized as soon as possible with the knee flexed to at least 120 degrees for the first 24 hours [54]. Keeping the knee flexed allows the quadriceps to remain elongated which helps prevent further hematoma formation and decreases the risk of developing myositis ossificans. Analgesics, such as NSAIDs, can be used in the acute setting but should not be used long-term due to their unknown effect on muscle healing. The use of a NSAID for at least the first 7 days in an athlete with a hematoma is encouraged to prevent the formation of myositis ossificans [54]. Most hematomas will resolve with conservative treatment; however, larger hematomas may require more invasive interventions such as percutaneous drainage or debridement [2, 24, 51, 52].
- *Return to play:* There is no clear return-to-play protocol after a contusion or hematoma. General guidelines include returning to play once an athlete has regained near normalization of the anatomic or physiologic deficit due to the injury and is able to perform sports-specific skills. The athlete should also feel comfortable returning [32]. An athlete may be able to return the same day after sustaining a contusion if it is not too severe. The use of protective padding may help to prevent recurrence [54].
- *Complications:* Some potential complications of a severe hematoma are myositis ossificans, compartment syndrome, and infection. Myositis ossificans, also known as heterotopic ossification, is generally seen with a quadriceps or gluteus hematoma. It is a tumorlike, non-neoplastic lesion consisting of proliferated bone and cartilage that develops at the site of the hematoma [53, 54]. It should be suspected in an athlete who has a hard, more painful mass at the hematoma site a few weeks post-injury. Early in its course, myositis ossificans may be visualized on radiography. Ultrasound will show shadowing of the cortical bone as it forms [53]. The diagnostic test of choice is a CT scan to evaluate mineralization and rule out any other etiologies such as malignancy or infection [53, 54]. Athletes can participate with a heterotopic ossified lesion but may experience flare-ups of pain and some loss of range of motion. Most cases ultimately require surgical intervention. This occurs once the lesion is completely mature, which is around 12 months on average [53, 54]. If the ossified lesion is removed before it is matured, recurrence is likely.

If there is a significant amount of intramuscular edema and swelling following a contusion or hematoma, the risk for developing compartment syndrome arises. The increase in compartment pressure leads to severe pain often out of proportion to exam. A hematoma is also potentially a nidus for infection, so close monitoring for increased redness, warmth, and fever is warranted.

Muscle Cramps

- Skeletal muscle cramping can be the result of numerous underlying medical conditions. The most common reason for cramping among athletes is exercise-associated muscle cramps (EAMC) [3, 7]. It is a painful, involuntary muscle contraction that can occur during or immediately after exercise. The exact etiology is unclear and controversial, but it is hypothesized that electrolyte disturbances, dehydration, and muscle fatigue causing altered neuromuscular control are amid the main contributing factors [3, 4, 7, 15]. In more recent years, it is believed that the electrolyte losses and dehydration theories are actually less likely causes of EAMC and that the more likely etiology is an imbalance between the inhibitory and excitatory drives at the spinal level [3–7, 15]. However, more conclusive research needs to be conducted in order to determine the primary pathophysiology.

Because various medical conditions and medications can make athletes more susceptible to cramping, obtain-

ing a thorough medical history is vital. It is important to consider a workup for an underlying etiology such as a metabolic or neurologic disorder in athletes with recurrent episodes of muscle cramping. Some examples of causes are diabetes, thyroid dysfunction, radiculopathy, or plexopathy [5–7]. Athletes with sickle cell trait and disease are also more prone to cramping. Exercise leads to hyperthermia, hypoxemia, and an acidotic state in the muscle which can provoke sickling of red blood cells [9–11, 48]. Sickled RBCs cause vaso-occlusion in small vessels leading to muscle pain and cramping. Athletes with sickle cell trait or disease who develop cramping need to cease physical activity and notify medical personnel immediately to prevent progression to a life-threatening condition [9–13].

- *Risk factors:* Multiple risk factors are believed to contribute to exercise-associated muscle cramps. Previous episode of EAMC, increased exercise intensity and duration, previous muscle injury, hot and humid environment, and physical deconditioning are just a few examples [3, 7, 15].
- *Management:* Static stretching is the most effective treatment for EAMC [5–7] and can be done immediately on the sideline. Hydration and electrolyte replenishment are still mainstays of management. Oral hydration is preferred over intravenous. Medications such as benzodiazepines, magnesium, and calcium can be used for severe cramping although these are not usually recommended due to the potential complications [7, 15]. In general, transfer to a tertiary care center should be considered in any athlete with persistent or worsening muscle cramping due to the risk of developing rhabdomyolysis. The threshold for transferring those with sickle cell trait should be low because of the numerous potential serious complications.
- *Return to play:* Many athletes with EAMC can return to sports the same day once the cramping has been alleviated with rest, stretching, and fluids. However, ultimately return to play requires clinical judgment based on the severity and cause of cramping [15].
- *Prevention:* Prevention consists of identifying and addressing the risk factors for every athlete. This includes adequate, regular stretching and muscle conditioning (plyometric exercises); appropriate nutrition and hydration before, during, and after exercise with modifications based on extrinsic factors (i.e., heat, humidity, altitude); and monitoring the duration and intensity of physical activity [5, 7, 15]. In athletes with recurrent EAMC, keeping a journal may be beneficial to identify and avoid triggers [4]. It is also important to educate athletes with sickle cell trait and disease, about their condition and warning signs during exercise.

Compartment Syndrome

- Compartment syndrome is a condition where the pressure inside the compartments of the extremities increases causing pain and symptoms of neurovascular compromise such as muscle weakness or claudication. The condition can be acute or chronic. Acute compartment syndrome (ACS) is a medical emergency requiring immediate recognition and intervention. ACS usually occurs after a trauma or prolonged immobilization. The injury to the extremity causes increased fluid accumulation within a compartment raising the pressure [38, 39]. It can be a complication of a fracture, muscle rupture, or hematoma. Nontraumatic causes of ACS are rhabdomyolysis and sickle cell trait. Acute compartment syndrome should be suspected in patients who are being treated for rhabdomyolysis where a second spike in their creatine kinase is observed [32, 33].

  Chronic exertional compartment syndrome (CECS) is more of an insidious onset with an unclear etiology causing it to often be misdiagnosed [37]. A few possible hypotheses are decreased fascial compliance, muscle hypertrophy, abnormally thickened fascia, or inappropriate muscle swelling during activity [50]. The incidence is equal between males and females and usually occurs in athletes in their early 20s. Ninety-five percent of cases involve the lower extremity [37]. The majority of cases are bilateral and occur with running [37]. Acute exertional compartment syndrome is also possible but is extremely rare.
- *Clinical presentation:* With chronic exertional compartment syndrome, the onset of pain is often predictable with a certain intensity and duration of exercise. There can also be sensory changes and muscle weakness [37]. Symptoms will improve or completely resolve with rest [43]. The clinical presentation of acute compartment syndrome will depend on the affected compartment. Pain, often out of proportion with the exam, will be present in almost all cases. Listed below are the major compartments of the upper and lower extremities and their contents.
  - *Lower leg* (Fig. 35.3)

    Anterior compartment – most common site for ACS. It contains the tibialis anterior, extensor hallucis longus, extensor digitorum longus, tibial artery and veins, and deep peroneal nerve [38–40]. Signs of compartment syndrome in this compartment would be decreased sensation at the first web space, pain with passive plantar flexion, and weakness of dorsiflexion. Late sequelae are foot drop and claw foot [38].

    Lateral – peroneus brevis and longus, peroneal artery, superficial peroneal nerve, and common

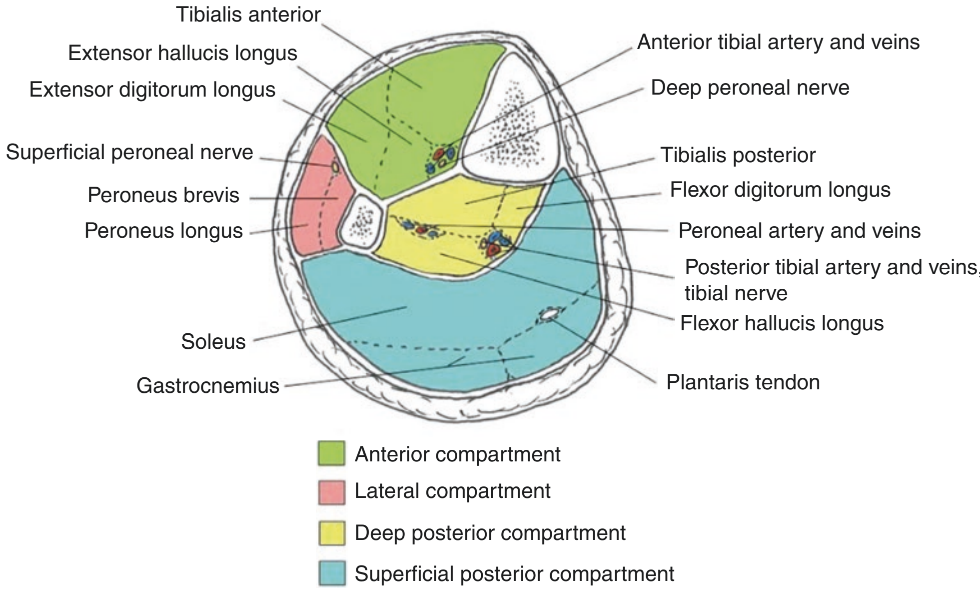

Fig. 35.3 Cross-sectional anatomy of the lower extremity

peroneal nerve [38–40]. Signs include weakness of eversion, inversion, dorsi- and plantar flexion, and decreased first web space sensation [38].

Superficial posterior – least likely to develop ACS. It contains the gastrocnemius, plantaris, and soleus which are palpable and painful on exam [38–40].

Deep posterior – posterior tibial artery/vein, peroneal artery/vein, tibial nerve, tibialis posterior, flexor hallucis longus, and flexor digitorum longus [38–40]. Presentation would be plantar hypesthesia, weakness of toe flexion, and pain with passive extension of toes [38].

– *Upper leg*

Anterior – rectus femoris, vastus medialis, vastus intermedius, vastus lateralis, and cutaneous branches of the femoral nerve and saphenous vein [40]. Signs would be weakness with knee extension, pain with passive knee flexion, and sensory changes of the thigh [38].

Posterior – biceps femoris, semitendinosus, and semimembranosus. Presentation would be pain with passive knee extension, weakness of knee flexion and plantar flexion of the ankle, and sensory deficits of the peroneal nerve [38].

Medial – adductor magnus, longus, brevis, and minimus; gracilis, pectineus, and obturator externus [40]. Signs may be weakness of hip adduction, pain with passive hip abduction, and sensory deficit of the obturator nerve [38].

– *Forearm*

Superficial volar – flexor carpi ulnaris, flexor digitorum superficialis, flexor carpi radialis, and pronator teres. The volar compartments also contain the median artery and nerve and the ulnar artery and nerve [40]. These two compartments (superficial and deep volar) are at higher risk for developing ACS following trauma [38].

Deep volar – flexor digitorum profundus and flexor pollicis longus [38–40].

Dorsal – supinator, extensor carpi ulnaris, extensor digitorum, extensor pollicis longus and brevis, abductor pollicis longus, and radial and posterior interosseous nerve and artery [40].

Lateral – brachioradialis and extensor carpi radialis brevis and longus [40].

– *Upper arm*

Anterior – biceps brachii, brachialis, median, and ulnar nerves.

Posterior – triceps and radial nerve.

- *Diagnosis:* The diagnosis of compartment syndrome is based on history, physical exam, intracompartmental pressure testing, and a high clinical suspicion. The five Ps (pain, pallor, paresthesias, pulselessness, paralysis) that are classically associated with the diagnosis of compartment syndrome are a misconception [38]. Pain, sometimes out of proportion of exam, is the symptom most commonly associated with compartment syndrome. Paresthesias can sometimes occur in the early stages [39]. However, pallor, pulselessness, and paralysis are all late findings, and ACS usually is diagnosed before this time. A manometer is used to measure intracompartmental pressure. The normal pressure in a compartment is 0–8 mmHg. Pain will usually develop with a pressure >20 mmHg [38]. In the acute setting, if a pressure is within 30 mmHg of the diastolic pressure, then emergent fasciotomy is warranted [38, 39]. This equates to a delta pressure of <30 mmHg. There is some dispute about what the cutoff pressure should be [37–39]. Chronic exertional compartment syndrome is diagnosed

when symptoms are present and any of the following criteria are met: a pre-exercise pressure of greater than or equal to 15 mmHg, a 1 minute post-exercise pressure of 30 mmHg or greater, or a 5 minute post-exercise pressure of 20 mmHg or greater [37, 50].

- *Management:* Management of acute compartment syndrome first begins with having a high clinical suspicion in patients at risk [38]. The affected limb should not be elevated as blood flow to it is already compromised [39]. If the exam is indicative of ACS with an intracompartmental pressure within 30 mmHg of the diastolic pressure, then emergent fasciotomy is the definitive treatment [38, 39]. In some cases, the patient can be observed for several hours with serial examinations and pressure testing. If the exam is improving and the pressure readings are decreasing, then a fasciotomy may not be needed [38, 39]. The definitive treatment for CECS is also fasciotomy [37, 39, 43]. Many athletes will often discontinue any physical activity that causes symptoms in lieu of surgical intervention. Nonoperative management in CECS has been unsuccessful in the past, but potential newer treatments are being studied with promising results. Some examples are physical therapy that aims to reduce myofascial restrictions and improve neuromuscular function and motor control deficits [37], modifying running mechanics to avoid heel striking [43], and botulinum toxin injections [42].
- *Return to play:* There are no strict return-to-play guidelines after compartment syndrome. It is case dependent. The severity of the presentation and intervention dictate the recovery and rehabilitation. Acute compartment syndrome tends to have a longer recovery phase as the surgery is often more aggressive with more complications [39, 44]. An athlete may never return to their pre-injury level [39, 44]. Athletes with CECS can generally anticipate a gradual return to full sports in 6–12 weeks, on average, whether the treatment is conservative or surgical [49, 50]. Much like with returning after any injury, the athlete should have normal function and strength of the extremity and be able to participate in sports-specific drills. Recurrence of symptoms can occur regardless of the treatment but are more likely with nonoperative management. Recurrence rates range from 3% to 20% [37, 39, 47, 49, 50].
- *Complications:* Several serious complications can occur if there is a delay in diagnosis and treatment of acute compartment syndrome. Muscle necrosis occurs within the first 3–6 hours which leads to contractures and permanent deformity [39]. If the ischemic insult is severe enough, amputation may be required. Persistence of intracompartmental pressure also leads to rhabdomyolysis which causes myoglobinemia and potentially acute kidney injury [39]. Post-fasciotomy infection, particularly osteomyelitis, is another potentially serious complication [44]. Other complications after a fasciotomy are chronic pain, nerve damage, and muscle weakness.

Rhabdomyolysis

- Exertional rhabdomyolysis (ER) is the breakdown of muscle tissue secondary to a metabolic or mechanical insult such as strenuous exercise or normal exercise under various circumstances such as dehydration, supplement/medication use, high environmental temperatures, or recent viral illness [15, 30, 32, 33]. When the myocyte breaks, its intracellular contents are released into circulation [32]. Exertional rhabdomyolysis may also be the initial manifestation of an underlying genetic disorder. A genetic cause such as RyR1 mutation (which is the same gene involved in malignant hyperthermia), myopathy, or sickle cell trait should be considered in athletes with recurrent episodes or when the severity of rhabdomyolysis exceeds the expected response to the exercise performed [30, 32, 33]. The hyperthermic, acidotic, and hypoxemic environment created during exercise leads to sickling of RBCs in athletes with sickle cell trait. The sickled cells cause vaso-occlusion of muscle leading to muscle breakdown and rhabdomyolysis [48].
- *Clinical presentation:* Exertional rhabdomyolysis can range from mild to severe. Athletes with mild cases may be unaware and believe they are experiencing normal post-exertion muscle soreness and not seek medical attention. These milder cases are physiologic. More severe cases, with a likely pathological cause, present with extreme myalgias, muscle swelling and weakness, and "cola-colored" urine within 36 hours after exercise [30, 32, 33]. Consider rhabdomyolysis in athletes complaining of myalgias after performing repetitive eccentric exercises (squats, pull-ups, push-ups, etc.) with minimal rest periods or in those who are unaccustomed to the physical activity performed [30, 33]. Other potential signs and symptoms are fatigue, nausea, vomiting, and fever [30].
- *Diagnosis:* Rhabdomyolysis is a diagnosis determined by degree of symptoms and associated laboratory findings. The definition of the two types of exertional rhabdomyolysis are listed below:
  - *Physiologic ER*: Creatine kinase (CK) elevated at least five times the upper limit of normal within 36 hours after exercise with none or minimal muscle pain, with a maximum CK level at 3–4 days, followed by normalization within 2 weeks of no physical activity. Myoglobinuria may or may not be present. A urine dipstick that is positive for blood but no red blood cells are seen on microscopy is an indirect marker of urinary myoglobin. Also, ER is likely to be

physiologic if a known inciting factor is present such as certain medications, supplements, or recent viral illness and there is no contributing personal or family history [30, 32, 33].
  - *Pathologic ER*: Symptomatic elevated CK which would include any of the following features: severe myalgias, swelling, and/or weakness. There is presence of severe myoglobinemia and/or myoglobinuria either by urine inspection or by laboratory testing. If complications such as acute kidney injury, significant electrolyte abnormalities, and delayed recovery are present, it is more likely to be pathologic, and also, if the patient has a personal or family history of a potential genetic cause or myopathy [30, 32, 41].

  Ethnicity and gender play a role in what the athlete's baseline CK value is. These variables should be considered when determining if an athlete's CK is elevated. African American men and athletic men have the highest baselines, and non-African American women have the lowest [33].

- *Management:* The primary goal in treating rhabdomyolysis is preserving intravascular volume in order to maintain renal blood flow and organ perfusion [15]. Mild cases can be managed in an outpatient setting with rest, aggressive oral hydration, and serial monitoring of CK levels. However, an athlete complaining of muscle pain out of proportion to the exercise performed, muscle weakness, and dark urine should be sent to the emergency department for evaluation. Hospitalization will likely be required if there is symptomatic elevated CK, electrolyte abnormalities, elevated myoglobin with evidence of renal injury, or minimal urine output [41]. Intravenous fluid hydration should be given at a rate that maintains at least 200–300 mL/h of urine output [30, 41]. Dialysis may be warranted if the athlete is severely acidotic and has significant renal failure, hyperkalemia with cardiac arrhythmias, or no urine output even with adequate hydration [30, 41]. The normalization of the CK level will often lag behind the resolution of symptoms [14].
- Another possible treatment is dantrolene which has an effect on the calcium channels and muscle contractility [15]. This is not routinely used on the sideline as there is not enough research to support its efficacy [15].
- *Return to play:* There is currently no evidence-based return-to-play protocol after an athlete develops exertional rhabdomyolysis. The most important thing is to determine if there is a risk for recurrence. Guidelines were created to identify athletes who are at high risk for recurrence of exertional rhabdomyolysis [32, 41]. The guidelines are as follows [32, 58]:
  1. Greater than 1 week to recover while resting
  2. CK persistently elevated 5 times the upper limit of normal after 2 weeks of rest
  3. Acute kidney injury
  4. History of sickle cell trait
  5. Personal or family history of myopathy, recurrent muscle cramps, or complications after general anesthesia (malignant hyperthermia)

  If an athlete has any one of these risk factors, a further workup is warranted before they can be either cleared to return with close monitoring or disqualified. This would consist of genetic testing and possibly a muscle biopsy [32].

  Athletes who are at lower risk for recurrence and likely had physiologic exertional rhabdomyolysis can follow a return-to-play protocol. After the athlete has rested for 72 hours with aggressive oral hydration, a CK level should be rechecked. If it is less than five times the upper limit of normal and the athlete has normal urine output, then they can begin light activities. This should continue for 1 week. If they remain asymptomatic, gradual progression to sports-related activities can occur [14, 30, 32, 58].
- *Complications:* Many complications can arise from exertional rhabdomyolysis because of the contents that are released from the myocyte. A large quantity of myoglobin is toxic to the renal tubules causing acute kidney injury and potentially renal failure [30, 32]. Excess extracellular potassium can lead to cardiac arrhythmias and possibly cardiac arrest [15, 30, 32]. The release of free radicals may cause tissue edema increasing the risk for compartment syndrome [32]. Disseminated intravascular coagulation is another possible complication [15].

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Ligaments

36

Daphne Amanda Scott and Yvonne Chow

Key Points

- Acute ligament injuries are usually classified as grade 1 (sprains), grade 2 (partial tears), or grade 3 (complete tear).
- Complete healing through fibrosis and remodeling may take months to years.
- Management and outcomes vary substantially, depending on the site and severity of injury.
- Insufficient evidence exists to support routine use of regenerative therapy in the management of acute ligament injuries.

Introduction

Acute ligament injuries are common in sports. Undiagnosed or improperly treated ligamentous injuries can have profound effects on joint stability and function, making a timely and accurate diagnosis essential to patient care. Acute injury occurs following trauma to the joint, with an overloading force on the ligament. Injuries are typically graded by the degree of disruption to the ligament. Grade 1 injuries are stretch injuries without any resultant defect in the ligament. Grade 1 injuries are also termed sprains. Grade 2 injuries involve a partial tear through the tissue. Grade 3 injuries represent a complete tear (rupture) through the ligament [1]. Injuries of multiple ligaments may also occur at the same time, such as anterior cruciate ligament (ACL) and medial collateral ligament (MCL). Concomitant fractures may also be present, particularly in skeletally immature athletes. Joint stability becomes compromised following an acute ligament injury, leading to increased risk for subsequent injuries to surrounding structures such as the tendons, bone, and cartilage.

Ligament healing occurs through three phases: an early phase, a reparative phase, and a remodeling phase [2]. The early phase involves hematoma formation and initiation of cell proliferation. The reparative phase consists of fibrous filling of the defect. This phase can last for several weeks. While in the reparative phase, the ligament remains weak and susceptible to reinjury. In the remodeling phase, which can last months to years, fibrous tissue decreases and collagen increases at the injury site. Tensile strength improves over the span of months. However, even fully remodeled tissue remains different from normal, uninjured tissue both on a cellular and functional level [3].

Although the phases of healing are well described, different ligaments vary in their ability to heal. For example, MCL tears usually heal spontaneously, while ACL tears generally undergo reconstruction surgery [4]. Thus, the location and severity of a ligament injury can affect treatment and outcomes. Depending on the specific injury, treatment options include nonoperative management with rest and physical therapy, primary surgical repair, or surgical reconstruction with a replacement graft. Regenerative therapies such as prolotherapy, platelet-rich plasma (PRP), stem cells, and autologous plasma are increasingly applied to musculoskeletal conditions. However, successful outcomes for treatment of ligament injuries have been limited to case series and animal models [5–7]. Meta-analysis showed no benefit in outcomes, [8] and one study found that PRP may even be harmful for acute ligament injury in an in vivo animal model [9]. The current available evidence does not appear to support the routine use of these therapies in the treatment of ligament injuries.

Complications of ligament injuries extend beyond joint instability and dysfunction. While these structural effects alone can lead to time loss in sports and reduced athletic performance, athletes also report fear of reinjury and pain

D. A. Scott (✉)
Primary Sports Medicine, Hospital for Special Surgery, New York, NY, USA

Y. Chow
Department of Internal Medicine, Division of Sports Medicine, Albany Medical Center, Albany, NY, USA

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perception as factors preventing a return to pre-injury activity [10]. Thus, treatment of ligament injuries should address both physical and psychological aspects of recovery to ensure a successful return to sport.

A detailed discussion of injuries to specific ligaments, management guidelines, and return to sports recommendations follows.

Acromioclavicular Ligaments

Anatomy

- Acromioclavicular ligament [11]:
  - This is a part of the ACJ capsule.
  - This ligament resists anteriorly and posteriorly directed forces.
- Coracoclavicular ligament [11]:
  - Resists superiorly and axially directed forces
  - Also serves as a secondary restraint to superior displacement
  - Helps to main the apposition of the acromion to the clavicle
  - Contributes to the suspension mechanism of the scapula
- Coracoacromial ligament [11]:
  - Provides secondary stability to the joint
  - Composed of two strong bands with a thinner center (Fig. 36.1)

Mechanism of Injury in Sports

- This injury is typically seen after a fall onto the top of the shoulder, the acromion, and can be seen in contact sports such as hockey, football, and wrestling [11, 12, 25].
- Another mechanism includes a direct blow on the shoulder.

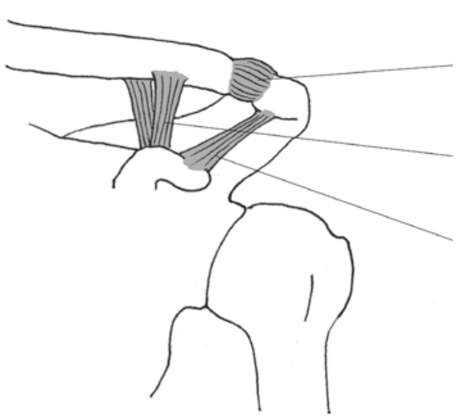

Fig. 36.1 Acromioclavicular ligaments

Table 36.1 Rockwood classification [11, 12, 25]

Grade	AC ligament	CC ligament	X-ray appearance
I	Intact	Intact	Normal
II	Partially disrupted	Intact	Widened AC appearance, <25% CC distance
III	Disrupted	Disrupted	Widened AC appearance, 25–100% CC distance
IV	Disrupted	Disrupted	Clavicle migrates into or through the trapezius muscle, increased CC distance
V	Disrupted	Disrupted	AC joint is dislocated, 100–300% CC distance
VI	Disrupted	Disrupted	Clavicle is displaced inferiorly; CC distance is decreased

Epidemiology

- An estimated 9% of shoulder girdle injuries involve damage to the AC joint [11, 12].
- Injuries typically occur in active young adults in the second–fourth decades of life (Table 36.1).

Classification

Clinical Presentation

- The patient typically presents with a history of trauma.
- Pain can be elicited with shoulder abduction and cross-body adduction.
- After an acute injury, there is typically tenderness to palpation at the acromioclavicular joint.
- Swelling and bruising can be seen in acute grade I–II injuries with gross deformity seen with grades II–VI [11, 12, 25, 26].

Diagnosis

- The diagnosis is typically made by history, exam, and radiographs [11, 25, 26].
- Xray: It is important to ask the patient to relax his or her shoulder; otherwise, the patient will keep his shoulder up for comfort which causes to underestimate the sprain's grade.
- AP, Zanca view (AP projection with 15° cephalic tilt) (Figs. 36.2 and 36.3)
  - Grade I: normal
  - Grade II: clavicular elevation relative to the acromion, but <25% displacement
  - Grade III–VI: 25–100% increase in the coracoclavicular interspace relative to the uninvolved shoulder
- CT can aid in evaluation of distal clavicle fractures.
- MRI is rarely indicated and it can help further characterize ligamentous injury.

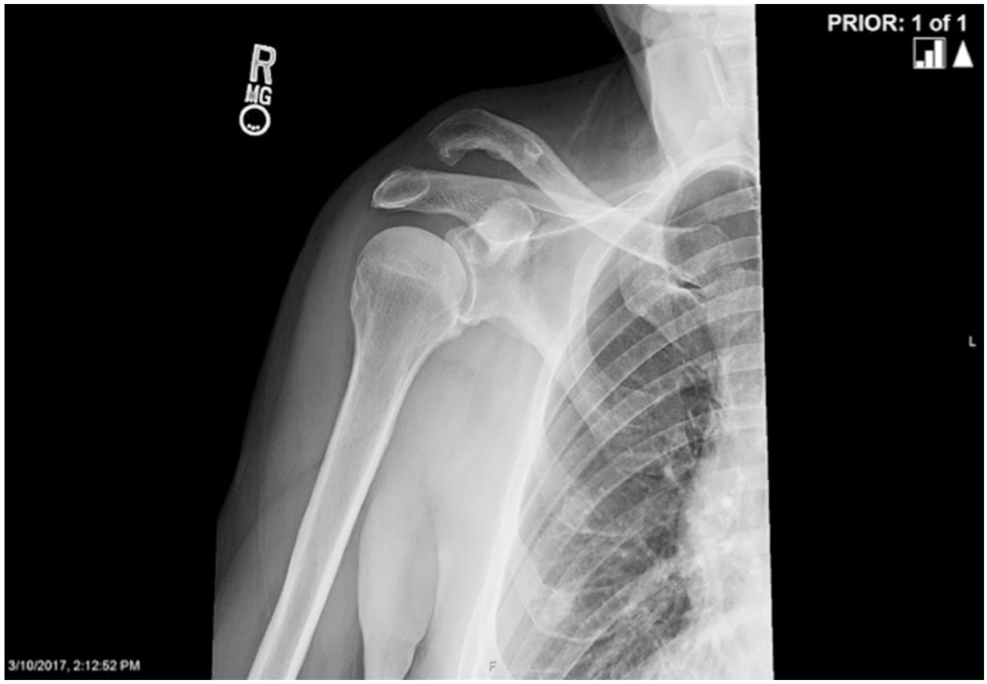

Fig. 36.2 A patient who presented 3 years after biking injury found to have a chronic grade V AC joint injury

Fig. 36.3 Postoperative Zanca view

Initial Management

- Grade I–II, III (nonoperative) [25]:
  - Analgesic and NSAIDs.
  - Cryotherapy.
  - A sling can be worn for comfort.

Indications for Orthopedic Referral

- Grade III: Consider operative management in athletes or manual/heavy laborers [18, 19, 25].
- Grade IV: open or closed reduction.
- Grade V–VI: Grade IV: open or closed reduction, fascial repair, ligament reconstruction.

Follow-Up Care (Nonoperative)

- Gentle range of motion within the first week
- Gradual progression to strengthening exercises for the shoulder and scapular stabilizers

Return to Sports

- Grade I, II, and III (nonoperative): The patient can return to sports once their pain has resolved and they are able to achieve full ROM and strength. Typically, this occurs within 2–4 weeks [18, 19].
- Grade III (operative management), VI–VI: Initial postoperative treatment consists of limited range of motion in a supervised physical therapy program with sling immobilization for 6 weeks. At 12 weeks, free range of motion and unlimited activities of daily living are permitted. Return to overhead and contact sports is not recommended prior to 6 months after surgery [18, 19].

Complications

- Fracture (acute)
- Pneumothorax (acute)
- Permanent deformity
- Stiffness
- Persistent instability
- Early osteoarthritis
- Distal clavicle osteolysis

Humeral Avulsion of the Glenohumeral Ligaments (HAGL)

Anatomy (Fig. 36.4)

- Inferior glenohumeral ligament:
  - The primary anterior stabilizer of the shoulder when the arm is at 90° of abduction and external rotation [20].
  - Failure of the IGHL has long been considered the essential lesion in anterior shoulder instability [20].
  - Three components: anterior band, posterior band, and interposed axillary pouch [20].
- Coracohumeral ligament:
  - Reinforces the rotator interval [20]
- Superior glenohumeral ligament:
  - Forms an anterior cover around the long head of the bicep tendon [20]
  - Part of the rotator interval
- Middle glenohumeral ligament

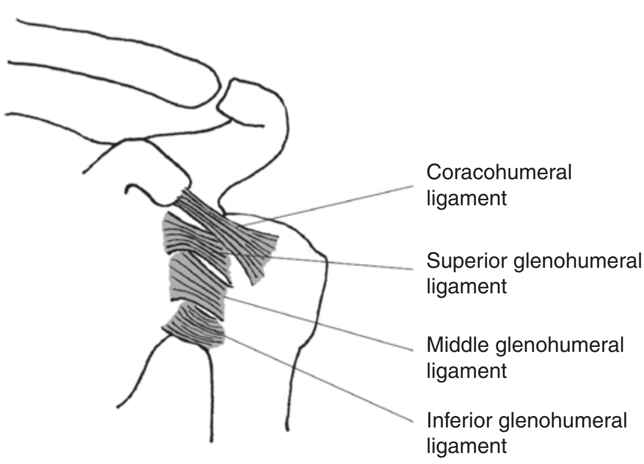

Fig. 36.4 Glenohumeral ligaments

Mechanism of Injury in Sports

- HAGL lesions are the result of acute traumatic glenohumeral subluxation or dislocation.
- There is no clearly proven injury pattern that is unique to HAGL lesions.
- Possible theories include [21–23]:
  - Hyperabduction and maximum external rotation
  - High energy trauma leading to shoulder dislocation
  - Repetitive microtrauma in overhead and throwing athletes
- Activities with the highest incidence of HAGL lesions: rugby, ice hockey, wrestling, motocross, skiing, and volleyball.

Epidemiology

- HAGL lesion is found in 1–9% of patients with glenohumeral instability [21]

Classification (Table 36.2)

Table 36.2 West point classification system [21–23]

HAGL lesion	Presence of bony avulsion	Presence of associated labral lesions	
Anterior HAGL	No	No	93% of reported cases
Anterior bony HAGL	Yes	No	
Floating anterior IGHL	Yes	Yes	Detachment at both the glenoid and humeral attachments
Posterior or reverse HAGL	No	No	7% of reported cases
Posterior bony HAGL	Yes	No	
Floating posterior IGHL	Yes	Yes	Detachment at both the glenoid and humeral

Clinical Presentation

- Patients typically present with vague complaints – weakness, pain, and decreased function [21, 22].
- Providers should have increased clinical suspicion in athletes with a history of shoulder stabilization surgery, especially those from certain high-risk sports, such as rugby and volleyball [21].

Diagnosis

- There is no specific finding that is pathognomonic for a HAGL lesion [21, 22].
- Provocative maneuvers include apprehension, relocation, load and shift, posterior stress. and posterior jerk tests.
- Routine radiographs rarely reveal any diagnostic evidence regarding capsular injury, but secondary findings such as a Hill–Sachs lesion or glenoid bone loss may be seen.
- MRI or MR arthrogram – the imaging modality of choice for the assessment of a suspected lesion [21]:
  – The characteristic U-shape created in the axillary pouch will appear J-shaped in the presence of a lesion to the IGHL because of extravasation of contrast or joint fluid across the torn capsule and ligament.

Initial Management

- Injury to the IGHL complex without humeral detachment cay be managed successfully nonoperatively with a course of physical therapy.
- Detached lesions can be managed initially with physical therapy, but most young athletes will continue to remain symptomatic necessitating surgical intervention

Indications for Orthopedic Referral

- HAGL lesions associated with instability are typically treated operatively [21, 22].
- The surgical approach is directed at a primary repair of the capsular disruption.
- Both open and arthroscopic approaches are utilized in treatment.

Return to Sports

- Typically considered with a patient who regains dynamic strength and range of motion.

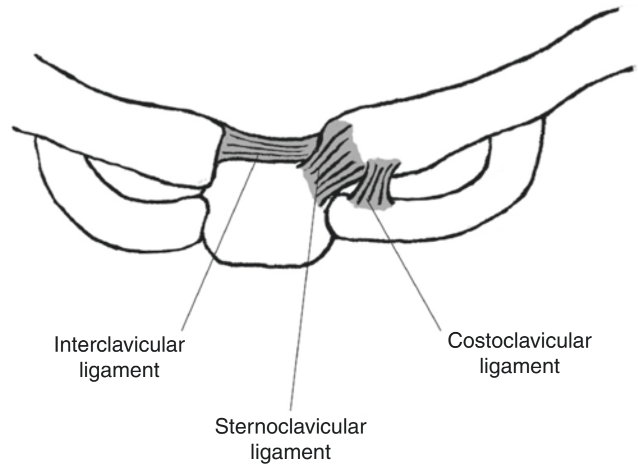

Fig. 36.5 Sternoclavicular ligaments

Sternoclavicular Ligaments

Anatomy

- The sternoclavicular joint is formed by the sternal end of the clavicle, the clavicular notch of the manubrium, and the cartilage of the first rib [26].
- The ligaments of the sternoclavicular joint are the anterior and posterior sternoclavicular ligaments, the costoclavicular ligament, the interclavicular ligament (Fig. 36.5) [26].
- Other stabilizing structures include the capsule:
  – The posterior capsule in the most important restraint for anterior and posterior translation of the sternoclavicular joint, and the anterior capsule helps restrain against anterior translation [26].

Mechanism of Injury in Sports

- Injuries typically occur by high-energy collisions involving a direct force to the clavicle or indirect force to the shoulder [24, 25].

Epidemiology

- Traumatic sternoclavicular joint injuries account for less than 3% of all traumatic joint injuries [27].
- Sprains of the ligament account for most injuries to the SC joint.

Classification

- Type I injury: mild sprain – ligamentous integrity is maintained [25]

- Type II injury: moderate sprain – partial ligamentous disruption [25]
- Type III injury: severe sprain – complete ligamentous disruption [25]

Clinical Presentation

- Type I injury: The patient typically presents with mild to moderate pain associated with movement of the upper extremity [25]:
  – The ligaments remain intact; therefore, instability is usually absent.
  – The SC joint may be tender to palpation and slightly swollen.
- Type II injury: At presentation, the joint may sublux when manually stressed but is not dislocatable [25].
  – Patients usually have more swelling and pain.
  – The SC joint is typically more tender to palpation.
- Type III injury: results in complete dislocation, either anterior or posterior, of the SC joint [25]:
  – Patients present with severe pain, exacerbated by movement of the upper extremity.
  – The ipsilateral shoulder may appear protracted.
  – Patients will often hold the affected arm across the chest in an adducted position and support it with the contralateral arm, and the head may be tilted toward the clavicle.

Diagnosis

- Imaging is important in evaluation if patients with sternoclavicular injuries.
- Radiographs can be difficult to interpret because of overlying shadows. A serendipity view (Fig. 36.6 beam tilted 40° from vertical and directed cephalad through the manubrium) may help to determine the position of the clavicle [26].
- A CT scan allows evaluation of both sternoclavicular joints, provides important information on the structures of the superior mediastinum, and helps to distinguish a physeal injury in pediatric patients.

Initial Management

- Type I and II injuries are typically treated nonoperatively.
- Short period (3–4 weeks) of immobilization in a sling oral analgesic and ice.
- Type II injuries with subluxation of the clavicle can be treated with shoulder retraction in a figure-of-eight sling [25, 26].
- Type III injuries, with complete SC joint dislocation, can be treated with closed reduction [25, 26]:
  – Closed reduction of a posteriorly displaced clavicle should be performed in an operating room under general anesthesia followed by 6–8 weeks of immobilization.

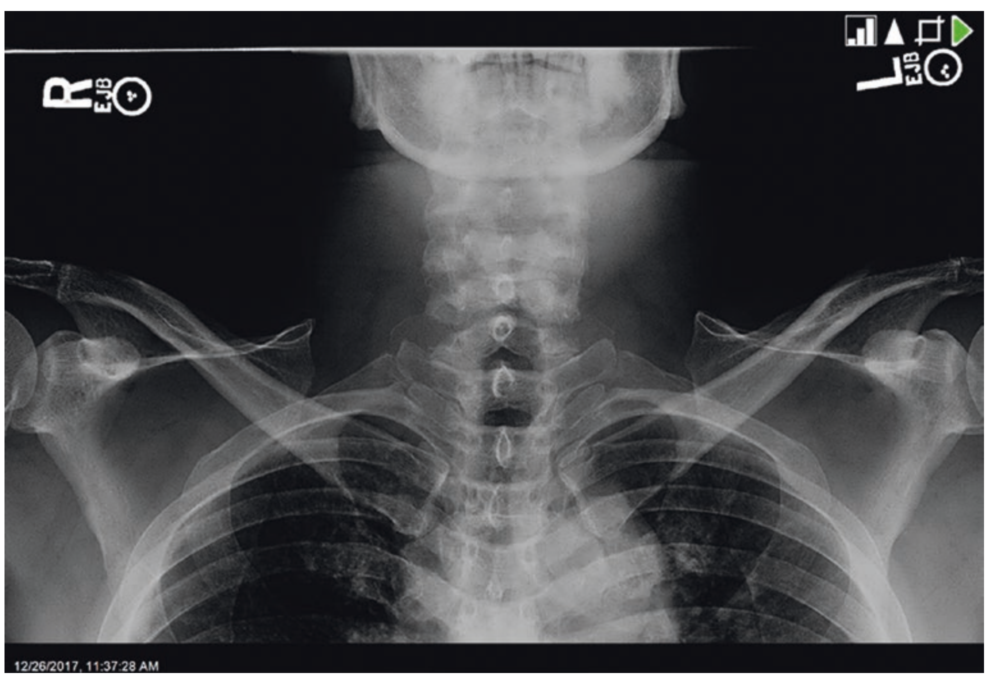

Fig. 36.6 Serendipity view

- If surgery is performed, it should be done with a cardiothoracic surgeon present [25, 26].
- Surgical treatment typically involves stabilization of the joint with pins and screws or soft tissue reconstruction of the acromioclavicular or coracoclavicular ligaments.

Indications for Orthopedic Referral

- Posterior dislocation
- Symptomatic SC joint instability

Follow-Up Care

- Gentle range of motion within the first week
- Gradual progression to strengthening exercises for the shoulder and scapular stabilizers

Return to Sports

- Nonoperative treatment: The patient can return to sports once their pain has resolved and they are able to achieve full ROM and strength [25–27].
- Operative treatment: A figure-of-eight clavicle splint or Valpeau dressing is used for 4–6 weeks. Patients should not elevate the arm more than 60° during this time, using the arm only for hygiene purposes. After 12 weeks, the patient may gradually increase the use of the arm, with return to sports no sooner than 6 months after operative fixation [27].

Fig. 36.7 Chronic anterior right SCJ dislocation in a 75-year-old man with no pain or tenderness

Complications

- Recurrent dislocation
- Pain
- Osteoarthritis
- Cosmetic deformity (Fig. 36.7)

Pediatric Considerations

- Sprain and physeal injury/fracture can be difficult to distinguish in the skeletally immature patient. Many dislocations in patients under the age of 25 are actually fractures through the physeal plate [27].
- Closed reduction can be attempted for anterior dislocations in pediatric patients but can be difficult to maintain. If a closed reduction fails, operative treatment is recommended [27].

Scapholunate Ligament

Anatomy

- The scapholunate ligament (SLL) is key in stability of the wrist (Fig. 36.8).
- It is an intra-articular C-shaped ligament composed of three different components: dorsal, volar, and proximal [12].
- Injury to the ligament is often missed, and isolated injury can lead to altered joint mechanics and subsequent degenerative changes.

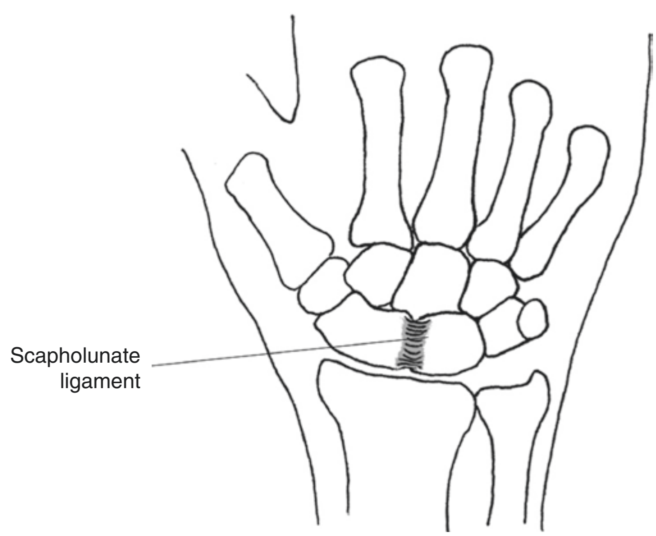

Fig. 36.8 Scapholunate ligament

Mechanism of Injury in Sports

- The most commonly cited mechanism for traumatic rupture is a fall onto the hypothenar eminence with the wrist in extension, ulnar deviation, and midcarpal supination [29].
- It is frequently associated with distal radius fractures, scaphoid fractures, and other ligamentous injuries.

Epidemiology

- It is estimated that 5% of all wrist sprains have an associated scapholunate ligament tear .

Classification (Table 36.3)

Clinical Presentation

- Swelling and pain over the dorsoradial aspect of the wrist
- Grip weakness
- Painful or decreased wrist motion
- Special testing:
  - Scaphoid shift test (Watson test) – may be used to detect laxity of the scaphoid ligaments [30].
  - The test is performed by the examiner passively moving the patient's hand from a position of ulnar deviation (Fig. 36.9a) to radial deviation (Fig. 36.9b), while the thumb of the clinician's other hand maintains firm pressure on the volar aspect of the scaphoid. Pain at the scaphoid with radial deviation signifies a positive test. The examiner can then release pressure on the scaphoid, and, if laxity is present, the subluxed scaphoid will move back into alignment with a palpable "clunk." Clinicians should compare injured to uninjured wrists.

Table 36.3 Classification of scapholunate ligament injuries

	Nonstress radiographs	Stress radiographs
Pre-dynamic	Normal	Normal
Dynamic	Normal	Pathologic widening present
Static	Pathologic widening, dorsiflexion intercalated segment instability (DISI pattern) present	

Diagnosis

- Radiographs.
  - Neutral PA and lateral views.
  - Stress views can be obtained but may require painful contracting and positioning. An example of a commonly used stress views is the PA or AP clenched fist-ulnar deviation (Fig. 36.10) [29, 30].
  - An interosseous distance greater than 3 mm between the scaphoid and lunate on plain radiographs suggests a ligamentous injury.
- Advanced imaging.
- MRI and MR arthrogram are the most commonly performed diagnostic tests [28, 31].
- MRA demonstrates improved sensitivity, specificity, and accuracy when evaluating for full-thickness tears of the SLL.
- Wrist arthroscopy is the gold standard for diagnosis.

Initial Management

- Nonoperative treatment can be considered for stable, partial SLL tears.

Indications for Orthopedic Referral

- Surgical treatment is the mainstay of SLL injury in the athlete.
  - Surgical indications vary based on the severity of instability.
  - Direct repair of an acute injury, typically within 3 weeks, is ideal for a successful outcome.

Follow-Up Care

- Immobilization for 4–6 weeks

Return to Sports

- Nonoperative:
  - With return of full ROM and strength
- Operative:
  - There is limited data on return to play.

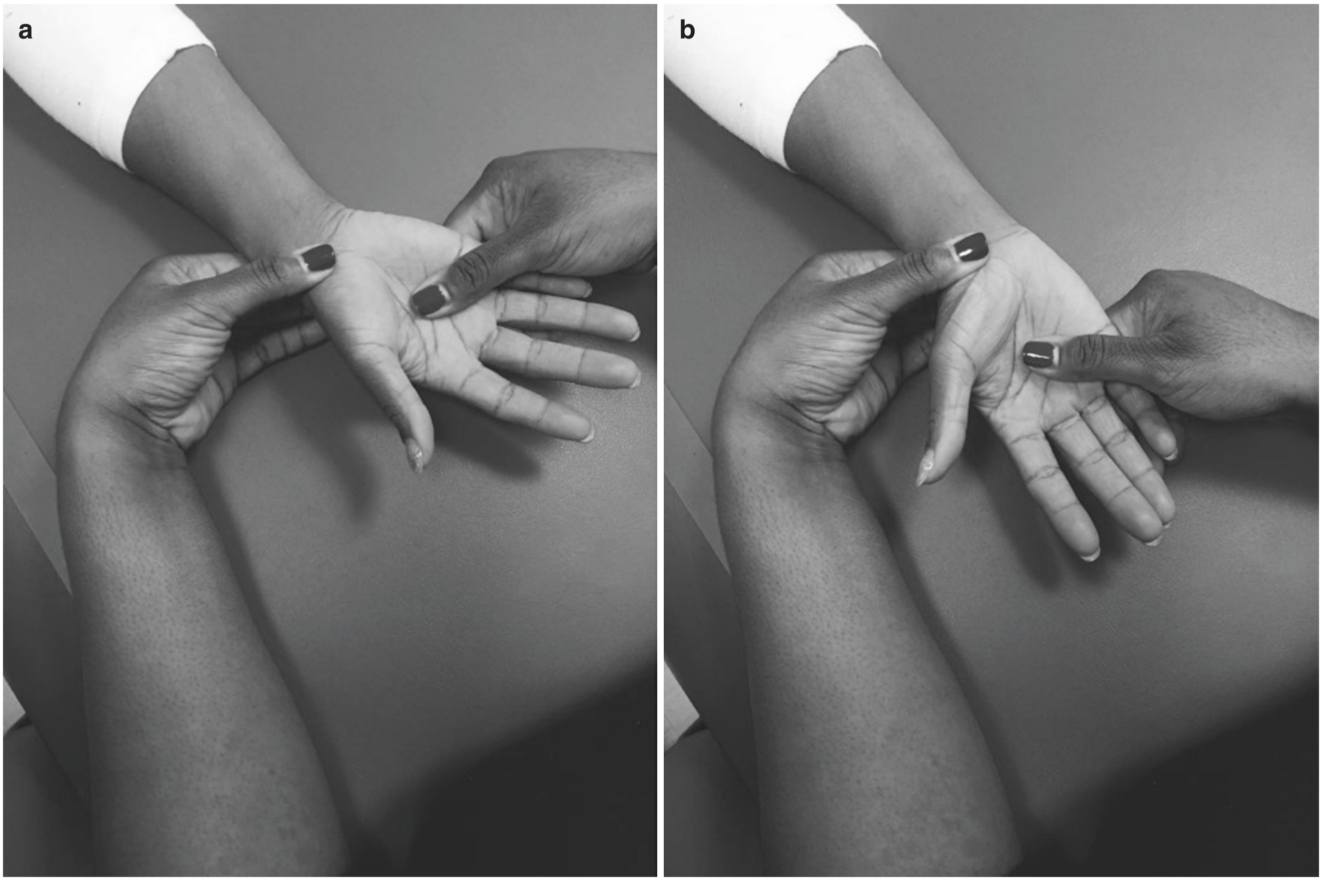

Fig. 36.9 Watson scaphoid shift test

– Literature shows that approximately 80% of professional athletes successfully return to sports within 4 months of undergoing the modified Brunelli procedure, with two-thirds returning to their preinjury level of competition [32, 33].

Complications

- Osteoarthritis – specifically SLAC (scapholunate advanced collapse) wrist
- Dorsal intercalated segment instability (DISI) [29]

Pediatric Considerations

- Scapholunate ligament injury is rare in adolescent and pediatric populations.
- The diagnosis can be complicated by normal development as the lunate is not radiographically recognized until the age of four and the scaphoid ossifies eccentrically giving a false impression of widening [32].
- Comparison with the contralateral wrist is unreliable because asymmetry in ossification can be seen [32].

Ulnar Collateral Ligament of the Elbow

Anatomy (Fig. 36.11)

- The ulnar collateral ligament comprises three ligamentous portions: anterior oblique bundle, posterior oblique bundle, and transverse ligament (Cooper ligament).
  – The anterior bundle is the primary stabilizer to valgus stress during the throwing motion.

Fig. 36.10 Stress view radiograph (ulnar deviation) demonstrates a widened scapholunate interval

Fig. 36.11 Ulnar collateral ligament

Mechanism of Injury in Sports

- Complete UCL tears typically occur due to repetitive valgus stress during throwing or other overhead sports and, less commonly, can occur with direct valgus force at the elbow.

Epidemiology

- Medial elbow injuries are the leading cause of missed game time in NCAA baseball players [34, 35, 38].
- As attention on the frequency of UCL reconstruction increases, it is estimated that 10% of active pitchers in MLB have undergone surgery, and there has been a 50% increase in reconstruction numbers in high school baseball players [38]

Classification

- Normal
- Partial tear
- Complete tear

Clinical Presentation

- Medial elbow pain that increases during the acceleration phase of throwing
- Tenderness to palpation distal to the medial epicondyle over the UCL
- Stress testing [36, 39]:
  - Valgus stress test – the patient's arm is stabilized, and a valgus force is applied at the distal forearm with full extension (Fig. 36.12a) and with 20–30° of flexion (Fig. 36.12b).
  - Moving valgus stress test – with the patient in the upright position, the shoulder is abducted to 90° and the elbow is maximally flexed. Vagus torque is applied to the elbow and maintained while the elbow is quickly extended to 30°. A positive test should produce pain from 120° of flexion to 70° of extension.

Diagnosis

- Radiographs – AP and lateral views, stress views [35, 36]:
  - Can help identify avulsion fractures and osseous remodeling.
  - Calcifications within the ligament may correlate with partial tearing.
  - Stress view – valgus force on the elbow that is flexed 20–30° with the forearm in full supination.
    - Widening of more than 0.5 mm suggests either a partial or complete UCL tear.

Fig. 36.12 Valgus stress test

- Gold standard – MR arthrogram [35, 36].
- Newer MRI techniques are more reliable and may obviate the need for MRA:
  – An MRI of the elbow performed in full extension places the anterior bundle on tension and can be used to distinguish between low-grade partial and high-grade partial, and full-thickness tears [35, 36].
- Dynamic ultrasound can be useful for identifying partial UCL tears and chronic ligament degeneration.

Initial Management

- A period of rest with cessation of throwing or strenuous elbow use

Indications for Orthopedic Referral

- UCL reconstruction is typically indicated for high-demand athletes with full-thickness UCL tears [35, 36, 41].
- The ideal group for early anatomic repair is young athletes with avulsion injuries at the UCL insertion [35, 36, 41].
- Surgical intervention can be considered for those patients that fail conservative treatment [36].

Follow-Up Care

- Nonoperative treatment is acceptable for low demand athletes and patients or those with partial UCL tears [35–39].
- Limited data is available on nonoperative treatment in the setting of UCL insufficiency:
  – Rattig et al.: rest/bracing for 8–12 weeks (prevent valgus force until pain free), followed by strengthening and throwing progression.
  – Wilk et al.: bracing to block valgus stress with a progressive flexion-extension range of motion program that is increased weekly until full ROM; advanced strengthening was initiated at 6 weeks.

- Any rehab protocol should aim to address breakdowns within the kinetic chain (focus of early ROM, glenohumeral and scapular strengthening, proprioceptive training, lower body, and core rehab).
- Postoperative rehab places emphasis on motion, strength, and neuromuscular control while gradually applying load to the healing tissue.
- An attempt to address biomechanical issues should be made as well.

Return to Sports

- Return to competition after nonoperative management – after pain-free completion of a rehab program focusing on ROM, strength, and kinetic chain deficits [14]
- Return to throwing after surgery – 9 to 12 months [40, 41]
- Return to competitive throwing after surgery – 1 to 2 years [40, 41]

Complications

- Re-rupture
- Ulnar neuropathy/neuropraxia
- Heterotopic ossification
- Medial epicondyle fracture

Pediatric Considerations

- Adolescent athletes may opt for nonoperative treatment to avoid surgery.

Lateral Collateral Ligament of the Elbow

Mechanism of Injury in Sports

- Most often caused by a traumatic event (i.e., dislocation, valgus stress with axial load) [42]

Clinical Presentation

- In an acute injury, the patient may complain of lateral elbow pain and mechanical symptoms (i.e., clicking, locking, or snapping).
- The examination of patients with nonacute injuries is typically normal.
  - Stress tests [42]:
    - Lateral pivot shift test (Fig. 36.13)
      - The patient is positioned in the supine positions on the examination table with the arm overhead, the shoulder in full external rotation, and the forearm in full supination. The lateral pivot shift maneuver is performed (similar to the pivot shift test of the knee) starting from the extended and supinated position of the elbow. In the extended and supinated position, the radial head is in the unstable, sublimated position. As the examiner brings the arm from extension to flexion, a valgus stress is placed on the elbow, and the forearm is allowed to be less supinated. This allows the forearm to pivot around the anterior bundle of the MUCL, and results in reduction of the elbow joint as the triceps becomes taught at around 40° of elbow flexion, often causing an audible or palpable clunk. In the awake patient, it is unusual to be able to elicit a positive lateral pivot shift due to guarding by the patient. Therefore, apprehension during the lateral pivot shift maneuver is often considered to be a positive test even without frank instability.
    - Chair push up test:
      - The patient pushes up from a chair with the hands on the arm rest. If there is apprehension or if dislocation occurs as the patient pushes the arm from flexion to extension, the test is positive.

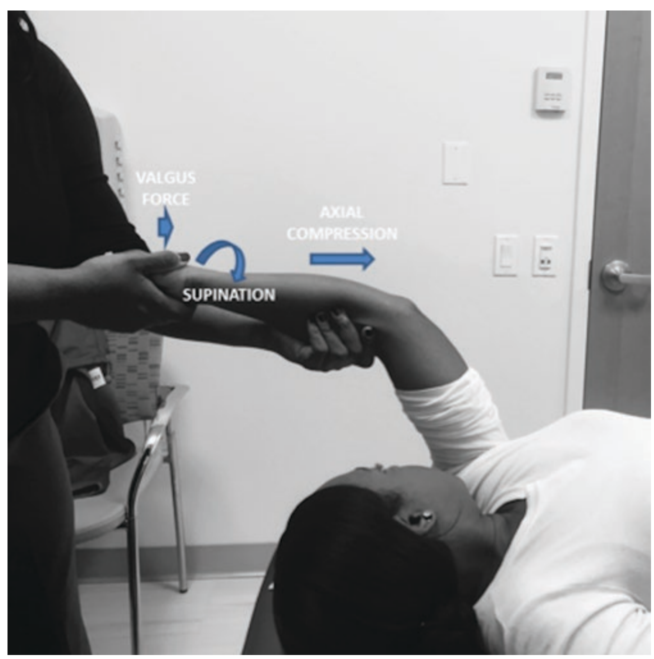

Fig. 36.13 Lateral pivot shift test

Diagnosis

- The diagnosis is typically made clinically.
- Plain radiographs of the elbow are typically obtained to evaluate for fracture, subluxation, or dislocation [42].
- Advanced imaging: MRI or MR arthrogram.

Initial Management

- Nonoperative treatment is often unsuccessful in chronic cases:
  - Physical therapy and NSAIDs can be utilized.
  - Elbow bracing can be implemented, but most patients do not tolerate it long term.

Indications for Orthopedic Referral

- The majority of patients with chronic posterolateral rotatory instability will require operative treatment [42].

Complications

- Recurrent subluxation
- Decreased elbow extension (typically seen with triceps fascia harvest) [42]

Pediatric Considerations

- Surgery is not indicated for skeletally immature patients.

Ulnar Collateral Ligament of the Thumb

Anatomy

- Ulnar collateral ligament injury is the most common injury to the thumb metacarpophalangeal joint [44].
- The ulnar collateral ligament (UCL) is a thick band composed of two distinct structures (proper and accessory UCL) resisting valgus stress at the thumb MCP joint (Fig. 36.14) [35].
  - The proper UCL gives max valgus stability at 30° of flexion.
  - The accessory UCL acts as a valgus restraint in full extension.

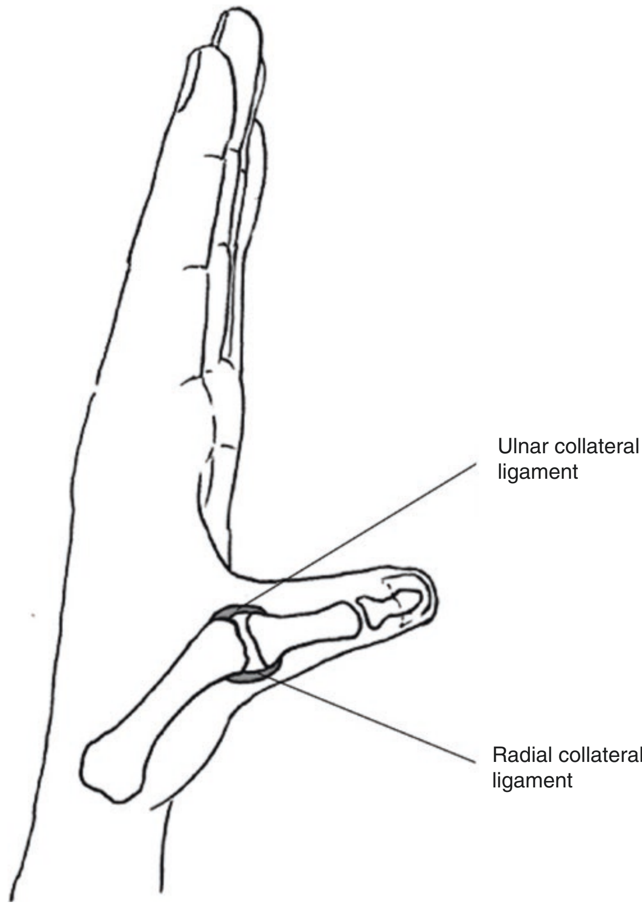

Fig. 36.14 Ulnar and radial collateral ligaments

Mechanism of Injury in Sports

- Ulnar collateral ligament injury was originally seen in skier's falling with their pole, known as skier's thumb [43, 44].
- Typically, valgus or radial-directed loads cause hyperabduction forces at the MCP joint leading to UCL injury [43, 44].
- Previously noted in high incidence among skiers:
  - Now, the injury is more commonly seen in falls onto an abducted thumb or when a ball or racquet hits the thumb.

Epidemiology

- Incidence is as high as 50 per 100,000 [43, 44)].
- UCL injuries at the thumb occur ten times more often than radial collateral ligament injuries at the thumb.

Classification

- Grade 1: pain, no instability
- Grade 2: partial tear with asymmetric laxity but firm endpoint appreciated
- Grade 3: complete tear with laxity and no appreciable endpoint

Clinical Presentation

- Ecchymosis and swelling are usually seen with acute injuries.
- Severe injuries may produce radial deviation and volar subluxation of the proximal phalanx on the metacarpal head.
- Tenderness along the ulnar side of the MCP joint.
- A palpable mass, a completely torn and retracted UCL, may be present at the ulnar side of the metacarpal neck. This is known as a Stener lesion [43].
- Stress testing: (Fig. 36.15)
  - Stabilize the metacarpal neck while producing a radial-directed force on the proximal phalanx [17, 43].
  - Positive testing:
    1. Laxity of 30°
    2. Increased laxity of >15° compared with the unaffected side
    3. Lack of endpoint

Diagnosis

- Diagnosis is primarily clinical.
- Radiographs: AP and lateral:
  - An avulsion fracture may be present, signifying a complete UCL rupture (Fig. 36.16).
- Advanced imaging: MRI and ultrasound:
  - US results vary widely, depending on the operator.
  - MRI remains more consistent.

Fig. 36.15 Ulnar collateral stress testing

Initial Management

- Grade 1 and 2 injuries are typically treated nonoperatively and typically do well without residual pain or disability [43, 44]:
  - These are typically initially treated with immobilization in a splint or cast for protection for 4–6 weeks.
  - Followed by mobilization and strengthening by 6–8 weeks.

Indications for Orthopedic Referral

- Grade 3 tears are typically treated with surgery [43].
- Symptomatic patients with grade 3 tears and associated osteoarthritis are best managed with a thumb MCP joint arthrodesis [12].

Fig. 36.16 PA radiograph shows avulsion fracture consistent with UCL ligament rupture, Stener lesion

Follow-Up Care

- After initial immobilization, a removable splint is applied and range of motion activities start, avoiding radial deviation or stress [43].
- Grip and pinch strengthening begins after attaining full range of motion, typically around 6 weeks [43].
- Postoperatively, the thumb is protected in a cast for 6 weeks, followed by initiation of physical therapy and range of motion exercises, avoiding radial stress [43].
- After cast immobilization, patients will transition to a removable splint and strengthening typically begins 8 weeks after surgery or when full range of motion is achieved [43].

Return to Sports

- Grade 1 and 2 tears are treated with at least 4 weeks of immobilization in a thumb spica cast.
- After cast removal, protection with a hand-based removable splint or dorsal radial thumb splint can be protective between weeks 4 and 8.
- Patients with more significant injuries and those in high-contact sports or sports with significant risk of reinjury should protect the joint for an additional 6 weeks.
- After operative repair, early return to sport is allowed if participation is possible without the use of the thumb.
- For others, unrestricted activity is allowed without a splint when the athlete is pain free and full range of motion and strength are restored, typically between 12 and 16 weeks.

Complications

- Persistent instability
- Neuropraxia to the dorsal sensory branches of the radial nerve
- Decreased pinch strength
- Decreased grip strength

Radial Collateral Ligament of the Thumb

Mechanism of Injury in Sports

- This type of injury is caused by an ulnar-directed force on the MCP joint of the thumb, typically a fall on the radial aspect of the thumb or by a player or ball producing an ulnarly directed stress on the proximal phalanx of the thumb [44].

Epidemiology

- 10–42% of collateral injuries of the thumb [44]

Classification

- Grade 1: incomplete tears with tenderness to palpation but no instability
- Grade 2: partial tears with asymmetric laxity but a firm endpoint
- Grade 3: complete tears with instability and no identifiable endpoint

Clinical Presentation

- Patients typically present with pain, swelling, and joint stiffness.
- Acute injuries may present with ecchymosis and swelling over the dorsal aspect of the MCP joint.
- Chronic injuries may have a dorsoradial prominence from the exposed radial metacarpal condyle [44]:
  - Examination [11, 13, 44]:
    - Tenderness over the radial aspect of the MCP and dorsal radial capsule.
    - Range of motion and stability in flexion and extension should be assessed.
    - Stability testing should be performed while stabilizing the metacarpal neck while providing an ulnar-directed force on the proximal phalanx in both full extension and 30° of flexion.
    - Anterior and posterior drawer testing should also be performed to assess for palmar subluxation.
    - Subluxation of >3 mm is typically found in a complete tear.
      - Other signs of complete tears include instability >30° or instability >15° when compared with the unaffected side.

Diagnosis

- Radiographs should be obtained to assess for avulsion injuries, fractures, and palmar MCP subluxation [44].
- Stress dynamic fluoroscopy may be helpful for evaluation of instability.
- If diagnosis is not obvious, ultrasound or MRI can be obtained:
  - Ultrasound can allow for static and dynamic testing.

Initial Management

- Grade 1 and 2 injuries are treated with immobilization in a thumb spica splint or cast for 4–6 weeks [44].
- Nonoperative treatment of grade 3 tears typically leads to symptomatic instability and degenerative changes of the MCP joint; therefore, operative repair is recommended for complete tears [44].

Indications for Orthopedic Referral

- Surgical indications are not clearly defined in the literature.
- Several small studies, including Dinowitz et al., have shown that operative treatment of complete tears may preserve grip strength and improve decreased range of motion typically seen with chronic RCL tears [44].

Follow-Up Care

- After initial immobilization, a removable splint is used for an additional 2 weeks while initiating higher demand activities [44, 45].

Return to Sports

- Complete tears treated surgically can typically return to unprotected play within 6–12 weeks after a period of immobilization [44].

Complications

- Decreased pinch strength
- Decreased grip strength
- Decreased ROM at the MCP joint

Collateral Ligaments of the Finger

Mechanism of Injury in Sports

- Forced ulnar or radial deviation can cause collateral ligament tears, typically known as a “jammed finger” [16].
- Low-speed forces can commonly produce a mid-substance tear.
- High-speed forces produce distal detachments and avulsion fractures.

Epidemiology

- In contact sports, 66% of injuries are metacarpal fractures and collateral ligament tears with volar and dorsal dislocations of the proximal interphalangeal (PIP) joint and distal interphalangeal (DIP) joint [14].

Injury Classification

- There are four distinct rupture patterns: [15]
  - Mid-substance tear
  - Proximal detachment
  - Distal detachment
  - Bony avulsion fracture

Clinical Presentation

- The injured typically present with tenderness at the involved collateral ligament.

Diagnosis

- The injury is evaluated by applying varus and valgus stress to the involved joint in 30° of flexion, while the MCP joint is flexed at 90° [14–16].
- Laxity should be compared with the contralateral finger.
- Plain radiographs are helpful in diagnosing subluxation or complete dislocation and can also help determine if an avulsion fracture is also present.
- AP, lateral, and oblique views are typically obtained.
- Varus and valgus stress views can aid in the diagnosis of suspected ligamentous injury.

Initial Management

- Ice and analgesia
- Buddy taping [16]

Indications for Orthopedic Referral

- Orthopedic referral is necessary for an unstable joint [14, 16].

Follow-Up Care

- Early protected motion is key [15].

Return to Sports

- Patient with collateral ligament injuries can continue to participate as tolerated, based on symptoms [16].

Complications

- Stiffness
- Decreased pinch strength

Pediatric Considerations

- Collateral ligament injuries in children can involve the growth plate, leaving a low threshold for referral to orthopedic or hand surgery [14–16].

Anterior Cruciate Ligament (ACL)

Mechanism of Injury in Sports

- Football, soccer, basketball, and lacrosse are the highest-risk sports.
- Contact injury results from direct impact on the knee by another player or object.
- Noncontact mechanisms for injury: [46]
  - Landing from jump
  - Plant and twist
  - Sudden deceleration
  - Hyperextension
- Synthetic playing surface has been shown to increase the risk of ACL injury in football, but not in soccer [47].
- Athletes with previous ACL injury have a significantly increased risk of a future second injury [48].

Epidemiology

- Annual incidence approximately 70 per 100,000 in the USA [49].
- Males > females.
- Peak in incidence is between 14 and 18 in females and 19 and 25 in males [49].
- Females have overall 1.5× higher rate of injury than males per exposure and 3x or higher rate of injury compared to males in similar sports [50–52].
- Noncontact mechanisms account for 40% or more of ACL injuries [51, 53].

Injury Classification

- Grade 1 – Sprain
- Grade 2 – Partial tear (less common)
- Grade 3 – Complete tear

Clinical Presentation

- Patients often feel a “pop” or sense a hyperextension of the knee.
- Acute swelling from hemarthrosis occurs within 2–4 h of injury.
- Physical examination maneuvers:
  - Lachman test – most accurate; positive likelihood ratio 10.2 [54].
  - Pivot shift test – highly specific at 98%, but low sensitivity at 24% due to difficultly performed test in acute setting from patient guarding [54].
  - Anterior drawer test – better for chronic injuries than acute injuries (92% sensitive and 91% specific versus 49% sensitive and 58% specific, respectively) [54].
- May have lack of full extension due to ACL stump catching in notch.
- Continued activity may provoke sensation of instability or “giving out.”

Diagnosis

- Three-view plain radiographs initially to rule out fracture or growth plate injury
  - Segond fracture at lateral tibial rim is highly specific for ACL tear [55] (Fig. 36.17).
  - Tibial plateau or tibial spine avulsion fracture can indicate ACL tear [56].
- The role of diagnostic ultrasound remains uncertain, although numerous techniques and approaches have been described [57].
- MRI is gold standard for confirmation of diagnosis:
  - Marrow contusion in lateral femoral condyle is commonly seen.

Initial Management

- Immediate cessation of sporting activity.
- Supportive care with ice, compression, elevation.
- Partial weight bearing as tolerated with crutches.
- Immobilization can cause motion loss and is not recommended [48].

Fig. 36.17 Standing AP knee radiograph showing a Segond avulsion fracture at the lateral tibial rim of the right knee (Image credit: Michael Cooley, MD)

- Consider early initiation of preoperative physical therapy to establish a normal gait pattern and range of motion, since preoperative ROM is an important predictor of post-operative ROM [58].

Indications for Orthopedic Referral

- Nonurgent referral to orthopedics for surgical reconstruction of complete ACL tears is recommended for young patients, elite athletes, and all patients with very active lifestyles [59].
- In general, surgery can be delayed several weeks until knee is no longer inflamed or painful, although optimal timing remains under debate [60, 61].
- Management of partial tears is dependent on the mechanical quality of the remnant ACL fibers assessed via examination under anesthesia and arthroscopic evaluation, with consideration given to the level and type of sports participation [62].
- Surgical reconstruction involves the use of tissue grafts to restore stability to the knee.
- Multiple techniques exist.
- Graft options:
  - Autograft – most commonly hamstring tendons or bone–patellar tendon–bone
  - Allograft – 4× risk of graft rupture in young patients; however, failure rates equalize as patient age reaches 40 years [63]
  - Synthetic grafts – no evidence to support use; historically low rates of success

Follow-Up Care

- Consider nonsurgical treatment for older or more sedentary individuals or athletes who are willing to modify their activity to exclude cutting and pivoting sports:
  - Variable proportion of conservative treatment group will go on to have surgery.
- A progressive rehabilitation program is indicated for both surgical and conservative treatment.

Return to Sports

- Typically 6 months after ACL reconstruction.
- Some advocate delayed return until at least 9 months in young female athletes to prevent second injury [64, 65].
- No standard set of criteria for return to sports currently exists.
- Suggested criteria for return to sports [66, 67]:

– Absence of pain
– Absence of subjective instability
– Absence of effusion
– Full range of motion
– KT-1000 side-to-side difference <3 mm
– Negative pivot-shift test
– Hamstring and quadriceps strength at 85–100% of the noninjured leg
– Functional testing battery >85% compared with noninjured leg

- Multiple criteria should be used to make an appropriate, individualized decision [68].
- A systematic review showed that overall, 82% of patients return to sport; however only 63% returned to their preinjury sport, and 44% returned at competitive levels [69].
- No evidence that use of ACL brace decreases the rate of reinjury.

Complications

- Concomitant injuries to medial collateral ligament, medial meniscus, and chondral surface are common.
- Prevalence of knee osteoarthritis after ACL injury is increased, regardless of treatment choice (conservative 31–48% versus reconstruction 29–51%) [68].

Pediatric Considerations

- Distal femoral physis remains open until age 14–16 in females and 16–18 in males.
- Concerns for iatrogenic growth disturbance from disruption of physis complicates care.
- Compliance with activity modifications is more difficult in younger age groups.
- Successfully techniques for physeal sparing, transphyseal, and all-physeal surgical reconstruction have been described [70–72].
- Older adolescents (girls older than 14, boys older than 16) at Tanner stage 5 and closed or near-closed physes can be treated as adults.

Posterior Cruciate Ligament (PCL)

Mechanism of Injury in Sports

- Soccer is the most common sport [73], along with football.
- Common mechanisms of injury:
  – Posteriorly directed force to proximal tibia while knee is flexed (dashboard injury).
  – Fall onto flexed knee with foot plantarflexed.
  – Sudden knee hyperflexion or hyperextension.

Epidemiology

- About 1–4% of sports-related knee injuries [73, 74]
- Rarely an isolated injury
- Most often injured in association with ACL, medial collateral ligament (MCL), or posterolateral corner (PLC)

Injury Classification

- Grade 1 – Sprain
- Grade 2 – Partial tear
- Grade 3 – Complete tear

Clinical Presentation

- Patients often have vague, nonspecific complaints of knee discomfort without associated "pop" or instability.
- Pain with kneeling.
- Difficulty with knee flexion, such as squatting or walking on stairs.
- Physical examination maneuvers:
  – Posterior drawer test – most sensitive (90%) and specific (99%) [75]
  – Posterior sag sign (Godfrey test)
  – Quadriceps active test (Fig. 36.18)
- Positive valgus stress test suggests concomitant MCL injury [76, 77].
- Positive reverse pivot shift and dial tests suggests concomitant PLC injury [76–78].
- Varus thrust gait also indicates combined PLC injury [76].

Diagnosis

- Three-view plain radiographs initially to rule out associated fractures:
  – Single-leg kneeling stress views effective for identifying posterior knee instability [76]
  – Additional stress views with applied varus stress useful for identifying concomitant PLC injury [79]
- The role of diagnostic ultrasound remains limited, with few studies examining its utility for assessing PCL injuries [80–82].
- MRI is gold standard for acute injury.

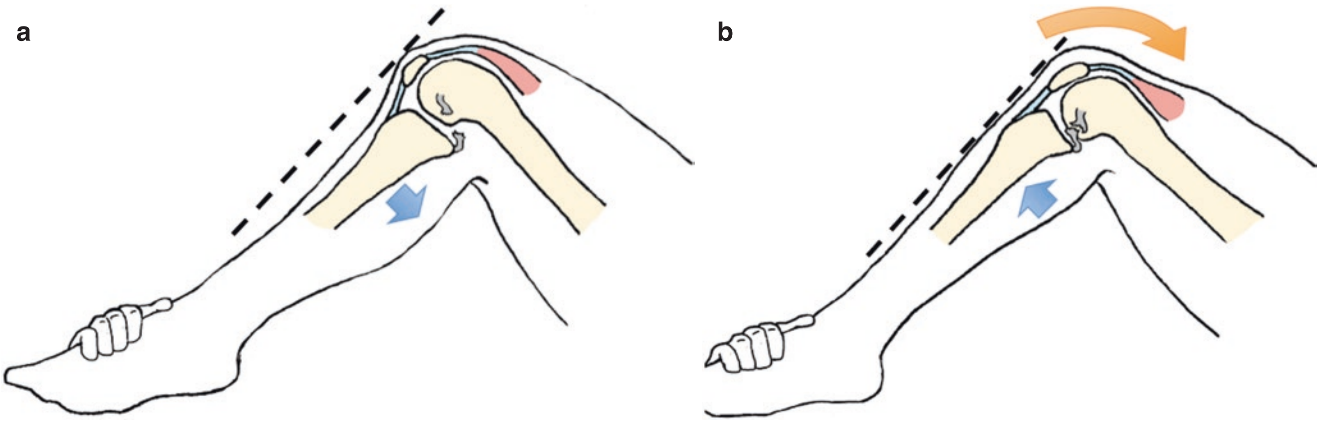

Fig. 36.18 Quadriceps active test. Position the patient supine, with the injured knee flexed at 90°. The examiner stabilizes the foot against the examination Table. (**a**) The tibia sags posteriorly at test due to a deficient PCL. (**b**) Active contraction of the quadriceps muscle by the patient dynamically reduces the tibia back into anatomical position via the extensor mechanism (quadriceps muscle–quadriceps tendon–patella–patellar tendon–tibial tubercle)

Initial Management

- Supportive care with ice, compression, elevation.
- Partial weight bearing as tolerated.
- Grade 1–2 injuries: Initiate early range of motion in prone position, and refer for quadriceps strengthening physical therapy program [83].
- Grade 3 injuries: Immobilize in full extension for 2–4 weeks to protect posterior structures from posterior translation of tibia [84].

Indications for Orthopedic Referral

- Refer to orthopedics within 1–2 weeks for surgical treatment of:
  - Grade 3 PCL injuries with instability
  - Isolated grade 3 injuries
  - Distal bony avulsion with greater than grade 1 laxity
- Delayed reconstruction of unstable injuries past 2–3 weeks increases the risk of arthrofibrosis [85].
- Injuries with bony avulsion should be repaired in acute phase, within 2 weeks [86–88].
- Early operative treatment can also be considered for grade 2 injuries in high-demand athletes, although 2% of elite college football players compete with chronic PCL-deficient knees [89].
- Nonurgent referral for surgical treatment in cases of failed conservative management with persistent instability.

Follow-Up Care

- Nonsurgical management recommended for isolated grade 1 or 2 PCL injuries and for grade 3 PCL injuries with mild symptoms or low activity demands.
- A progressive rehabilitation protocol is indicated for both surgical and conservative treatment.
- Treatment of chronic or delayed presentation PCL injuries focuses on quadriceps strengthening and activity modification.

Return to Sports

- Lack of sufficient high-quality evidence for a clear return to sports protocol.
- Typically 6–12 months for surgical or conservative treatment, after 85–90% of quadriceps strength is regained [77, 83].
- Elite athletes may tolerate accelerated return to sports after 6–8 weeks of nonsurgical management, with risk of increased residual joint laxity [90].

Complications

- Progressive medial compartment and patellofemoral osteoarthritis
- Residual laxity, even with surgical management

Pediatric Considerations

- Less concern for iatrogenic physeal injury than for ACL injuries, due to lower overall incidence of PCL injuries and good nonoperative outcomes
- Same guidelines for nonoperative versus surgical treatments as adults [91]

Posterolateral Corner (PLC)

Mechanism of Injury in Sports

- Contact sports such as football [92], wrestling [93], and skiing [94]
- Common mechanisms of injury:
  - Direct blow to anteromedial knee in extension
  - Hyperextension with external rotation
  - Noncontact varus stress

Epidemiology

- Exact incidence is unknown.
- Estimated to account for 16% of all ligamentous knee injuries [95].
- Typically occurs with ACL or PCL injury; only 28% occur as isolated injuries [96].

Injury Classification

- Hughston grading scale [97]:
  - Grade 1 – 0 to5 mm varus instability
  - Grade 2 – 5 to 10 mm varus instability
  - Grade 3 – >10 mm varus instability
- Fanelli grading scale [98]:
  - Type A – mainly rotational instability
  - Type B – rotational instability with mild varus stress gapping
  - Type C – marked external rotation and varus instability

Clinical Presentation

- Patients usually recall a specific trauma to the knee.
- Pain and instability near extension.
- Varus thrust gait may be seen.
- Paresthesias at first webspace or dorsum of foot and/or foot drop if concurrent common peroneal nerve injury exists.
- Physical examination maneuvers:
  - Varus stress test
  - Dial test
  - Reverse pivot shift
  - External rotation recurvatum test – less sensitive
- Varus stress at 0° and 30° (to isolate LCL from the stabilizing effects of the cruciate ligaments at full extension):
  - Grade 1 – no laxity
  - Grade 2 – varus laxity 5–10 mm, but with good endpoint
  - Grade 3 – marked varus instability >10 mm
- Positive varus laxity at 30° only indicates isolated LCL injury with potential involvement of PLC structures.
- Positive varus laxity at 0° and 30° indicates combined LCL, PLC, and PCL injury.
- Dial test at 30° and 90° of knee flexion:
  - >10° of external rotation compared with uninjured side suggests PLC injury.
- Positive dial test at 30° only indicates isolated PLC injury.
- Positive dial test at 30° and 90° indicates combined PLC/PCL injury.
- Consider performing ankle–brachial index (ABI) to screen for vascular injury in setting of knee dislocation.

Diagnosis

- Three-view plain radiographs initially to assess for fracture.
- Varus stress views should be obtained to assess for lateral gapping [79]).
- MRI is helpful to assess for grade and location of injuries.
- Ultrasound may be an emerging modality to evaluate PLC injuries, but evaluation of the PLC is technically challenging, and widespread utility is limited by operator skill [99–101].
- CT or MR angiography should be considered to assess for vascular injury in setting of knee dislocation if hard signs present or if screening ABI <0.9 [102, 103].

Initial Management

- Paucity of evidence on acute initial management of PLC injuries.
- Acute stabilization depends on the degree of laxity and any concomitant injuries:
  - Immobilize in extension if significant rotational or posterior instability.
  - Avoid immobilization if concurrent ACL injury is suspected.
- Assess neurovascular status to screen for nerve or vascular injury.

- Partial weight bearing as tolerated.
- Supportive care with ice, compression, elevation, and NSAIDs.

Indications for Orthopedic Referral

- Refer to orthopedics in 1–2 weeks for surgical treatment of:
  – Isolated grade 3 injuries
  – Combined injuries with ACL or PCL
  – Displaced or unstable avulsion fractures
- Acute surgical intervention within 3 weeks is recommended [104, 105].
- Nonurgent referral for surgical treatment in cases of failed conservative management with persistent instability.

Follow-Up Care

- Nonoperative treatment recommended for isolated grade 1 and 2 injuries
- One study describes immobilization for up to 4 weeks, followed by progressive rehabilitation protocol [106].
- Alternatively, protected weight bearing in a hinged knee brace with or without extension locking for 2–4 weeks followed by progressive rehabilitation protocol can be used [107].

Return to Sports

- No clear return to sports protocol.
- Generally, when normal strength, stability, and ROM have been regained.
- May be as soon as 9 weeks, or up to 3–4 months, for grade 1–2 injuries treated conservatively [103, 106].
- 6–9 months for grade 3 injuries after surgery, based on concurrent ligament surgery [108].
- Consider the use of medial unloader brace to decrease varus stress upon return to sport [103].

Complications

- Peroneal nerve injury occurs in 26% of PLC injuries [109].
- Missed diagnosis can lead to early failed ACL or PCL reconstruction due to increased loading forces to graft.
- Accelerated osteoarthritis.

Pediatric Considerations

- Concomitant intra-articular injuries less common than in adults.
- Unstable PLC injuries can be treated surgically with good outcomes [94].
- Associated distal femoral avulsion fractures can involve the physis and lead to growth disturbance with valgus angulation [94]
  – Close monitoring for physeal arrest until physis closes is recommended for both surgical and nonoperative management.

Medial Collateral Ligament (MCL) of the Knee

Mechanism of Injury in Sports

- Common sports include football, soccer, rugby, ice hockey, skiing, and wrestling [74, 110, 111].
- Typical mechanisms for injury:
  – Direct contact with a valgus force to knee
  – Cutting maneuver with foot planted
- Overuse injuries in swimmers have been reported [112].

Epidemiology

- Most commonly injured knee structure in athletes [74].
- Annual incidence approximately 700 per 100,000 in the USA [111].
- Males 2.6× higher rate than females per exposure.
- Approximately 70% of injuries are from direct contact [113].

Injury Classification

- Grade 1 – Sprain
- Grade 2 – Partial tear
- Grade 3 – Complete tear

Clinical Presentation

- Most patients hear a “pop” and can localize pain to medial knee.
- May have sensation of instability.
- Point tenderness along course of MCL and medial joint line.

- Ecchymosis, deformity, or effusion suggests greater extent of injury such as concomitant ligament or meniscal injury, physeal injury, or fracture.
- Valgus stress at 0° and 30° (to isolate MCL from the stabilizing effects of the cruciate ligaments at full extension):
  - Grade 1 – no laxity
  - Grade 2 – valgus laxity 5–10 mm, but with good endpoint
  - Grade 3 – significant valgus instability >10 mm
- Positive valgus laxity at 30° only suggests isolated MCL injury.
- Positive valgus laxity at 0° and 30° suggests concomitant ACL or joint capsule injury.
- Positive Lachman test suggests concomitant ACL injury.
- Positive McMurray test suggests concomitant meniscus injury.

Diagnosis

- Three-view plain radiographs initially to rule out fracture, dislocation, or physeal injury:
  - Valgus stress views can help show medial gapping [114].
  - Pellegrini–Stieda lesion (calcification of MCL) is seen in chronic injury (Fig. 36.19).
- Ultrasound has been shown to accurately diagnose MCL injuries, but widespread utility is limited by operator skill [115–118].
- MRI is gold standard:
  - Useful to assess grade and location of injury – usually proximal
  - Helps identify or rule out other injuries
  - Can show Stener's lesion of the knee – interposition of the pes anserinus tendons between torn MCL and its tibial attachment, which can impair healing [119] (Fig. 36.20)

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs
- Partial weight bearing as tolerated
- Long leg brace to prevent further valgus force for grade 2 and 3 injuries

Indications for Orthopedic Referral

- Refer to orthopedics in 1–2 weeks for surgical treatment of:
  - MCL tears combined with other knee injuries
  - Pes anserinus interposition
  - Tibial attachment disruption or bony avulsion [120]
- Combined MCL/ACL injuries may be treated with delayed ACL reconstruction 4–6 weeks after injury and nonoperative MCL care.
- Nonurgent referral for surgical treatment in cases of failed conservative management with persistent instability.

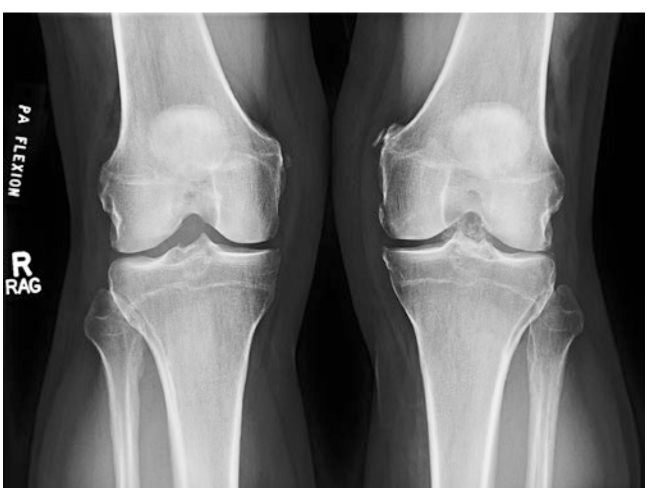

Fig. 36.19 Pellegrini-Stieda lesion (calcification of MCL) is seen in chronic injury

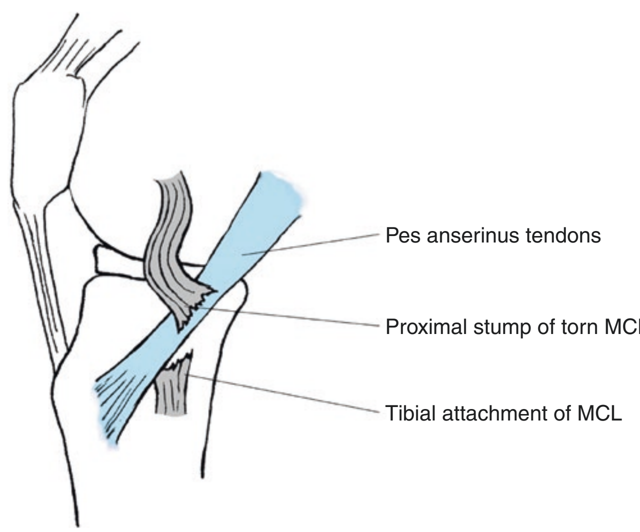

Fig. 36.20 Stener's lesion of the knee

Follow-Up Care

- Nonoperative treatment is standard of care for grade 1 and 2 injuries [110, 121], but is more controversial in grade 3 injuries.
- Initiate a progressive rehabilitation protocol starting with early ROM on stationary bike.

Return to Sports

- Lack of sufficient high-quality evidence for a clear return to sports protocol [122].
- Criteria for return [110, 122]:
  - Full ROM without pain
  - About 85–90% of quadriceps and hamstring strength
  - No instability on the examination
- Potential timelines for return to sports:
  - 1–2 weeks after grade 1 injury
  - 3–4 weeks after grade 2 injury
  - 5–7 weeks after grade 3 injury (nonoperative treatment)
  - 6–9 months after surgical reconstruction
- Return to sports decisions should be individualized; one study showed that despite return to sports at 5 weeks following successful nonoperative treatment of grade 3 injuries, many athletes in the cohort required 8–16 weeks to feel that their knee was back to full function [123].
- Although controversial [124], hinged functional MCL brace is generally recommended for return to sport [110].

Complications

- Chronic valgus instability can lead to degenerative osteoarthritis [124].

Pediatric Considerations

- MCL injuries have been reported with epiphyseal knee injuries.
- Salter–Harris type III fracture through tibial physis; requires fixation of fracture fragment and primary repair of MCL [124].
  - Close postoperative monitoring for physeal arrest and genu valgum deformity
- Combined ACL/MCL injuries can be treated with primary ACL transphyseal reconstruction techniques and MCL bracing with good outcomes [125].

Lateral (Fibular) Collateral Ligament (LCL) of the Knee

Mechanism of Injury in Sports

- Wrestling most common sport, along with football and soccer [126]
- Common mechanisms of injury:
  - Direct varus stress
  - Twisting injury
  - Hyperextension

Epidemiology

- Exact incidence is unknown.
- Estimated to account for 7% of all knee injuries in high school athletes [74].
- Rarely occurs in isolation; injury often involves other structures in the posterolateral corner (PLC) or cruciate ligaments.

Injury Classification

- Grade 1 – Sprain
- Grade 2 – Partial tear
- Grade 3 – Complete tear

Clinical Presentation

- Acute lateral knee pain with associated “pop”.
- Instability or difficulty weight bearing with higher-grade injuries.
- Significant swelling or effusion suggests greater extent of injury such as concomitant cruciate ligament injury.
- Point tenderness along length of LCL.
- Palpable defect may be present with higher grade injuries, with knee in “figure-of-4” position.
- Varus stress at 0° and 30° (to isolate LCL from the stabilizing effects of the cruciate ligaments at full extension):
  - Grade 1 – no laxity
  - Grade 2 – varus laxity 5–10 mm, but with good endpoint
  - Grade 3 – marked varus instability >10 mm
- Positive varus laxity at 30° only indicates isolated LCL injury.
- Positive varus laxity at 0° and 30° indicates combined LCL, PLC, and PCL injury.
- Positive Lachman test suggests concurrent ACL injury.

- Positive posterior drawer test suggests concurrent PCL injury.
- Positive dial or external rotation recurvatum tests suggests concurrent PLC injury.

Diagnosis

- Plain radiographs, including weight-bearing 45° flexion AP view.
- Varus stress views should be obtained to assess for lateral gapping [79].
- MRI is helpful to assess for grade and location of injuries.
- Ultrasound appears to have limited utility for the evaluation of LCL injuries, and utility is limited by operator skill [100, 116].

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs.
- Partial weight bearing as tolerated.
- Hinged brace limiting flexion to 45–60°.
- Initial limited immobilization <1 week may be considered for grade 2 or 3 injuries.

Indications for Orthopedic Referral

- Refer to orthopedics in 1–2 weeks for surgical treatment of:
  – Grade 3 injuries
  – LCL tears combined with other knee injuries
  – Displaced avulsion fractures
- Optimal timing for surgery on isolated grade 3 LCL injuries is not well defined; however, early referral is suggested to accommodate surgery within 3 weeks as recommended for combined injuries.

Follow-Up Care

- Nonoperative treatment with locking brace permitting gradual return to full flexion over 4–6 weeks and progressive rehabilitation protocol is standard of care for grade 1 and 2 injuries [105].

Return to Sports

- Potential timelines for return to sports [126–128]:
  – About 1–2 weeks after grade 1 injury
  – About 3–4 weeks after grade 2 injury
  – About 4–5 weeks after grade 3 injury treated nonoperatively
  – About 6 months after grade 3 injury with isolated LCL reconstruction
  – About 9 months after grade 3 injury with combined ligament reconstruction
- Use of functional knee brace is recommended for up to 1 year after return to sports [128].

Complications

- Persistent instability can result in medial compartment meniscal injury or osteoarthritis.
- Untreated LCL instability can lead to early ACL or PCL graft failure.

Pediatric Considerations

- Associated distal femoral avulsion fractures can involve the physis and lead to growth disturbance with valgus angulation [94]:
  – Close monitoring for physeal arrest until physis closes is recommended for both surgical and nonoperative management.

Syndesmosis

Mechanism of Injury in Sports

- Collision sports such as football, rugby, basketball, soccer, lacrosse.
- Sports that immobilize the ankle in a boot such as hockey, slalom skiing.
- Mechanism of injury is hyperdorsiflexion and external rotation of the ankle.
- Uneven terrain and synthetic playing surfaces have been shown to increase the risk of syndesmosis injury [129, 130].

Epidemiology

- Annual incidence of 2.09 per 100,000 [131]
- Most prevalent between ages 18 and 34 years
- Accounts for up to 20% of ankle sprains in athletes [132]
- Frequently associated with deltoid ligament injury

Injury Classifications

- Also termed "high ankle sprain."
- Isolated: syndesmotic injury without ankle fracture.
- Nonisolated: syndesmotic injury with associated fracture.
- Multiple classification systems based on ligamentous involvement, joint stability, and radiologic findings [133–137].
- West Point Ankle Grading System is one of the most widely used [138]:
  – Grade 1 – stable joint with mild tenderness and normal radiographs
  – Grade 2 – partial tear with positive external rotation and squeeze test and normal radiographs
  – Grade 3 – complete tear with frank instability
- European Society of Sports Traumatology, Knee Surgery, Arthroscopy – the Foot & Foot Associates (ESSKA–AFAS) recommends classifying acute isolated syndesmosis injuries as stable or unstable to facilitate treatment decisions [139]:
  – Stable – deltoid ligament intact
  – Unstable – latent or frank diastasis present

Clinical Presentation

- Pain at anterior ankle that is worse with weight bearing or pushing off.
- Less soft tissue swelling than seen in lateral ankle sprains.
- Point tenderness at anterior ankle joint line and anterior syndesmosis.
- Physical examination maneuvers (Fig. 36.21):
  – Squeeze test – highest specificity 88–94% [140, 141]
  – Cotton test
  – Dorsiflexion–external rotation test – 71% sensitivity [141], 85% specificity [140]
  – Fibular translation test
- Bony tenderness at malleoli or proximal fibula is suggestive of concomitant fracture.

Diagnosis

- Three-view plain radiographs of the ankle initially, including standing AP and mortise views, to assess for fracture and ankle diastasis [142] (Fig. 36.22):
  – Additional stress views useful to diagnose latent diastasis
- MRI has higher sensitivity for ligament injury, but cannot provide dynamic assessment.
- Dynamic ultrasound under stress testing is highly sensitive (66–100%) and specific (91–100%) for diagnosis of syndesmotic injuries, but is operator dependent [143–145].
- Bilateral standing CT is useful for showing dynamic instability and is preferred by some surgeons [146].

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs
- Immobilize with splint or walking boot
- Non-weight bearing with crutches

Indications for Orthopedic Referral

- Refer all unstable syndesmotic injuries, with or without fractures, within 1–2 weeks for early surgical management [147, 148].
- Nonurgent referral for surgical treatment in cases of failed conservative management with persistent instability or pain, as these may represent incorrectly classified unstable injuries.

Follow-Up Care

- Stable injuries are treated nonoperatively with non-weight bearing and immobilization in splint or walking boot for 3 weeks, followed by weight bearing in walking boot for 3 weeks with initiation of proprioceptive rehabilitation exercises [147].
- Elite athletes may tolerate a more accelerated progression through immobilization and weight bearing over 2 weeks [148].
- Lace-up ankle brace can be used for symptom reduction as needed after weaning [149] out of walking boot [150].
- Use of platelet-rich plasma (PRP) remains controversial due to limited high-quality evidence, although two small series have shown that PRP leads to significantly faster return to sports by 20 days [151, 152].

Return to Sports

- Typically after the was patient able to single-leg hop for 30 s without pain [153]:
  – As early as 6–8 weeks or up to >14 weeks for stable injuries [153, 154]

Fig. 36.21 Physical examination maneuvers for syndesmosis injury. (**a**) Syndesmosis squeeze test: the patient is seated with the knee flexed at 90° over the edge of the examination table. The examiner applies lateral and medial pressure along the mid-calf. Pain at the anterior ankle is a positive test. (**b**) Cotton test: the patient is supine with the leg extended. The examiner stabilizes the distal tibia with one hand and grasps the heel with the other hand and then attempts to translate the foot medially. Medial laxity is a positive test. (**c**) Dorsiflexion-external rotation test: the patient is seated with the knee flexed at 90° over the edge of the examination table. The examiner stabilizes the distal tibia with one hand and grasps the forefoot with the other hand and then dorsiflexes and externally rotates the foot. Pain at the anterior ankle is a positive test. (**d**) Fibular translation test: the patient is supine with the leg extended and internally rotated. The examiner stabilizes the distal tibia with one hand and grasps the distal fibula above the lateral malleolus with the other hand and then attempts to translate the fibula anteriorly and posteriorly. Pain and laxity of the fibula is a positive test

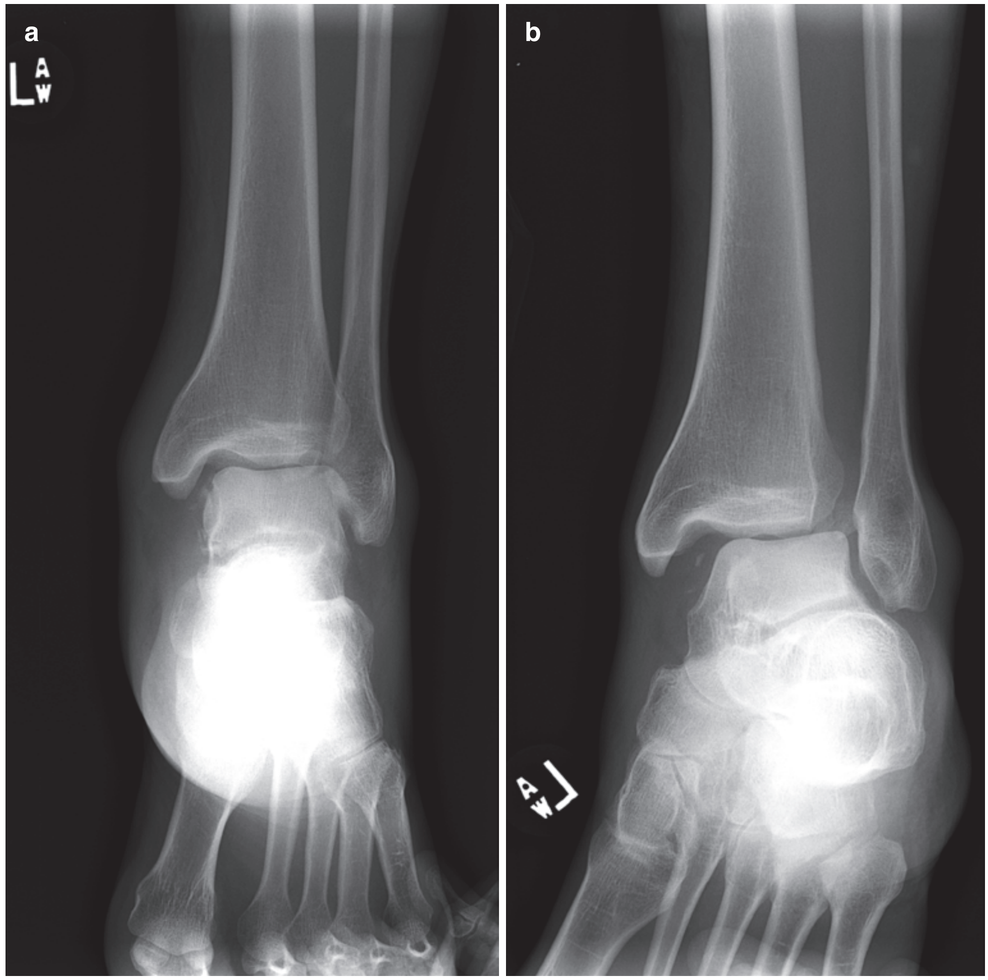

Fig. 36.22 Standing (**a**) AP and (**b**) mortise views of a left ankle, showing diastasis at the medial ankle along with an associated avulsion fracture

- As early as 8–10 weeks or up to 6 months after surgical fixation for unstable injuries, depending on procedure [146, 149, 153]

- Treatment goals and guidelines for stable and unstable injuries same as the adults.

Complications

- Substantial time missed from sports.
- Delayed diagnosis or insufficient treatment may lead to long-term dysfunction of ankle mobility.
- Postsurgical complications include malreduction with persistent instability, screw failure, osteolysis, heterotopic ossification, skin irritation, and implant-related pain [155].

Pediatric Considerations

- Isolated syndesmosis injuries rarely described in skeletally immature patients.
- Open distal tibial physis may be protective against syndesmosis injury, with resultant Salter–Harris I or II fracture of distal tibia and/or fibula instead [156].

Lateral Ankle Ligaments (Anterior Talofibular, Calcaneofibular, and Posterior Talofibular Ligaments)

Anatomy

- The lateral ankle ligaments are comprised of the anterior talofibular ligament, the posterior talofibular ligament, and the calcaneofibular ligament (Fig. 36.23).

Mechanism of Injury in Sports

- Basketball most common; other high risk sports include soccer, volleyball, football, running, dance, and gymnastics [157, 158].
- Mechanism due to inversion of the ankle.

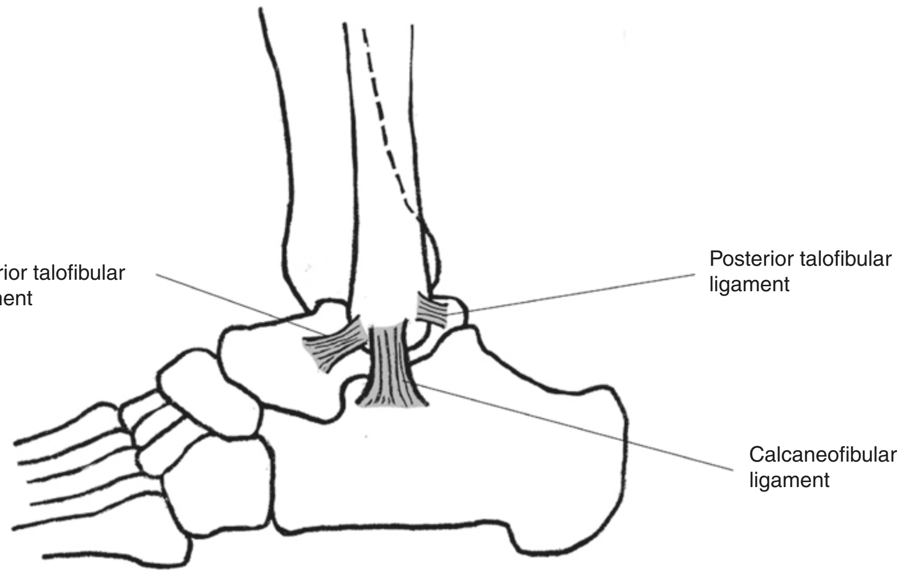

Fig. 36.23 Anatomy of the lateral ankle ligaments

- Previous injury is a strong risk factor for future injury [159–161].
- Preventive bracing without history of previous injury has not been shown to be effective for non-elite athletes [162].

Epidemiology

- Most common injury sustained in sports [157]
- Estimated annual incidence of 215 per 100,000 in the USA, with a higher incidence among athletic populations [158]
- Males, higher incidence between ages 15 and 24 years [158]
- Females, higher incidence after age 30 [158]
- Anterior talofibular ligament (ATLF) predominantly injured [163, 164]

Injury Classification

- Grade 1 – Sprain
- Grade 2 – Partial tear
- Grade 3 – Complete tear

Clinical Presentation

- Patients usually recall inversion injury with audible "pop."
- Lateral ankle pain and difficulty weight bearing.
- May have sensation of instability.
- Swelling and ecchymosis over the lateral ankle.
- Tenderness directly over ATFL or CFL.
- Provocative testing is less useful in acute setting due to patient guarding; however, delayed examination 5 days after injury has 98% sensitivity and 84% specificity for ATFL injury [165].
- Physical examination maneuvers:
  - Anterior drawer test
  - Talar tilt
- Evaluate for concomitant injuries:
  - Palpate bony landmarks including bilateral malleoli, navicular, and fifth metatarsal base to assess for fracture, per the Ottawa ankle rules for diagnosing ankle fractures [166, 167]
  - Medial ankle examination for deltoid injury
  - Squeeze test and dorsiflexion-external rotation test for syndesmosis injury

Diagnosis

- Three-view plain radiographs of the ankle to rule out fractures only if indicated by the Ottawa ankle rules: tenderness at any of the four bony landmarks listed above or inability to bear weight either immediately or in the emergency department [166, 167].
- Ultrasound has been shown to have excellent accuracy (91–95%) of detecting ATFL injury, but is operator dependent [168–170].
- MRI only indicated for acute injuries in elite athletes.

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs.
- Partial weight bearing as tolerated with crutches.
- Elastic compression wrap plus semirigid air stirrup brace leads to faster return to full weight bearing [171].
- Consider immobilization with walking boot for comfort in higher-grade injuries.

Indications for Orthopedic Referral

- Consider early referral to orthopedics for elite athletes with grade 3 injuries [172–174].
- Nonurgent referral for surgery after 6 months of failed nonoperative treatment.

Follow-Up Care

- Nonsurgical management with a progressive rehabilitation program is indicated.
- Early range of motion is recommended:
  - May consider short period <10 days of immobilization with short leg cast or walking boot in grade 3 injuries [173]
- Up to 6 weeks of functional bracing with tape, semirigid brace, or lace-up brace for grade 2 and 3 injuries, in conjunction with a progressive rehabilitation program [175, 176].
- Platelet-rich plasma has minimal effect on recovery, compared with standard treatment [177].

Return to Sports

- Typically after full ROM, strength, and proprioception have been regained.
- Traditionally, athletes return within 2–3 weeks or even immediately [129]; however, premature return to sports may be risk factor for recurrent injury:
  - Postural control is impaired for at least 21 days post injury [178].
  - Around 60–90% athletes resumed pre-injury levels of sport at 12 weeks [179].
- Around 10–12 weeks after surgery for grade 3 injuries [180].
- Prophylactic bracing or taping should be used for 6 months upon return to sports to decrease risk of recurrent injury [181, 182]:
  - Consider continued use for up to 12 months post injury [183].

Complications

- Around 20% of injuries develop chronic instability [184].
- Inadequately treated injuries can lead to recurrent injury and additional time lost from sports.

Pediatric Considerations

- Isolated ligament injury less likely in the skeletally immature athlete.
- Avulsion fracture of lateral malleolus can be treated as a lateral ankle sprain [185].
- Salter–Harris I fracture of distal fibula presents similar to lateral ankle sprain, with normal radiographs and tenderness over distal fibula:
  - Historically managed with immobilization and orthopedic follow-up
  - Recent prospective cohort study found incidence of fracture to be only 3%, and no difference in outcomes when treated as a lateral ankle sprain [186]
- Unique fracture patterns in athletes nearing skeletal maturity between age 12 and 15, due to the medial portion of the physis being closed:
  - Salter–Harris III fracture of lateral distal tibial physis (Tillaux fracture) – eversion mechanism
  - Salter–Harris IV fracture of distal tibia (triplane fracture) – external rotation mechanism

Deltoid Ligament

Mechanism of Injury in Sports

- Football, basketball, soccer, long jump, and triple jump [187]
- Requires considerable force to be disrupted
- Common mechanisms for injury:
  - Pronation and eversion
  - Supination and external rotation

Epidemiology

- Accounts for 3–4% of all ankle ligament injuries [164, 187]
- Rarely occurs in isolation; often associated with ankle fractures or syndesmosis injury

Injury Classifications

- Partial or complete rupture; traditional 1–3 grading for ligament injuries is not typically used for deltoid injuries.
- Acute injuries can be further categorized as [187]:
  - Isolated deltoid injury
  - Associated with ankle fracture(s)
  - Combination ligament injuries

Clinical Presentation

- Medial ankle pain and instability with weight bearing
- Patients may feel a “pop”
- Inability to ambulate

- Ecchymosis and tenderness at medial ankle over deltoid ligament
- Evaluate for concomitant injuries:
  - Palpate bony landmarks including malleoli and proximal fibula to assess for fracture
  - Anterolateral ankle examination for lateral sprain
  - Squeeze test and dorsiflexion-external rotation test for syndesmosis injury

Diagnosis

- Three-view plain radiographs of the ankle to exclude fractures:
  - Isolated injuries may have normal radiographs.
  - Stress views are not recommended due to lack of additional information and potential for further damage [188].
- Ultrasound has been shown in three small series to have excellent sensitivity (100%) and specificity (92–100%) for diagnosis of complete deltoid ruptures, but is much less sensitive (50%) for partial tear and is limited by operator skill [189–191]:
  - May help guide management by distinguishing between stable and unstable injuries
- MRI helpful to assess extent of ligament injury and to identify other injuries or osteochondral lesions.
- CT if further characterization of associated fracture or avulsion is needed.

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs
- Immobilize and non-weight bearing if associated fracture or syndesmosis injury suspected

Indications for Orthopedic Referral

- Referral to orthopedics for surgical management is indicated for:
  - Unstable ankle fractures
  - Unstable syndesmotic injuries
  - Acute ruptures in professional athletes
- No clear time frame for operative management has been described; urgent referral within 1 week is suggested given unstable nature of injury.
- Nonurgent referral for surgical treatment in cases of failed conservative management or chronic instability.

Follow-Up Care

- Isolated stable injuries are treated with period of immobilization and non-weight bearing followed by progressive rehabilitation program.
- No consensus on length of immobilization; recommendations vary from 1 to 6 weeks [188, 192].

Return to Sports

- Limited evidence available to guide return to sports
- Generally following successful completion of progressive rehabilitation program without pain or instability
- At least 4 weeks for stable, isolated injuries treated conservatively [185]
- As early as 8–10 weeks after surgical repair [193]
- One cohort of elite football players returned to high-level competition 6 months after surgery [194]

Complications

- Missed injuries or inadequate treatment can lead to chronic instability, hindfoot valgus deformity, and osteoarthritis [188].

Pediatric Considerations

- Deltoid injuries in skeletally immature patients are poorly described, however have been described to occur with ankle fractures [195, 196].
- Associated medial malleolar physeal injuries can lead to growth arrest and ankle deformity [197].

Lisfranc Ligament

Anatomy

- The Lisfranc ligament stabilizes the Lisfranc joint complex between the first (medial) cuneiform and the base of the second metatarsal (Fig. 36.24).

Mechanism of Injury in Sports

- Most commonly seen in football, women's gymnastics, equestrian sports, and surfing [198–200]
- Mechanisms for low-energy athletic injuries:

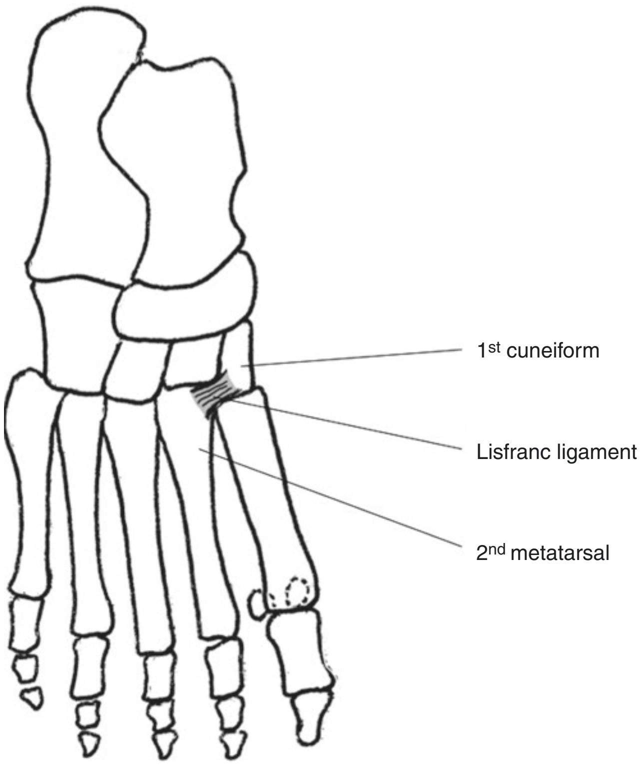

Fig. 36.24 Anatomy of the Lisfranc ligament

- Forced plantar flexion with axial load (football, gymnastics)
- Sudden forefoot abduction against fixed hindfoot (foot caught in stirrup)

- Can also occur in high-energy trauma outside of sports

Epidemiology

- Previous reported annual incidence of 1.8 per 100,000 in the USA [201]
- Higher incidence in athletes; 4% of collegiate football players [202]
- Rarely an isolated injury; >90% associated with midfoot or metatarsal fractures, dislocations, or fracture dislocations [203, 204]

Injury Classification

- Widely used Nunley and Vertullo classification for low-energy injuries [205]:
  - Stage 1 – midfoot pain and inability to participate in sports, without radiographic instability
  - Stage 2 – 1 to 5 mm of diastasis at Lisfranc joint without loss of arch height
  - Stage 3 – >5 mm diastasis at Lisfranc joint with loss of arch height
- Proposed modified classification [206]:
  - Grade 1 – tenderness on examination without radiographic instability
  - Grade 2 – ≤2 mm displacement on radiographs
  - Grade 3 – >2 mm diastasis

Clinical Presentation

- Patients usually recall awkward twisting injury to the foot.
- Pain with weight bearing or inability to weight bear.
- Midfoot pain walking down stairs.
- Inability to perform single-limb heel raise [207].
- Plantar ecchymosis is strongly suggestive for Lisfranc injury [208].
- Tenderness directly over dorsal TMT joint.
- A variety of physical examination maneuvers have been described to stress Lisfranc joint:
  - Passive plantarflexion and rotation of foot
  - Midfoot compression
  - Dorsal/plantar manipulation of first metatarsal head
  - Forefoot compression at metatarsal heads

Diagnosis

- Up to 20% of injuries missed on initial radiographs [209].
- Three-view weight bearing plain radiographs of the foot, including weight bearing bilateral AP view for comparison with contralateral foot:
  - Fleck sign – avulsion fracture of base of second MT or distal medial cuneiform indicates unstable injury (Fig. 36.25).
  - Abduction stress views can help reveal dynamic instability [210].
- MRI is helpful in injuries without obvious findings on plain radiographs.

Initial Management

- Supportive care with ice, compression, elevation, and NSAIDs
- Immobilize with splint or walking boot
- Non-weight bearing with crutches

Fig. 36.25 AP radiograph of the foot showing an avulsion fracture at the base of the second metatarsal, known as the Fleck sign

Indications for Orthopedic Referral

- Referral to orthopedics for surgical management is indicated for all stage/grade 2 and 3 injuries.
- Optimal timing of surgery is unclear; delayed percutaneous fixation between 2 and 6 weeks is generally acceptable [198, 207, 210, 211].
- Delayed surgery >6 weeks may require open reduction or arthrodesis.

Follow-Up Care

- Nonoperative management is recommended for stage/grade 1 injuries:
  - Non-weight bearing in short leg cast for 6 weeks, followed by walking boot for 4 weeks if continued pain [205].
  - Immobilize for 2 weeks in walking boot with protected weight bearing, followed by reassessment for tenderness or radiographic instability before progressing to weight bearing as tolerated in boot and discontinuing boot after pain free with abduction stress, usually 6–8 weeks after injury [210].
- Stiff soled shoe with rigid orthotic support for 6 months after injury.

Return to Sports

- Same criteria and timeline for return to sports for both nonoperative and surgical management: [210]
  - Resume sports-specific training after athlete can walk down several flights of stairs pain free.
  - No cutting or running on uneven surfaces for at least 3–4 months and only after being able to perform single-leg hop without pain.
- Expect 6 months before return to competition:
  - >90% of elite athletes able to return to high level competition after 6–11 months [212, 213].
- About 35% of foot and ankle specialists do not recommend return to high impact sports such as football, basketball, and soccer after Lisfranc arthrodesis surgery [205].

Complications

- Delayed diagnosis or management can lead to prolonged return to play.
- Significant proportion develop posttraumatic arthritis even after surgery.
- Postsurgical complications include complex regional pain syndrome, symptomatic hardware, hardware failure, superficial or deep peroneal nerve injury, loss of reduction.

Pediatric Considerations

- Paucity of literature exists on pediatric Lisfranc injuries, with no data to sufficiently guide treatment plans or long-term prognosis [214].

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Bursa

37

Morteza Khodaee

Key Points

- Traumatic superficial bursitis is an uncommon condition in sports.
- Aspiration under sterile condition can improve symptoms and shorten the recovery and return to sports particularly in patients with acute large traumatic superficial bursitis.

Introduction

A bursa is a fluid-filled synovial pouch that can be deep or superficial and functions as a cushion to reduce friction between two structures such as the tendon, bone, or skin [1]. Superficial bursae are located in the subcutaneous tissue between the bone and overlying skin. There are many superficial bursae in the body, but only olecranon, prepatellar, superficial infrapatellar, and subcutaneous calcaneal bursitis have been reported in the literature. Historically, enlargement of the bursa (e.g., prepatellar and olecranon) has been called "bursitis," although in the majority of cases no true inflammatory process exists [2]. Chronic microtrauma is the most common type of superficial bursitis as a result of chronic friction of the overlying tissue and underlying bony prominence [3].

Acute Traumatic (Hemorrhagic) Superficial Bursitis

The exact incidence of acute traumatic (hemorrhagic) superficial bursitis is unknown, but it seems to be an uncommon finding among athletes. This could be due to the fact that most cases are treated successfully in the community and most studies are done in hospital settings. In addition, many patients have mild symptoms and most likely do not seek medical attention. Olecranon and prepatellar are the most common locations of traumatic superficial bursitis. In rare cases, traumatic superficial infrapatellar and subcutaneous calcaneal bursae can also be affected. There is no reported cases of acute traumatic (hemorrhagic) bursitis in deeper bursae such as subacromial and iliopsoas. Acute traumatic olecranon bursitis is more common among male adult athletes (e.g., wrestlers and gymnasts). Acute traumatic prepatellar bursitis is more common among athletes playing sports with a risk of fall on the knees particularly playing on turf surface [1–5]. Similar to prepatellar bursitis, superficial infrapatellar (pretibial) bursitis can happen with the same mechanism. Subcutaneous calcaneal bursitis is common among figure skaters; however, the mechanism of injury is usually microtrauma due to improperly fitting footwear or an underlying bony prominence (Haglund's deformity) [6].

Acute traumatic bursitis typically occurs due to a direct blow to the area causing bleeding into the superficial bursa [2, 3, 7]. In athletes with coagulopathies or on anticoagulation therapy, spontaneous hemorrhagic bursitis may occur [1]. Despite different anatomical locations, etiologies, and prevalence, traumatic superficial bursitis share similar clinical presentations, types, and management.

Clinical Presentations

When evaluating a patient with superficial bursitis, it is imperative to differentiate between traumatic (either acute or chronic), aseptic inflammatory, and septic bursitis. Patients with acute traumatic and inflammatory superficial bursitis often present with significant pain, tenderness, and decreased range of motion of the adjacent joint. Taking a comprehensive history including presence of a recent trauma or any occupational or recreational risk factors is important as these

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

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conditions are more common among particular athletes and professions. A clinical history of skin trauma (break) near an affected bursa leads to a significantly higher risk of septic bursitis [7–11]. Patients with acute traumatic bursitis often present with significant pain, tenderness, and decreased range of motion of the adjacent joint. Localized swelling (bursal enlargement) is the hallmark of the superficial bursitis (Fig. 37.1). The swelling is usually limited to the subcuta-

Fig. 37.1 Acute traumatic (hemorrhagic) superficial bursitis: (**a**) a traumatic left olecranon bursitis in a 50-year-old male 6 weeks after falling on his elbow; (**b**) 10 ml serosanguinous fluid was aspirated; (**c**) a traumatic right prepatellar bursitis in a 35-year-old woman 2 weeks after falling on a rock directly on her knee; (**d**) short axis ultrasound confirmed the diagnosis of prepatellar bursitis (∗); (**e**) 5 mL of hemorrhagic fluid was aspirated

neous tissue over the bony prominence and does not extend to the underlying joint, unless concomitant cellulitis or inflammation of adjacent tissues are present.

Diagnosis

The differential diagnosis of acute traumatic superficial bursitis is broad (particularly in cases of >2–3 days post-trauma) and includes adjacent joint effusion (e.g., hemorrhage), septic and inflammatory arthritis, cellulitis, and Morel-Lavallée lesion [12, 13]. Plain radiography is only indicated if there is a concern for fracture, retention of foreign body, or concern for underlying bony abnormality (e.g., Haglund's deformity) or calcification [1, 9, 14]. Ultrasound can be a useful modality in visualizing the enlarged bursa particularly in cases of significant soft tissue edema due to cellulitis or abrasion (Fig. 37.1d) [8, 15]. Color Doppler may help visualize inflammatory process in and around the bursa. Ultrasound can also help with needle placement for aspiration in difficult cases (e.g., smaller bursae and adjacent cellulitis or abrasion) [8, 15]. Though the presence of enhancement in MRI cannot differentiate between septic and aseptic bursitis, the absence is indicative of aseptic bursitis [8, 16, 17].

After history, physical examination, and applicable blood tests and imaging, if there is still uncertainty regarding the etiology of the bursitis, and particularly if there is a high suspicion of septic bursitis, an aspiration under sterile conditions of the bursal fluid should be performed [7, 8, 10, 13, 18–21]. A Z-track method of inserting the needle in which the skin and deeper layers are not in the same plane after needle placement helps seal the path [22]. This can be achieved by pulling the overlying skin during the needle insertion. The bursal aspiration should be performed with a large-bore needle (e.g., 18–22 gauge) prior to the administration of the first dose of antibiotics [18]. The aspirated fluid should be sent for cell count, gram stain, culture, glucose, and crystal analysis [9, 13, 14]. In general, bursal fluid that is cloudy or purulent in appearance is more likely to represent a septic bursitis. Obviously, bursal fluid appears bloody in hemorrhagic bursitis. In addition to aiding in diagnosis, bursal aspiration can improve symptoms and reduce bacterial load in septic bursitis and may be repeated daily as part of the treatment regimen if indicated [7, 8, 18]. The use of a compressive bandage after aspiration may help reduce re-accumulation of the bursal fluid [7, 18, 23]. It seems that aspiration in patients with therapeutic INR is safe and does not significantly increases the risk of hematoma or bleeding [24].

Treatment

After ruling out fractures, most acute traumatic superficial bursitis cases can be managed conservatively with ice, elevation, relative rest, and analgesics [3]. Bursal aspiration may shorten the duration of symptoms in cases of acute hemorrhagic bursitis with significant bursal enlargement and interference with daily activities (Table 37.1). It is critical to weigh the potential benefits over the potential risks of aspiration on an individual basis. If aspiration is considered, it is better to be performed in a few hours of its occurrence or after the disintegration of the coagulated bursal fluid is started a couple of weeks later [3]. Padding may decrease the risk of traumatic hemorrhagic bursitis in high-risk sports (e.g., wrestling). Padding should not limit the adjacent joint range of motion.

Table 37.1 SORT: key clinical recommendations

Clinical recommendation	Evidence rating
Bursal fluid aspiration should be performed in patients with acute large traumatic superficial bursitis [2, 3, 5, 25]	C
Bursal fluid aspiration and culture should be performed in patients with suspecting septic superficial bursitis [7, 13, 14, 18, 19, 21, 26–29]	C

Return to Sports

Athletes should be able to return to their sports when the affected bursitis is not interfering with their performance.

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Nerve

38

Benjamin Marshall and Rachel Brakke Holman

Key Points

- Peripheral nerve injuries in sport, though common, are usually brief and self-limited
- Higher-grade injuries are generally categorized into three major subtypes as detailed in Fig. 38.1.
- Most injuries can be managed nonoperatively with protection/splinting and therapeutic exercise
- Prognosis is highly dependent on severity of injury and can be further delineated with NCS/EMG testing
- NCS/EMG testing is commonly performed at >3 weeks, 3 months, and 6 months following acute nerve injury
- Return to sport decisions can be complex and are generally achieved through shared decision-making regarding the degree of functional recovery and susceptibility of nerve to reinjury during participation

Introduction and Neuroanatomy

Peripheral nerve injury is a relatively common occurrence in sport and recreation though usually relatively mild; more severe injuries do occur and can result in significant disability if not recognized and managed appropriately. Knowledge of the basic structure and functional characteristics of the peripheral nervous system is essential to the diagnosis and management of nerve injury. Peripheral nerves contain an assortment of nerve fiber types that vary in the information they are transmitting (e.g., pain, temperature, pressure, motor signaling) and their microscopic anatomy (e.g., diameter, degree of myelination). Generally larger and more myelinated nerve fibers conduct signals faster and are more specific in functional innervation. For example, small unmyelinated C fibers transmit dull poorly localized pain signals, while large A-delta "fast" fibers transmit sharp, well-localized pain.

Somatic peripheral nerves emanate from the spinal nuclei where the primary neurofunctional organization for the upper and lower extremities occurs in the brachial and lumbosacral plexuses, respectively, and then continue as singular nerves to their various target tissues. These peripheral nerves are supported externally by a connective tissue sheath, the epineurium, which encases several nerve groupings or fascicles, supported by their own perineural connective tissue and the associated vascular supply. Inside of each nerve fascicule are several individual nerve fibers insulated by an endoneurial connective tissue sheath. Each individual neural axon is supported by Schwann cells which form myelin sheaths and facilitate more rapid, saltatory conduction. This organizational hierarchy of connective tissue scaffolding plays a central role in nerve injury and regeneration.

Nerve Injury Severity Classification

The most clinically useful classification system of nerve injury was first proposed in the 1940s by Dr. Herbert Seddon, a British orthopedist, and remains relatively unchanged today. It separates neural injury into three categories of increasing severity *(*Fig. 38.1*)*. Neuropraxia is the least severe and represents a focal lesion to the nerve myelin sheath without damage of the underlying axon. These types of injuries represent a dysfunction in the transmission of neural depolarization, termed a conduction block, and can result in weakness or sensory loss that can last from minutes

B. Marshall (✉)
Department of Physical Medicine and Rehabilitation, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: Benjamin.j.marshall@cuanschutz.edu

R. B. Holman
Department of Physical Medicine and Rehabilitation, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: RACHEL.BRAKKEHOLMAN@cuanschutz.edu

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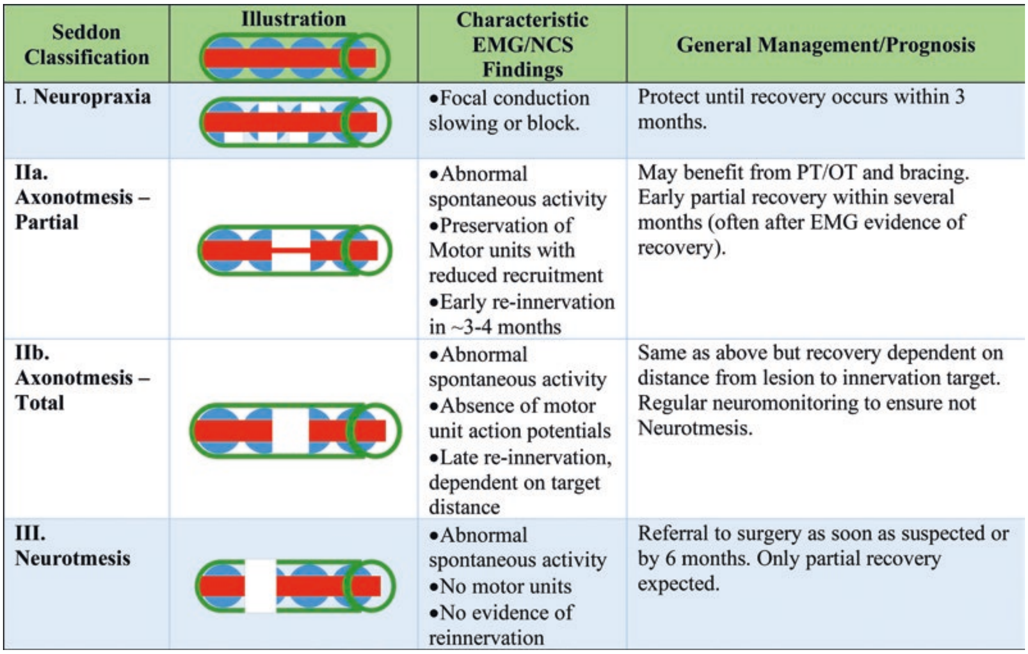

Seddon Classification	Illustration	Characteristic EMG/NCS Findings	General Management/Prognosis
I. **Neuropraxia**		•Focal conduction slowing or block.	Protect until recovery occurs within 3 months.
IIa. Axonotmesis – Partial		•Abnormal spontaneous activity •Preservation of Motor units with reduced recruitment •Early re-innervation in ~3-4 months	May benefit from PT/OT and bracing. Early partial recovery within several months (often after EMG evidence of recovery).
IIb. Axonotmesis – Total		•Abnormal spontaneous activity •Absence of motor unit action potentials •Late re-innervation, dependent on target distance	Same as above but recovery dependent on distance from lesion to innervation target. Regular neuromonitoring to ensure not Neurotmesis.
III. Neurotmesis		•Abnormal spontaneous activity •No motor units •No evidence of reinnervation	Referral to surgery as soon as suspected or by 6 months. Only partial recovery expected.

Fig. 38.1 Nerve injury categories

to a maximum of 3 months. These generally have an excellent prognosis for complete recovery, though they may be difficult to distinguish from more severe injury initially [11]. For example, following a "stinger"-type injury, a player may report complete upper extremity paresis that resolves in minutes to hours.

Axonotmesis is the next level of severity and represents irrevocable damage to the axon in addition to its myelin sheath but without substantial damage to the supporting connective tissue scaffold (e.g., endoneurium or perineurium). This axonal discontinuity results in denervation of the target tissue with immediate clinical deficits from loss of neuronal transmission. With a focal axonal injury, unlike isolated myelin damage, the neuron will undergo Wallerian degeneration of the distal axonal segment starting within several days following the insult. This active degenerative process occurs to the orphaned axon may occur over a period of days to weeks. The length of this process which is mostly determined by the distance from the location of axonal injury to the target tissues. The cardinal feature of axonotmesis is the capacity for axonal regrowth. Though the axonal damage is irreparable, with the neuronal tract still in place, the axon will subsequently regrow down the intact scaffold to reinnervate its prior target. This process of regeneration, however, is notoriously slow compared to other soft tissue healing and generally occurs at a rate of 1 mm/day or approximately 1 in./month. Occasionally adjacent intact neurons can sprout additional terminal branches to innervate the orphaned tissue more rapidly. This process can occur within a few weeks but generally takes several months to form mature synapses [19]. A relatively common example of this would be axillary nerve injury following proximal humeral fractures or glenohumeral dislocations. This may significantly prolong recovery from the initial trauma from months to a year or more given the resultant deltoid weakness.

The most severe category of nerve injury, neurotmesis, is notable for complete discontinuity of the axon, myelin sheath, and surrounding connective stroma. Without any substantive neural continuity, self-regeneration is not possible without surgical intervention [9]. This generally does not occur in sport without a significant concomitant injury such as peroneal nerve rupture during a severe multiligamentous knee injury.

Epidemiology and Mechanism of Injury

Injury to a peripheral nerve can occur at any time during competition or practice but is rarely severe enough to cause significant debility. For example, arguably the most common injury in sport is the "stinger" or "burner" which is a traumatic upper brachial radiculoplexopathy that often results from rapid neck and shoulder distraction. This injury is common in contact sports with 50–60% of American football and rugby union athletes retrospectively acknowledging at least 1 prior episode or at an observed rate 1.5 occurrences per NCAA football team/season [3, 10]. However, fortunately, 64% of the football athletes had resolution within hours, and <6% of injuries resulted in a loss of playing time longer than 3 weeks [10]. When severe nerve injury does occur, it is

commonly in association with adjacent soft tissue injury or fracture [17]. Additionally, iatrogenic nerve injury from direct insult or positioning is a known, although rare, complication of many sport surgeries [15]. Mechanism of injury varies between sports and even among various positional players within single sports (Table 38.1).

Table 38.1 Common nerve injuries by sport

1. Archery
(a) Digital nerve compression
(b) Median neuropathy at wrist
(c) Median neuropathy at pronator teres
(d) Long thoracic nerve palsy
2. Arm wrestling
(a) Radial nerve palsy
3. Auto racing
(a) Brachial plexopathy
(b) Sciatic neuropathy
(c) Peroneal neuropathy
4. Ballet dancing
(a) Suprascapular neuropathy
(b) Femoral neuropathy
(c) Peroneal neuropathy
(d) Sural neuropathy
(e) Dorsal cutaneous neuropathy
(f) Morton's neuroma
5. Baseball
(a) Suprascapular neuropathy
(b) Radial neuropathy
(c) Ulnar neuropathy
(d) Musculocutaneous neuropathy
(e) Median neuropathy at pronator teres
(f) Thoracic outlet syndrome
(g) Axillary neuropathy with quadrilateral space syndrome
(h) Digital neuropathy at thumb
(i) Brachial plexopathy (pitcher's arm)
6. Basketball
(a) Suprascapular neuropathy
(b) Stinger
(c) Median neuropathy at the wrist (wheelchair athletes)
(d) Ulnar neuropathy at the wrist (wheelchair athletes)
7. Bicycling
(a) Ulnar neuropathy at Guyon's canal
(b) Ulnar neuropathy at the elbow
(c) Median neuropathy at the wrist
(d) Pudendal neuropathy
(e) Posterior cutaneous nerve of the thigh neuropathy
(f) Sciatic nerve palsies (unicyclists)
8. Bodybuilding/weight lifting
(a) Ulnar neuropathy at the deep motor branch
(b) Ulnar neuropathy at flexor carpi ulnaris
(c) Ulnar neuropathy at the deep palmar branch
(d) Ulnar neuropathy at the elbow
(e) Posterior interosseous neuropathy
(f) Medial pectoral neuropathy
(g) Suprascapular neuropathy
(h) Median neuropathy at the wrist
(i) Long thoracic neuropathy
(j) Lateral antebrachial cutaneous neuropathy
(k) Musculocutaneous neuropathy

Table 38.1 (continued)

(l) Femoral neuropathy
(m) Thoracodorsal neuropathy
(n) Dorsoscapular neuropathy
(o) Stinger
(p) Rectus abdominis syndrome with rhabdomyolysis
9. Bowling
(a) Digital neuropathy of the thumb
10. Boxing
(a) Stinger
11. Cheerleading
(a) Digital neuropathy
(b) Median neuropathy at the palmar branch
12. Football
(a) Stinger
(b) Upper trunk brachial plexopathy
(c) Radiculopathy of C5, C6, L5, or S1 roots
(d) Axillary neuropathy with or without dislocated shoulder
(e) Suprascapular neuropathy
(f) Ulnar neuropathy at the elbow
(g) Median neuropathy at the wrist
(h) Long thoracic neuropathy
(i) Radial neuropathy
(j) Thoracic outlet syndrome
(k) Iliohypogastric neuropathy
(l) Peroneal neuropathy with knee dislocation
(m) Sciatic nerve (hamstring syndrome)
13. Frisbee
(a) Posterior interosseous neuropathy
14. Golf
(a) Median neuropathy distal to wrist
(b) Carpal tunnel syndrome (CTS)
(c) Ulnar neuropathy at flexor carpi ulnaris
15.Gymnastics
(a) Posterior interosseous neuropathy
(b) Lateral femoral cutaneous neuropathy
(c) Femoral neuropathy
16. Handball
(a) Handball goalie's elbow
17. Hockey
(a) Stinger
(b) Axillary neuropathy
(c) Tibial neuropathy attributable to tarsal tunnel syndrome
(d) Peroneal neuropathy
18. In-line skating, roller skating, and skateboarding
(a) Superficial peroneal neuropathy
19. Judo, karate, and kickboxing
(a) Morton's neuroma of a plantar nerve
(b) Ulnar neuropathy at trauma site
(c) Axillary neuropathy at trauma site
(d) Spinal accessory neuropathy at trauma site
(e) Long thoracic neuropathy at trauma site
(f) Peroneal neuropathy at trauma site
20. Mountain climbing, hiking
(a) Tarsal tunnel syndrome
(b) Rucksack paralysis—brachial plexopathy (upper and middle trunks)
(c) Suprascapular neuropathy
(d) Axillary neuropathy
(e) Long thoracic neuropathy

(continued)

Table 38.1 (continued)

21. Rugby/Australian rules football
(a) Axillary neuropathy
(b) Obturator neuropathy
22. Running
(a) Peroneal neuropathy
(b) Lateral femoral cutaneous neuropathy
(c) Tibial neuropathy at the tarsal tunnel
(d) Posterior tibial neuropathy
(e) Morton's neuroma of a plantar nerve
(f) Interdigital neuropathies
(g) Plantar neuropathies
(h) Calcaneal neuropathy
(i) Sural neuropathy
(j) Superficial peroneal neuropathy
(k) Saphenous neuropathy
(l) Rhabdomyolysis
23. Scuba diving
(a) Lateral femoral cutaneous neuropathy
24. Shooting
(a) Long thoracic neuropathy
25. Skiing, snowboarding, sledding, and ski jumping
(a) Femoral neuropathy (cross-country skiing)
(b) Ulnar neuropathy (cross-country skiing)
(c) Brachial plexus injury (snowboarding)
26. Snowmobiling and all-terrain vehicle riding
(a) Brachial plexopathy
(b) Ulnar neuropathy at Guyon's canal
27. Soccer
(a) Peroneal neuropathy
28. Surfing
(a) Common peroneal neuropathy
(b) Saphenous neuropathy
29. Swimming
(a) Thoracic outlet syndrome
30. Tennis/racquetball
(a) Posterior interosseous neuropathy at the arcade of Frohse
(b) Suprascapular neuropathy
(c) Long thoracic neuropathy
(d) Lateral antebrachial cutaneous neuropathy
(e) Radial neuropathy secondary to fibrous arches at lateral head of triceps
(f) Digital neuropathy
(g) Superficial radial neuropathy attributable to constrictive sweatband
(h) Thoracic outlet syndrome
31. Volleyball
(a) Suprascapular neuropathy
(b) Axillary neuropathy
(c) Long thoracic neuropathy
32. Wrestling
(a) Stinger
(b) Brachial plexopathy
(c) Axillary neuropathy
(d) Ulnar neuropathy
(e) Median neuropathy at the wrist
(f) Long thoracic neuropathy
(g) Suprascapular neuropathy
33. Yoga
(a) Sciatic neuropathy

Adapted from: Toth ([21])

Generally, peripheral nerve injury occurs from laceration, traction, compression, or ischemia. Laceration is a very uncommon cause of sport-related injury, can be either "blunt" or "sharp," and is usually due to forceful contact with sporting equipment such as ice skates or a field hockey stick. Stretch- or traction-type injury is more common but requires a significant addition of kinetic energy to result in substantial neural damage. This is due to the peripheral nerves inherent accommodation to stretch. They are composed primarily of connective elastic and fibrous tissue which can generally accept 10–20% additional elongation without structural failure [1]. These nerves also have varying degrees of excess length to allow for dynamic alterations in limb positioning. Thus stretch injuries tend to occur in locations where these mechanical advantages are minimized, such as the axillary nerve, which has a relatively short distance to its terminal innervation from a proximal anchor on the substantially more stable brachial plexus. Compression injury typically occurs from blunt trauma to a superficial nerve. Superficial nerves are particularly susceptible to crush injury as they pass over nonconforming bony prominences such as the peroneal nerve at the fibular head or the ulnar nerve at the medial epicondyle of the humerus. Chronic low-grade compression also commonly results in nerve injury, often where nerves are contained by fibro-osseous structures such as the carpal or tarsal tunnels. Ischemia is a rare form of injury as nerves are typically more resistant than skeletal muscle. Significant neuropathy usually requires >8 h of hypoxemia which may occur from microvascular damage or associated compression from a hematoma or compartment syndrome [6].

Diagnosis and Imaging

Diagnosis of peripheral nerve injury starts with localization of the injury along the neuroaxis. This is usually accomplished with a comprehensive history and peripheral neuromuscular exam. In most cases, seemingly similar complaints (i.e., grip weakness from ulnar mononeuropathy or lower cervical radiculopathy) can be confidently discriminated in this manner. In cases where localization is difficult or still unclear, nerve conduction studies or electromyography (EMG/NCS) may be utilized as an extension of the physical exam. EMG/NCS may better characterize the location but can also elucidate the extent of the peripheral nerve lesion. EMG/NCS is a quantitative functional neuromuscular evaluation of peripheral nerve lesions that is generally performed by a neurologist or physiatrist but is not without its limitations. Electrodiagnostic evaluation of acute peripheral nerve injury will locate a nerve injury but only if it is severe enough to significantly affect neuronal function of large sensory or motor fibers. Small caliber neuronal fibers such as sensory C-fiber injuries or dysfunction, for example, may result in substantial clinical symptoms (i.e., paresthesia, dull or achy

pain) but is not evaluated well by EMG/NCS [19]. Furthermore, there are some electrodiagnostic limitations based on injury location. The most relevant of which is sensory neuronal injuries proximal to the dorsal root ganglion such as with radiculopathic injury due to a herniated intervertebral disc. Patients may experience significant radicular pain, paresthesia, or numbness; however, due to the injury's location proximate to the sensory neuronal cell body, a standard electrodiagnostic evaluation may appear normal unless motor neuron function is impaired [19]. Finally, the timing of electrodiagnostic testing may further limit evaluation of the nerve injury. Electrodiagnostic evaluation of axonal lesions is partly dependent on the process of Wallerian degeneration and may be of limited value until this process is completed [4]. For example, EMG/NCS testing performed immediately after an axonometric injury to the ulnar nerve can resemble a neuropraxic lesion as the ulnar nerve distal to the injury is still mostly intact and functional. However, after ~3 weeks following injury, Wallerian degeneration of the ulnar nerve distal to the lesion results in changes in the electrophysiology of the denervated muscle that may be elicited by EMG testing.

EMG/NCS may be most useful to the evaluating physician in the differentiation of the neuronal injury severity. Electrodiagnostic differentiation between neuropraxia, axonotmesis, and neurotmesis is based on the functional characteristics of the nerve and innervated muscle. Neuropraxic lesions demonstrate focal slowing of nerve conduction across the lesion due to disruption of saltatory conduction from demyelination. Such slowing may be so severe that neural depolarization fails to propagate across the lesion resulting in a complete conduction block. Complete blocks may be difficult to distinguish from axonal lesions initially and partly rely on timing of evaluation, as discussed above, to convincingly discriminate the two. Axonometic injury is largely determined by the electrophysiologic response of the denervated muscle tissue. Such orphaned muscle cells spontaneously depolarize in stereotyped patterns that can be characterized with electromyography. Neurotmetic injuries demonstrate similar patterns of increased spontaneous activity of myocytes but fail to demonstrate restoration of immature motor unit action potentials, the so-called reinnervation or nascent potentials, which develop with restoration of neural continuity. Practically this suggests EMG/NCS testing is most useful ~3 weeks and 2–3 months after injury for an athlete with ongoing neurologic deficits. At 3 weeks to determine if the injury was predominantly neuropraxic or if significant axonal damage occurred and then at 3 months, to determine if the axonal injury appears to be the result of an axonometric or neurotmetic injury giving the provider a better understanding of prognosis and guiding treatment recommendations.

Magnetic resonance imaging (MRI) may occasionally also play a role in diagnostic workup of an acute nerve injury. Generally the utility of MRI is in the analysis of associated or contributing extraneural factors such as a compressive cyst or bone fragment as well as ligament or tendinous disruption. MRI may note other features suggestive of nerve injury including increased neuronal caliber or T2 hyperintensity with loose fascicular definition, possibly with eventual neuroma formation which would suggest some degree of stroma disruption [16]. Some imaging facilities and academic centers have started to offer MR neurography to aide in the assessment of neuronal imaging. Currently there is no standard definition of what this imaging sequence entails, but it is generally composed of high-resolution T1- and a heavily fat-suppressed T2-weighted series to better visualize pathologic changes in neural tissues. Another imaging technology that is not yet readily available for clinical use but has significant potential for improving evaluation of nerve injury is MR diffusion tensor tractography (DTT). DTT is a novel imaging modality that has the potential to provide more quantitative information on axonal continuity following injury [12]. In addition to MR technologies, high-resolution ultrasound is increasingly used by physicians in the diagnosis and treatment of nerve injury [18]. Over the past decade, improvements in musculoskeletal ultrasound technology, as well as the increasing availability and affordability of commercial units, have allowed for incorporation of this technology into many sports practitioners' clinical practice. The primary advantage of ultrasound for the imaging of nerve injury is its ability to dynamically access the nerve in real time. This is particularly useful in athletes where nerve instability, such as subluxation of the ulnar nerve at the elbow, is relatively common. High-resolution ultrasound is not without limitation as imaging of nervous tissue near bone or at increasing depth limits resolution. Ultrasound is particularly adept at the structural analysis of superficial nerves and can be used in office to further examine the peripheral nerve along the length of its anatomic course. Sonographic-pathoanatomic changes that occur with nerve injury will mirror the findings on MRI. Injured nerves may appear increasingly hypoechoic with a loss of fascicular architecture and may have neuromas-in-continuity [12]. Additionally, many compressive neuropathies result in increased neural caliber relative to proximate nerve segments. As such sonographic diagnostic criteria now exist for an increasing number of neuropathies which appear complimentary to electrodiagnostic testing and have the potential to achieve similar levels of specificity and sensitivity [7].

Prognosis and General Management Principals

General management of nerve injury in an athlete relies heavily on the believed degree of injury severity as this plays a central role in the prognosis and probability of neural

recovery. Isolated neuropraxic injuries generally experience complete recovery of prior neural function within 3 months depending on severity and segmental length of injury. The primary goal in these cases is to identify any ongoing factors, such as compressive equipment or poor sport-specific mechanics, which may be perpetuating the neuronal trauma [4]. Additionally, it is important to protect any at-risk structures due to the nerve dysfunction. An example of this is ankle bracing or the use of an ankle foot orthosis with peroneal nerve injuries as motor dysfunction in the ankle dorsiflexors and evertors increases risk of ankle instability and inversion sprains. Axonometric injury recovery is tied to the degree of adjacent neuronal injury. That is, if a peripheral nerve undergoes only partial axonometric injury, the neighboring intact axons may generate nodal or terminal sprouts that migrate to the denervated tissue to reestablish neuronal signaling [2]. This process is generally faster than would be allowed should these axons need to undergo the full process of Wallerian degeneration and axonal regrowth. Often electrodiagnostic evidence of recovery in these muscles is detected starting approximately 2 months following the injury, though clinically evident recovery generally takes significantly longer. If complete nerve axonal disruption occurs, whether through axonometric or neurotmetic injury, recovery is prolonged and often incomplete. If the injury is clearly a complete neurotmetic disruption, such as in open transection, urgent neurosurgery referral is needed to reappose the nerve. However, most injuries of this severity in sport are closed with an unclear degree of severity despite imaging and electrodiagnostic evaluation. The clinician's role is primarily to track the athlete's progress closely for signs of reinnervation to attempt to better classify the degree of injury. This is typically done with serial physical exams and EMG/NCS at regular intervals starting 3 weeks post-injury. As stated previously, electrodiagnostic testing is more sensitive to early reinnervation in the effected muscle and, if neural continuity is found within 4–6 months, can indicate a good prognosis with ongoing conservative care [9, 19]. If no evidence of neural recovery is found on EMG/NCS within 6 months, the injury is considered to be neurotmetic, and surgical referral is warranted. In these cases, the neural injury was generally substantial enough to disrupt the supporting tissues and connective tissue architecture and not allow for axonal regeneration. The nerve will still attempt to bridge the lesion, but this usually results in a disorganized proliferation of axon, Schwann cells, and connective tissue that results in neuroma scar formation. Surgery in these cases generally consists of resection of this nonfunctional nerve proliferation and either direct or graft-based reanastomosis of the opposing nerve endings to encourage axonal regrowth. The outcome of such an intervention is partly dependent on surgical factors as well as the quality of the innervated tissue. If the target muscle is substantially replaced by fatty atrophy, a process that occurs after 12–24 months, reinnervation and functional recovery become much less likely. Available literature on surgical nerve repair indicates outcomes start declining at about 9 months with a more precipitous decline in outcomes after 12–15 months [9, 13]. Surgical constraints also appear to play a role as direct nerve repair tends to result in the highest degree of functional improvement with less favorable outcomes if grafting is required to maintain a tension-free repair. In cases where nerve injury has persisted past these time points, often only function salvaging surgeries, such as tendon transfer or joint fusion, are available [9, 14].

Return to Sports

The decision to allow an athlete to return to competitive sport following nerve injury can be difficult and the practitioner must balance a multitude of factors to make the best possible recommendation. Unfortunately, there is scarce empirical evidence and no formal organizational guidelines to help with the decision. Therefore, return to play criteria have mostly been guided by expert opinion. Current attempts at a classification system for return to play following nerve injury are most common among "stinger"-type injuries. No system predominates but significant similarities exist among recommendations [5, 20]. Generally, the athlete must experience complete resolution of all symptoms (including during sport-specific tasks) and have pain-free, full, cervical range of motion without the presence of significant known conditions that would predispose to recurrent injury (i.e., cervical stenosis, spondylolisthesis, etc). If the athlete does have recurrent stingers in the same season, generally more advanced workup is warranted before allowing the athlete to return. Possible specialist referral and termination of participation in collision sports, if applicable, should be considered following three or more "stinger" episodes. It is probably reasonable to extrapolate these criteria as a general guideline governing return to play for most peripheral nerve injuries. However, it is even more difficult in an athlete only experiencing partial neural recovery, as this is not an absolute contraindication to sport participation.

It is primarily important that the physician ensure that presence of ongoing neurologic deficits is a result of incomplete terminal recovery and not due to significant ongoing neuronal healing. Differentiating the two is often the task of the treating physician and is accomplished by utilizing the diagnostic modalities discussed in the prior section. If there is incomplete neurologic recovery, there are several important considerations before a return to play decision can be reached. Firstly, has the athlete reached a level of functional recovery that would safely allow for sport-specific tasks? If, because of the ongoing functional deficit, the athlete cannot

perform at a level commensurate with the demands of their sport or if the deficit would result in a higher risk of injuries to other tissues, this could preclude them from returning to sport. This determination is often made in close collaboration with the training room or physical therapy staff since subtle functional deficits may be difficult to access in the office but can result in significant relative debility in the high-demand setting of sport competition. Also, consideration of additional measures that may be taken to improve the performance or safety deficit from the impairment should help inform this determination. For example, even trace peroneal dysfunction and associated ankle eversion weakness may increase the likelihood of lateral ankle sprains. However, with ankle bracing or taping, this risk may be improved to an acceptable level to allow for a safe return to sport.

Also worrisome is the potential for reinjury given a return to the environment that likely precipitated the injury. Any allowable additional protective equipment or splinting that may reduce the likelihood of recurrent injury should be recommended despite scant evidence that commonly used devices significantly reduce the risk of reinjury. The predominant example of this is the use of cervical collars or neck rolls for "stinger" prevention, which has been shown to reduce excessive cervical hyperextension but not lateral flexion in laboratory testing [8] but lack any significant analysis during competition.

Similarly, a comprehensive evaluation of biomechanical factors that would have predisposed the injury should be addressed. This is seen frequently with baseball pitchers following ulnar nerve injury about the elbow. Often improved pitching mechanics can significantly reduce the vulnerability of the previously injured nerve without sacrificing performance. Although physical protection or activity modification is ideal to help facilitate a safe return to sport, occasionally avoidance of the factors that resulted in the initial injury is not feasible, and the treating physician must do their best to ensure optimal neural recovery before resumption of sport-specific activities.

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Vascular

39

Andrew McBride, Amy M. Singer, and Holly Beach

Key Points

- Vascular injuries can occur in isolation or concomitantly with nerve injuries; therefore, it is important to perform a thorough history and physical examination.
- In athletes, complete occlusive vascular injuries are less common than nonocclusive vascular injuries.
- If a pallor-like appearance is noted, a venous obstruction is more likely than an arterial obstruction.
- Cyanosis and edema are more specific signs of an arterial lesion.
- Duplex ultrasonography is the diagnostic test of choice in evaluation of DVT.
- Angiography is the gold standard for diagnosis of arterial disease.
- Referral to vascular surgery is warranted in the setting of acute vascular compromise or refractory vascular insufficiency.

Introduction

An athlete with a vascular injury may initially present following a traumatic injury with stereotypical symptoms including pulsatile bleeding, an expanding hematoma, cyanosis, and skin that is cool to the touch. However, it is not uncommon for them to present in the absence of trauma with an unremarkable physical examination. Pain, pallor, pulselessness, paresthesia, and paralysis are rarely all displayed [1]. A high index of suspicion is crucial, and further provocative testing may be needed to diagnose an underlying vascular injury [2]. Specific vascular diagnostic tests can be utilized when indicated.

A. McBride (✉)
Department of Family Medicine, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: andrew.mcbride@ucdenver.edu

A. M. Singer
Department of Family Medicine, Banner UMC, Tucson, AZ, USA
e-mail: amy.singer@bannerhealth.com

H. Beach
Department of Family and Community Medicine, Primary Care Sports Medicine, University of Arizona, Tucson, AZ, USA
e-mail: Holly.mcnulty@bannerhealth.com

Vascular Clinical and Diagnostic Tests

Clinical Vascular Tests

There are many clinical tests which have been historically performed for peripheral vascular diseases. These tests build off the standard capillary refill assessment and peripheral pulse palpation. Unfortunately, many of these tests have poor sensitivity and specificity. A few of these tests are summarized in Table 39.1.

Ankle Brachial Index (ABI)

ABI measures ratio of systolic pressure at the ankle (tibial artery) in comparison to the systolic pressure at the upper arm (brachial artery). Measurements are usually made pre- and post-exercise to assess for vascular causes of extremity pain. Patient with an ABI of <0.5 or side to side difference of >0.18 after 1 min of maximum exercise should have further arterial evaluation [1].

Angiography

Angiography is the gold standard for arterial disease diagnosis [1]. A radiopaque contrast material is injected into an artery to reveal vessel abnormalities on X-ray imaging. Digital subtraction angiography is an adaptation that can permit improved visualization of blood vessels in dense soft tissue or around bone.

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Table 39.1 Summary of a few common clinical tests performed for peripheral vascular diseases

Name of test	Anatomy assessed	Description of test	Positive findings	Sensitivity and specificity
Cervical quadrant test	Vertebral artery	It is performed with the patient supine while the head and neck of the patient are passively extended and side bent laterally. The patient's head is then passively rotated in the same direction as they are side bent and held in that position for 30 s [1]	Dizziness or nystagmus may indicate ipsilateral vertebral artery injury [1]	Very poor sensitivity (<10% for blood flow impedance) with specificity ranging from 60 to 90% [3]
Allen test	Radial and ulnar arteries	It is performed with both the radial and ulnar arteries being obstructed at the same time with external pressure applied by the examiner. Return of blood flow upon release of pressure is assessed in the ulnar artery by maintaining pressure on the radial artery and releasing pressure on the ulnar artery. In a patent unobstructed artery, blood flow should return quickly (5–15 s). The test is then repeated in the same fashion but for the radial artery [1]	If return of blood flow is delayed (usually >15 s) or absent, the artery may be occluded [1]	This test has a sensitivity of 55–70% and specificity of about 92% [4]
Adson maneuver	Subclavian artery/ thoracic outlet syndrome	This is a test for assessing subclavian artery/thoracic outlet syndrome. It is performed by palpating the radial pulse of the extremity of concern, while having the patient rotate his or her head toward the affected limb. The patient then actively extends their neck while their shoulder is passively extended and externally rotated. The patient then takes a deep breath with the pulse still being palpated [1]	Diminishing or loss of pulse is concerning for occlusion or compression of the subclavian artery [1]	The sensitivity ranges from 18 to 87% with a specificity around 94% [5]
Roos and Wright tests	Thoracic outlet syndrome	The Roos test is performed by having patient actively abduct (90°) and externally rotate (90°) his or her shoulder, while at the same time closing and opening their hands forming a fist [1]. The Wright test incorporates palpation of the radial pulse of the affected extremity [1]	Numbness, tingling, and/or pain in the hands are considered a positive Roos test [1]. A positive Wright test includes the above or absence of a palpable radial pulse in the testing position [1]	One study suggests the Roos test has a sensitivity of 84% with a specificity of 30% [6]. The Wright test has a sensitivity of 90% and a specificity of 29% (for symptom reproduction) and sensitivity of 70% and specificity of 53% for pulse abolition [7]
Halstead maneuver	Thoracic outlet syndrome	This test is performed with patient in sitting or standing position. The patient is then asked to retract their scapulae and depress their shoulders, while palpating the radial pulse on the affected extremity [3]	Numbness, tingling, and/or pain in the hands or absence of radial pulse are considered a positive test [3]	Unknown
Buerger test	Lower extremity arterial flow	It is performed with the patient laying supine, and their lower extremities are passively elevated at 45° of hip flexion. The lower extremities are then monitored for skin changes or collapse of more prominent veins. The test is further confirmed by having the patient move to a seated position with his or her legs hanging down [3]	Blanching of the foot or collapse of veins is considered a positive test. If there is a delay in return to normal skin color or filling of veins of longer than 60 s, the test is considered positive [3]	Unknown. However, patients with positive test usually have extensive peripheral vascular disease [8]
Homan's test	Lower extremity DVT	The test is performed with the patient in a seated or supine position with their knees flexed. The patient's distal foot is then forcibly dorsiflexed at the ankle [3]	Pain in the calf can be suggestive of a DVT [3]	This test has wide ranges of sensitivity (10–50%) and poor specificity (40–90%) [3, 8]

Advanced Imaging Angiography

Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) allow visualization of vascular structures and the surrounding anatomy simultaneously. Contrast is injected intravenous versus intra-arterial [1].

Duplex Ultrasonography

It utilizes sound waves to produce imaging of arteries and veins. It is the method of choice for initial evaluation for DVT.

Traumatic Peripheral Arterial and Venous Injuries

Initial Management

The initial evaluation of suspected vascular injury should begin with taking a focused history and a physical examination. The location of injury should be assessed for observation of the position in which the extremity is held, the presence of obvious deformity of a long bone or joint, the presence or absence of crepitus or an open wound, and the skin color compared to contralateral side along with capillary refill followed by a complete motor or sensory exam [9]. Direct pressure or a pressure dressing should be applied to the site of external bleeding if present. If these options have not controlled hemorrhage, a blood pressure cuff inflated to greater than the systolic blood pressure or a proximal tourniquet may be applied [9]. If distal pulses are not palpable or capillary refill is delayed, more severe trauma should be suspected. If Doppler ultrasound is available, it may be used to assess for distal flow [9]. If the trauma involves a long bone or joint and there is suspicion of compromised blood flow, immediate reduction and splinting or a traction device should be used [9]. Distal pulses should also be reassessed following these procedures to determine if flow is restored. Another consideration is the environment or field of play where the injury occurred. In order to best maintain an athlete's euthermic body temperature, the athlete should be assessed in an environment avoiding extremes of temperature if able.

Arterial injuries historically can have associated "hard" findings including pulsatile bleeding, loss of distal pulses, an audible bruit or palpable thrill, or an expanding or pulsatile hematoma [10]. The presence of these hard findings should lead to earlier emergent imaging and surgical evaluation [10]. Other "soft" findings include palpable yet diminished pulse compared to contralateral extremity, isolated peripheral nerve injury, unexplained hypotension, history of severe hemorrhage from site of injury, or large non-pulsatile hematoma [10]. These findings help to better delineate arterial from venous injuries which have a slower rate of blood loss and are often more occult injuries. Depending on the injury setting, type, and severity, direct pressure may not be sufficient for hemostasis. Physicians and advanced practitioners must be competent in suturing techniques in order to minimize blood loss in rare, life-threatening injuries.

Below we will discuss the evaluation and management of specific traumatic vascular injuries.

Axillary Artery Injury

Mechanism

The axillary artery can be traumatically injured in athletes with shoulder dislocations or reductions with the vessel becoming taut during shoulder abduction and external rotation [1]. The artery is then forced anteriorly against the pectoralis minor where the artery becomes damaged [1]. The artery can also be injured with repetitive forceful arm elevation and extension in overuse settings and multiple subluxations [10].

Epidemiology

Injuries to axillary vasculature (artery and vein) constitutes 3–9% of all vascular injuries [11]. Male athletes appear to be at greater risk for axillary artery injury than female athletes [10]. Baseball pitchers and volleyball players appear to be the most susceptible to atraumatic axillary artery injury due to repetitive forceful arm elevation and extension [10]. This mechanism of injury leads to intimal hyperplasia and stenosis which can lead to pseudoaneurysm or aneurysm formation potentially leading to acute thromboembolism or dissection [10]. In trauma of the general population, penetrating trauma predominates, while blunt trauma leading to proximal humerus fracture, shoulder dislocation, or reduction may be more frequently seen in athletes [11].

Clinical Presentation

The athlete with a suspected axillary artery injury will likely have an injury with either a fall or directed trauma onto the proximal humerus or a forced abduction and external rotation leading to anterior shoulder dislocation [12, 13]. The symptoms seen may range from arm fatigue and pain with shoulder exertion to digital pain [10]. On physical examination, the athlete may have a cool upper extremity with decreased or absent brachial, radial, or ulnar pulses [10]. If distal embolization has occurred, digital ulcerations may be seen [10].

Diagnosis

The combination of the above physical examination findings and suspicious mechanism of injury should lead to further diagnostic study. The gold standard for diagnosis of artery injury is catheter-directed angiography with chest and shoulder X-ray recommended for evaluation of bony trauma that could lead to arterial injury [10]. If occlusion of artery is suspected, elevation of arm during study should be performed if occlusion is not present with arm in neutral position [10].

Management

The definitive treatment for axillary artery injury involving arterial dissection or laceration is surgical repair [10]. In cases of acute arterial thrombosis, systemic anticoagulation is the initial treatment with stenting or bypass planning depending on the arterial lesion and degree of thrombosis [10].

Complications

Traumatic axillary artery injury can lead to distal extremity ischemia, necrosis, and morbidity if not diagnosed and treated appropriately. Mortality appears to occur only from concomitant injuries in multisystem trauma [14].

Brachial Artery Injury

Mechanism

Injury (laceration, occlusion, or thrombosis) of the brachial artery can occur with penetrating trauma, fracture of supracondylar humerus, or elbow dislocation [9]. It can also be injured in the closed reduction of a humeral fracture [9].

Epidemiology

Brachial artery injuries are the most common major vascular injury in the upper extremity in the general population [11]. Case report and series data have found brachial artery injuries to occur in up to 1.7% of elbow dislocations [11]. Injuries to the elbow that can predispose to brachial artery injury make up 9% of injuries in rock climbing and bouldering [15].

Clinical Presentation

Athletes with brachial artery injury may present after a fall onto outstretched hand or trauma directly to the elbow or humerus [9]. The athlete may have severe pain along with deformity of the elbow with possible pallor, mottling, or cyanosis. The radial pulse may be absent in as much as 75% of cases of brachial artery injury [11].

Diagnosis

The diagnosis of brachial artery injury involves physical examination with suspicious mechanism as above. Absence of radial pulse should increase suspicion of brachial artery injury. Plain radiography of the elbow should be performed given the mechanism of injury along with duplex ultrasonography regardless of vascular exam [15]. CT angiography should be used to definitively diagnose the degree of arterial injury in cases based off suspicious physical exam, mechanism of injury, or abnormal duplex ultrasonography.

Management

Management of brachial artery injury involves immediate splinting of extremity followed by duplex ultrasonography and angiography as described above. These arterial injuries should be surgically repaired in almost all cases due to high amputation rate with surgical ligation [11]. The median nerve should be assessed prior to repair given its proximity with the artery [11].

Complications

Limb salvage rates are near 100% due to prehospital advances, improved surgical repair, and earlier administration of antibiotics [11].

Effort Thrombosis (Paget-von Schroetter Syndrome)

Mechanism

Paget-von Schroetter syndrome is a rare but serious injury involving thrombosis of the axillary or subclavian veins after trauma or intense repetitive upper extremity activity [1]. The subsequent venous compression and thrombosis which occur with compression against the first rib may be acute or chronic [10].

Epidemiology

While rare in the general population, this syndrome is the most frequently encountered vascular issue in young competitive athletes [16]. The mean age at diagnosis is in the early 30s, and there appears to be a 2:1 male to female predilection [17]. This syndrome is most often seen in athletes with repetitive abduction of the shoulder, such as baseball pitchers, football players, tennis players, swimmers, and weight lifters [10].

Clinical Presentation

Presenting signs and symptoms may consist of dull pain in the affected extremity along with swelling and intact pulses if compression is chronic and low grade. If acute compression has occurred, there is usually a rapid onset of symptoms such as pain, cyanosis, and non-pitting edema [2]. Pulses should remain intact unless the thrombosis is massive or collateral vasculature has not developed [1].

Diagnosis

A suspicious physical examination and history in a young active athlete with unexplained upper extremity swelling or unexplained pulmonary embolism should prompt evaluation for this syndrome [10]. Duplex ultrasonography is the diagnostic modality of choice given its ease of use and availability [1]. This study has a sensitivity of 78–100% and a specificity of 82–100% in this syndrome [18]. A hypercoagulability workup is recommended that generally includes testing for factor V Leiden, lupus anticoagulant, and proteins C and S [1].

Management

Emergent thrombolysis and confirmatory venography are recommended in acute cases [1]. Temporary (about 8 weeks) anticoagulation therapy and partial venotomy is recommended to remove the stenosed portion [1]. Thoracic inlet decompression involving the costoclavicular junction is accepted as appropriate therapy to correct the underlying anatomic etiology [17]. An average of 3.5 months for return to play was found on a review [1].

Complications

Pulmonary embolism can occur in up to 36% of patients [19]. There is a higher rate of recurrent DVT than the general population in these athletes [1].

Vascular Thoracic Outlet Syndrome (V-TOS)

Mechanism

V-TOS usually results from compressive trauma or an osseous abnormality near the thoracic inlet which can lead to stenosis or thrombosis affecting the subclavian artery (this can also affect the subclavian vein or brachial plexus, and treatment may vary depending on the type of TOS) [20]. Key structures of the thoracic outlet include the scalene triangle (anterior, posterior, and middle scalene), costoclavicular space, and subcoracoid/retropectoralis minor space (Fig. 39.1) [1].

Epidemiology

This syndrome is commonly seen in athletes with repetitive throwing or abduction of the shoulder, such as baseball pitchers, tennis players, and weight lifters [10].

Clinical Presentation

Patients may complain of numbness or exhibit pallor, digital ulceration or gangrene, weak pulses, or a reduction in arm blood pressure with positional maneuvers [20].The patient may also have unilateral scalene tenderness to palpation or bruits on auscultation (less likely) of the supraclavicular fossa [1]. Additionally, an abnormality in the first rib or clavicle may be palpated.

Diagnosis

Provocative testing may be helpful (e.g., Roos, Wright, and Adson maneuvers) [2]. The gold standard for venous thoracic outlet syndrome is angiography. However, if this is not available, basic cervical or chest X-ray imaging may help identify bony abnormalities of the rib or clavicle, and duplex ultrasound can be effective in assessing for compression, thrombosis, or damage to the arterial wall [20].

Management

Vascular surgery evaluation and intervention may be warranted. However, nonsurgical treatment may be acceptable if there is no vascular compromise identified on imaging. If evidence of aneurysm is present, surgical repair is recommended to prevent distal embolization [20]. In most cases of chronic vascular thoracic outlet syndrome, conservative treatment is favored for 4–6 months prior to consideration of surgery [1]. This includes avoidance of overhead activities, postural reeducation, physical therapy, and pain control [1].

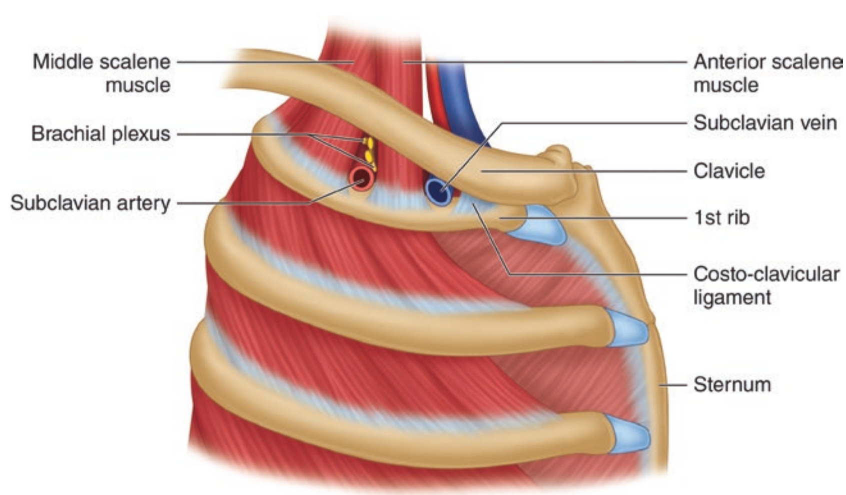

Fig. 39.1 Thoracic outlet anatomy

Complications

In the absence of a hypercoagulable state, athletes can often return to full sports activity following treatment of this syndrome [20].

Subclavian Steal Syndrome

Mechanism

This syndrome is the result of retrograde flow (usually down the ipsilateral vertebral artery) due to stenosis or occlusion of the subclavian artery. This can be caused by compressive trauma in the setting of high-intensity activity and/or atherosclerosis [2].

Epidemiology

The prevalence of subclavian steal syndrome in the general population is reported between 0.6% and 6.4% [21]. This is most common in males (2 to 1 predilection) over the age of 50 [21]. Upper extremity exertion in athletics can lead to this steal phenomenon through increased blood flow demand [21].

Clinical Presentation

The patient may present with presyncope/syncopal episodes, neurological deficits, arm weakness, paresthesia, loss of radial pulse, and claudication pain [2].

Diagnosis

Just as with thoracic outlet syndrome, provocative testing (e.g., repetitive activity on the affected extremity) may cause onset of symptoms described above. Angiography is the gold standard, but duplex US, MRA, or CTA can also be used. In addition to diagnostic testing, blood pressure discrepancy may be assessed in the office (more than 20 mmHg in affected arm) or appreciation of subclavian bruits [2].

Treatment

Vascular surgery evaluation/intervention with stenting or angioplasty is the main form of treatment. In more serious cases, arterial bypass may be warranted [2].

Quadrilateral Space Syndrome and Posterior Humeral Circumflex Artery Injury

Mechanism

Quadrilateral space syndrome can be seen in conjunction with an axillary nerve entrapment and posterior humeral circumflex artery injury after blunt shoulder trauma/injury (e.g., shoulder dislocation, humeral fracture) or following surgery [22]. In this injury, pressure exerted from bony fragments, expanding hematoma, fibrous bands, or hypertrophied muscles causes occlusion of the posterior humeral circumflex artery (Fig. 39.2) [2].

Epidemiology

This rare condition occurs most commonly in athletes 20–40 years of age who participate in contact (leading to fracture or dislocation) or overhead/throwing sports such as volleyball [22].

Clinical Presentation

An athlete may have signs and symptoms including pain, cramping, weakness, tingling, coolness to touch, pallor, or cyanosis of the affected extremity following mechanism of injury as described above [22]. They may have had a history of previous intermittent pain in the shoulder with activities.

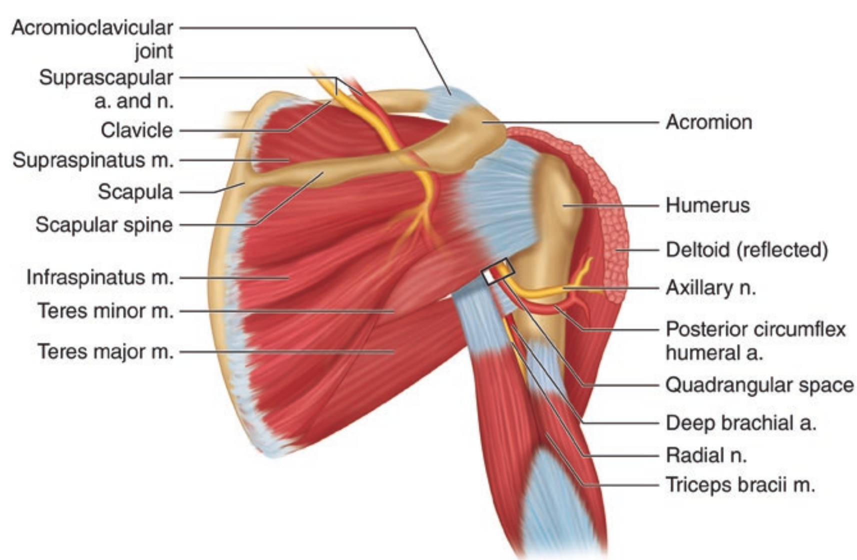

Fig. 39.2 Quadrilateral space anatomy

Forced abduction and external rotation of the shoulder may exacerbate symptoms [22].

Diagnosis

The patient with any of the above presentations should be evaluated with a thorough physical examination with distal neurovascular assessment. An MRI can be used to evaluate the quadrilateral space and determine chronicity of symptoms (presence of isolated teres minor atrophy) in atraumatic cases [22]. In acute trauma, plain radiography of the shoulder should be performed to evaluate for compression by bony elements, and arteriography should be considered in suspected posterior humeral circumflex artery injury following trauma [22]. Distal embolization can occur with arterial thrombosis, and the presence of digital ulceration or reduced vascular flow on physical examination should prompt arteriography [23].

Management

Thrombolysis or surgical thrombectomy should be considered in cases of arterial thrombosis with or without distal embolization [2]. Following this initial management, a rehabilitation program with early ROM (avoiding overhead movements) is suggested with a 6-week return to full ROM and 3–4 months before full activity can be started [23].

Complications

The complications associated with this syndrome and vascular injury involve distal embolic phenomenon that can be avoided or minimized with early diagnosis and management.

Palmar Arch Injury and Hypothenar Hammer Syndrome

Mechanism

Palmar arch injury occurs as a result of high-impact trauma in sports such as cricket, baseball, and handball leading to ulnar arterial thrombosis or aneurysm formation [10]. Hypothenar hammer syndrome, an example of chronic palmar arch injury, can occur as a result of repetitive trauma to the hypothenar area of the hand leading to ulnar arterial damage where it overlies the hamate bone (Fig. 39.3).

Epidemiology

This injury tends to present in sports with repetitive and sometimes high-impact trauma to the hand. Cricket, baseball, and handball players are at risk of acute palmar arch injury [10]. Baseball players, mountain bikers, and golfers are at higher risk for hypothenar hammer syndrome [24].

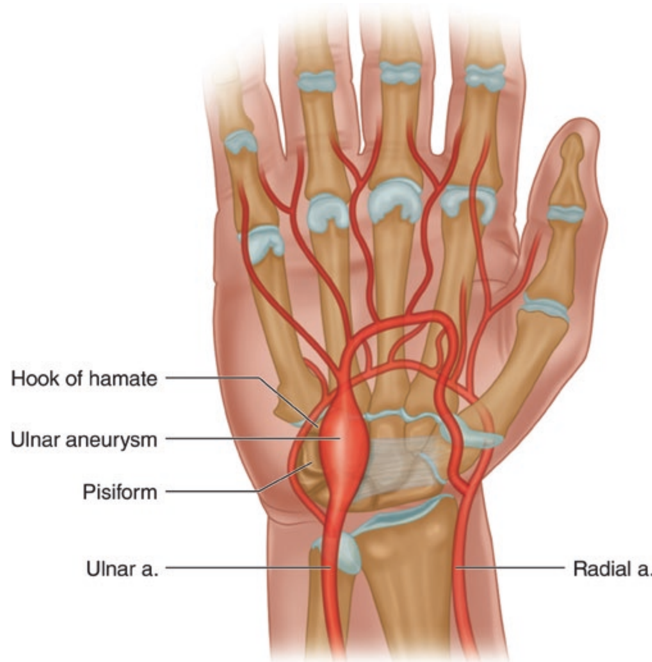

Fig. 39.3 Palmar arch anatomy with an aneurysm

Clinical Presentation

An athlete with palmar arch injury will likely have suffered from direct trauma from a ball or baseball/cricket bat causing direct trauma to the palmar arch [10]. The injury is usually unilateral in nature and symptoms may include pain, paresthesias, temperature sensitivity, claudication, or discoloration of fingers/fingertips [1]. Patients may also present with a pulsatile mass, splinter hemorrhages, ulcerations, or signs of gangrene [2].

Diagnosis

An athlete with the above presentation and physical examination findings along with a positive Allen's test is concerning for palmar arch and ulnar artery injury [10]. Angiography is the gold standard for diagnosis; however, CTA may be used to evaluate for hamate fracture, and MRA can be used to assess for ulnar artery trauma [2].

Management

Conservative treatment is reasonable most of the time, except in cases of ischemia or ulceration where surgical intervention may be required [10]. Conservative treatment includes avoidance of provocative activities, trial of calcium channel blockers, antiplatelet agents, pentoxifylline, smoking cessation, activity modification/avoidance of repetitive activity, wearing padded gloves, and avoiding the cold [10].

Complications

Complications can include digital ischemia and ulceration in cases of thrombosis or aneurysm formation [10]. A thorough

physical examination and appropriate angiography can prevent this complication.

Acute Compartment Syndrome

Mechanism

Acute compartment syndrome (ACS) typically results from a fracture, severe blunt trauma from a high-energy mechanism, tendon (Fig. 11.4) or vascular injuries [25]. This trauma leads to increased intracompartmental pressure that, if untreated, leads to tissue ischemia, necrosis, and permanent disability [26].

Epidemiology

The average annual incidence of ACS is estimated from 1 to 7.3 per 100,000 [25]. Men are ten times more likely to suffer ACS than women [25]. The mean age for males is 30 years while it is 44 years in females [25]. The leg and forearm are most frequently affected [25]. Over two-thirds of cases are associated with an underlying fracture [26]. This syndrome has been reported in a wide variety of sports including handball, soccer, rugby, cycling, kick boxing, karate, lacrosse, baseball, and football [1].

Clinical Presentation

Patients who have suffered extremity trauma can develop swelling and pallor of the extremity. Severe disproportionate pain is also present with the later development of paresthesias and tingling. Irreversible damage may occur as early as 12 hours post-onset and pain may be absent in late ACS [25].

Diagnosis

The diagnosis of ACS is clinical at this time focusing on the associated five “Ps”: pain, paresthesia, pallor, paralysis, and (high intracompartment) pressure [26]. While the “five Ps” have been described, they rarely are all present. A compartment pressure higher than 30 mmHg is indicative of compartment syndrome with a normal resting compartment pressure being accepted as 10 mmHg [26]. There are variable surgical indications, and current research is focusing on more objective methods of diagnosis.

Management

Initial treatment involves removal of any tight-fitting splint, cast, or clothing over the area, while the limb should be maintained at heart level. The athlete should be transported to an emergency room for close monitoring and surgical consult for possible fasciotomy (Fig. 11.4). The pressure threshold for fasciotomy is debated although a threshold of 30 mmHg (20 mmHg in hypotensive patients) is proposed by previous studies [26]. More recent research has shown the use of delta pressure (ΔP = diastolic pressure – intracompartmental pressure) of $\leq$ compartmental diagnostic threshold for fasciotomy [26].

Complications

Delay in diagnosis and fascial release can lead to irreversible necrosis, deformity, and mortality. There is a reported mortality rate of 47% after acute compartment syndrome of the thigh [25].

Popliteal Artery Injury

Mechanism

Injury to the popliteal artery is most commonly caused by posterior knee dislocation as bone fragments directly lacerate or cause thrombosis of the artery [11]. This type of dislocation occurs during knee flexion with a posteriorly directed force onto the tibia [27]. Injury can also be caused by displaced fractures in the knee as seen with lateral tibial plateau fractures [11]. Sports injuries are often moderate energy and are less likely to lead to arterial trauma than high-energy (often involving motor vehicles) mechanisms [28].

Epidemiology

Popliteal artery injury occurs in 25–33% of knee dislocations [11]. However, the injury may be clinically occult in up to 40% of cases requiring high index of suspicion in dislocation with spontaneous reduction (may occur in up to 50% of cases) and multiligament knee injuries [11]. This multiligamentous injury, which more often occurs as the result of a high-energy trauma, can be associated with multiple other traumatic injuries and can cause disruption to the neurovasculature resulting in inadequate perfusion leading to muscle and tissue necrosis within hours [28]. These traumatic injuries are more likely in contact sports such as football, soccer, and rugby [27].

Clinical Presentation

Popliteal artery injury should be suspected in an athlete complaining of severe knee pain or instability following a mechanism concerning for dislocation or fracture [27]. There may be deformity of the limb, or the appearance may be more occult in cases of spontaneous reduction. The distal extremity may be cool, pale, cyanotic, and mottled and may have delayed capillary refill [28]. The “hard” findings of arterial injury as described above may be present and warrant emergent vascular imaging.

Diagnosis

Immediate neurovascular physical examination should be performed in cases of knee dislocation. This involves palpation of pedal pulses, sensation, and peroneal nerve testing as the peroneal nerve can be injured in up to one-third of dislocations [27]. While there is no consensus on diagnostic approach to detect popliteal artery injury following suspected knee dislocation, ABI and duplex ultrasonography are noninvasive imaging options that are often employed prior to

angiography [11]. The gold standard of diagnosis of vascular injury is CT angiography, and most centers use this in selected cases (equivocal or suspicious noninvasive testing, mechanism of injury, or physical exam) or when "hard" findings of vascular injury are present [11].

Management

Advanced Trauma Life Support protocols should be followed in patients with high-velocity injury and concomitant life-threatening injuries [28]. The initial management in athletes with lower-energy trauma is to splint the knee in extension or most comfortable position with emergent hospital transport [27]. An attempt to reduce the dislocation should be immediately performed followed by neurovascular reassessment [27]. This can be performed on the sideline, locker room, or emergency department. The above studies should be performed to assess for degree of vascular injury followed by appropriate surgical management.

Complications

Rapid identification of arterial injury is vital as a delay in ischemia treatment of only 8 h nearly always results in amputation [28]. Amputation rate in patients with knee dislocation and popliteal artery injury can be as high as 20% [29].

External Iliac Artery Endofibrosis (EIAE)

Mechanism

EIAE is a condition caused by a combination of mechanical and shear stress and results in a subsequent progressive fibrosis which ultimately can lead to stenosis. Arterial kinking alone can be a cause of EIAE. Common aggravating factor is the repetitive hip flexion endured in the setting of high blood flow to the affected area during exertion [2]. Acute on chronic conditions has been reported [30].

Epidemiology

EIAE is a rare condition most commonly found in young competitive cyclists and triathletes without significant risk factors for atherosclerosis [7]. EIAE can happen among cross-country skiers, endurance runners, weight lifters, speed skaters, and rugby players.

Clinical Presentation

Patients can have unilateral limb pain or weakness during high-intensity activity. Swelling and paresthesias may also be noted. Worsening of symptoms may occur over the duration of activity.

Diagnosis

Angiography is the standard diagnostic modality. ABI, duplex US, or MRA may also be considered. Testing with US and ABI may be improved if completed pre- and post-exertion.

Management

It can range from conservative management to surgical interventions. Conservative treatment mostly involves activity modification. Medical therapy with antiplatelet agents and/or statin has not proven to be affective in treatment [2]. Surgical intervention primarily involves percutaneous transluminal balloon angioplasty. If there is only arterial kinking without stenosis, a surgical release of arterial branches to the hip flexors may be completed. More definitive treatment consists of arterial resection and vein grafting or endarterectomy and vein patch angioplasty [2].

Return to Play

Return-to-play guidelines after vascular injuries in sports are variable and depend on whether conservative or surgical treatment was necessary. In most cases, relative rest and gradual return to activity should suffice with or without anticoagulation. In addition, a good sports-specific rehabilitation program is key to return to function and sports-specific activity. In cases where immediate surgical intervention is warranted, the return-to-play timeline is sports specific and may vary depending on the presence of operative or postoperative complications and the area affected.

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Check for updates

Skin and Subcutaneous Tissue

40

Rajwinder S. Deu and Morteza Khodaee

Key Points
- Most lacerations should be repaired within 8 h except for facial lacerations which can be repaired up to 24 h after injury.
- Abrasions are superficial injuries to the skin due to friction or a scrape.
- Aspiration of a Morel-Lavallée lesion has shown quicker return to play in athletes.
- Methods to protect against sunburn include the use of sunscreen, avoidance of direct sunlight, and wearing protective clothing including a hat.
- Refreezing must be avoided in cases of frostbite.

Introduction

Skin consists of three layers: epidermis, dermis, and subcutaneous fat. The epidermis is the outer layer of skin that prevents bacteria, viruses, and other foreign substances from entering the body. It also gives skin its cosmetic appearance. The dermis is the next layer and contains nerve endings, sweat and oil glands, hair follicles, and blood vessels. It is the key layer for healing of wounds. The subcutaneous fat primarily provides protective padding and insulation.

In many sports, an athlete's skin is left unprotected and vulnerable to injury secondary to trauma, mechanical friction, pressure, and the environment. As a result, management of skin trauma is a common occurrence for a sideline provider. Many times, this occurs during a game, making prompt diagnosis and management necessary to return the athlete to play. Specifically, a decision needs to be made on whether the wound can be treated temporarily to allow immediate return or needs definitive repair causing removal from competition.

R. S. Deu
Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: rdeu1@jhmi.edu

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

Traumatic

Lacerations

Lacerations refer to full-thickness disruption of the epidermis and dermis secondary to trauma. They generally occur when exposed skin contacts an opponent or the playing surface. Various mechanisms include direct trauma (an elbow in basketball or fist in boxing), equipment (loose-fitting helmet, stick, or ball), and the ground (road or trail in bicycling) (Fig. 40.1). There are approximately 6–7 million traumatic lacerations treated in the emergency department each year [1–3]. For facial injuries, published data has shown 3–29% are due to sports participation with most injuries (60–90%) occurring in males aged 10–29 years [4]. Preparation is key and providers must ensure that their medical bag is well-stocked (Table 40.1).

Evaluation of the athlete starts with determination of time, location, and mechanism of injury [6–8]. Time is important as the literature recommends wound closure for most injuries to be done within 8–12 h [5, 7, 8]. Facial lacerations can be closed up to 24 h after injury [5, 7, 8]. Mechanism (sharp vs blunt) and location can help predict the risk of contamination or presence of a foreign body [6–8]. In addition, the mechanism allows one to determine if there is any other associated injury such as fracture, nerve, or tendon injury. Lastly, tetanus immunization status should be confirmed.

The first step in treatment is to control the bleeding which is typically performed by holding direct pressure. The pro-

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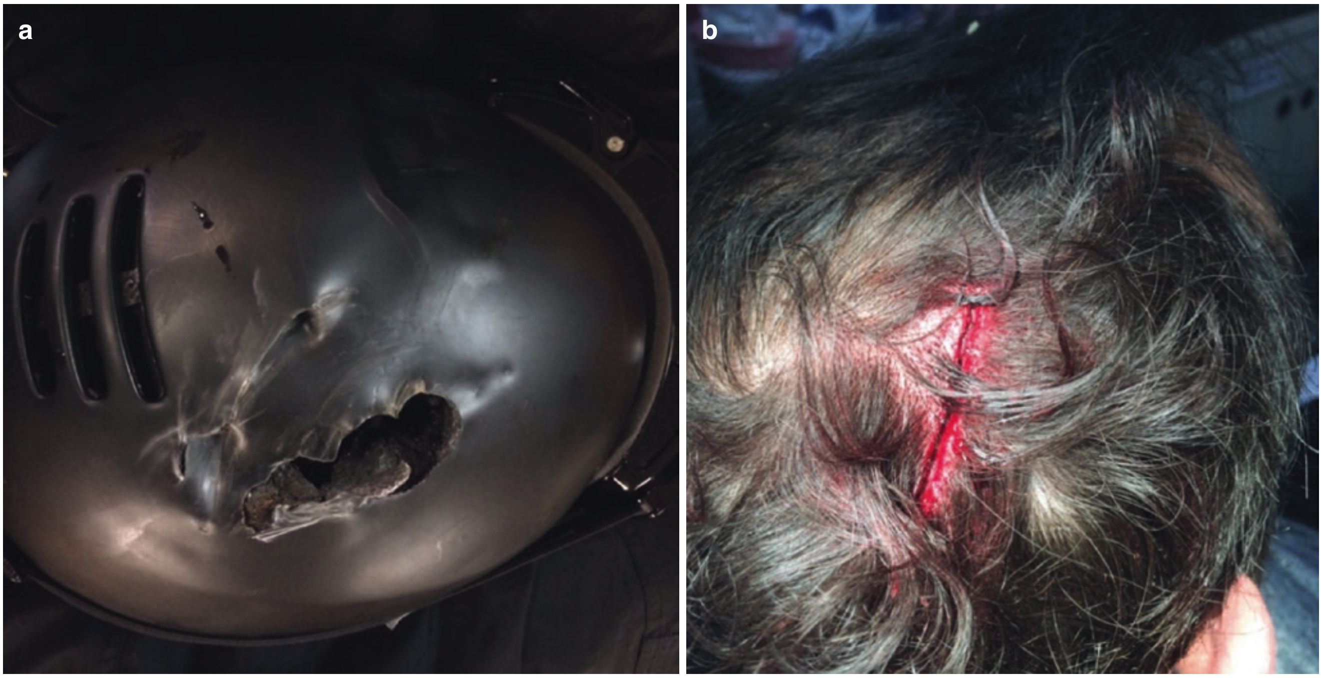

Fig. 40.1 Scalp laceration. Tree branch pierced through ski helmet causing laceration

Table 40.1 Medical bag [5–7]

Disposable suture kit	Suture
Sterile fields/drapes	Anesthetic
Antimicrobial cleaning agents	Needles
Syringes (including 60 mL for irrigation)	Skin stapler
Skin adhesive	Surgical tape/strips
Dressings	Protective skin covering
Headlamp	Sunscreen
Informed consent sheets	Aftercare instructions

vider should follow universal precautions with use of gloves and other available barriers. After hemostasis is achieved, a quick decision needs to be made regarding the urgency of treatment and whether the area can be taped or patched to allow the athlete to return. Once the athlete is ready for repair of the laceration, a local anesthetic block should be performed. Anesthetics using epinephrine can provide increased hemostasis but should not be used in areas where blood supply is limited (nose, fingers, penis, toes). Topical anesthetics may offer an effective, noninvasive approach to analgesia prior to suturing [9]. Lidocaine-epinephrine-tetracaine gel has been found to provide effective analgesia in children for treatment of simple lacerations [10, 11]. Early anesthesia will allow for more aggressive irrigation, inspection for foreign body and trauma to deeper structures, and test for nerve and tendon function (Fig. 40.2). All wounds should be considered contaminated, so irrigation is vital to decreasing the risk of infection [7]. Irrigation with normal saline (tap water if saline is not available) particularly with a protective cup is recommended.

Fig. 40.2 Partial extensor tendon rupture of the second finger in a 28-year-old female after her finger was cut by her snowboard. This could have been missed, if the adequate exploration was not performed

Several different methods have been used to close skin including sutures, surgical taping, staples, and tissue adhesives. The main goal of laceration repair is to approximate the tissue to achieve a good cosmetic result. In so doing, one

Table 40.2 Suture selection for body region and follow-up [5, 6, 13]

Body region	Superficial lacerations (monofilament, nonabsorbable)	Deep lacerations (monofilament or braided, absorbable)	Days to removal
Scalp	5-0 or 4-0	4-0	7
Face	6-0 or 5-0	5-0	3–5
Lips	5-0 (outer portion of lip/vermilion border/skin) 4-0/3-0 mucous membranes	4-0	
Eyelid	7-0 or 6-0	Not recommended	3–5
Eyebrow	6-0 or 5-0	5-0	3–5
Trunk	5-0 (low tension)	3-0 or 4-0	7–10 (low tension)
	4-0 (high tension)		10–14 (high tension)
Extremities	5-0 or 4-0	4-0	7–10
Joints	4-0	4-0	10–14
Hand	5-0, 4-0 (palms)	5-0	7–10
Foot	4-0, 3-0 (sole)	4-0	10–14

strives to provide hemostasis, prevent infection, and eliminate dead space.

Suturing is the most dependable method for laceration repair. Selecting the appropriate suture material, size, and technique are critical for successful repair and cosmetic result [5]. This is dependent on the size, depth, and location of the wound along with the supplies that are available. Skin tension differs in different body regions necessitating use of various suture sizes (Table 40.2). The two main categories of suture are absorbable and nonabsorbable. Absorbable suture is used to eliminate dead space in deep wounds, while nonabsorbable suture is for skin closure. The synthetic polymers are typically used secondary to higher tensile strength, less tissue reactivity, and lower rates of infection [5, 12].

The skin can be approximated using a variety of techniques. A simple interrupted suture offers redundancy, maintaining the wound edge should one of the stitches break (Fig. 40.3). Running sutures can be performed quickly and distribute the tension evenly. It is at risk of becoming loose if one of the knots break. Subcuticular running sutures can be used in lacerations with low tension. It offers improved cosmetics but can separate if too much pressure is placed on the wound. Tapes or glues can be used to supplement this stitch [8]. Mattress sutures, vertical and horizontal, are used in areas of higher skin tension particularly when wound edges must be brought together over distance [13]. They promote skin edge eversion which limits scarring. Vertical mattress sutures are commonly used in wounds that occur on a concave surface due to the tendency of its edges to invert [13]. Horizontal mattress suture is commonly used for pulling wound edges over distance [13]. They are useful on palms and soles [8].

Surgical taping can be performed for repair of very small, superficial wounds that are not under tension. These are ideal for children as the pain is minimal. Most often, it is used after suture or staple placement as an adjunct to reinforce the repair or after suture removal to prevent dehiscence.

Skin stapling can be an alternative to suturing particularly in areas where there is low tension and excellent cosmetic result is not required. It is quicker and provides less tissue reaction than suturing. It is ideal for long, linear wounds of the scalp, proximal extremities, or torso [14]. It should be avoided in facial or neck tissue, small mobile joints, wounds that are macerated or infected, or areas of large tissue loss [14]. The technique starts similarly as to suturing with anesthesia, irrigation, and exploration of the wound. It can be performed as a lone operator or with an assistant. The operator starts at one end of the wound, approximating the skin edges with forceps, lines the stapler arrow over the incision, and presses down on the trigger to engage the staple. This is repeated until the wound is closed. Typically, staples will stay in place for 8–10 days and can be removed with a staple remover.

Tissue adhesives offer multiple benefits; the most important in the athletic setting is quick application allowing faster return to play. They can be used for wounds under low tension as an alternative to 5-0 or smaller suture, most commonly on the face, scalp, torso, or extremity wounds [5, 15]. Treatment with an adhesive is faster and less painful and does not require dressing changes or need to be removed. Due to these factors, it is an excellent choice for children. Studies have shown that it can be as effective as suturing in appropriate patients [16–19]. Initial wound irrigation is still needed but the wound must be dry and bloodless prior to application. The wound edges are approximated and typically three coats of adhesive are applied. Care should be made to not get any of the adhesive into the wound as it can cause a tissue reaction and delay healing. Patients may shower immediately but should not bathe or swim for 2–3 days [5]. The adhesive typically sloughs off spontaneously within 5–10 days [3].

The wound should be kept dry and inspected daily for signs of infection (redness, swelling, warmth, tenderness, or drainage). Patients with sutures or staples may gently cleanse the area and pat dry [5]. Facial sutures should be removed in about 3–5 days, scalp and extremity wounds in 7–8 days, and 10–14 days for wounds under higher tension such as those over mobile joints, soles, palms, or back (Table 40.2) [5, 7]. Typically, adhesive strips are applied after suture removal to provide initial support while the wound continues to heal. Table 40.3 summarizes different laceration treatment options. Table 40.4 summarizes the level of evidence for key clinical recommendations for management of lacerations.

Return to play is dependent on multiple factors including wound severity, type of sport, wound contamination, and risk of contamination to others [7]. The NCAA requires that in an injured athlete, bleeding must be stopped and the open

Fig. 40.3 Good approximation of three different lacerations by sutures. A simple linear laceration (**a, b**), a triangular laceration (**c, d**), and a linear eyebrow lacreation (**e, f**)

Table 40.3 Summary of laceration treatment options [3, 14, 15]

	Sutures	Staples	Adhesives
Indications	Most lacerations	Wounds under low tension and where excellent cosmetic result is not needed Ideal for long, linear wounds of the scalp, proximal extremities, and torso	Linear wounds under low tension Wounds <8 cm Fragile skin Under splints/casts
Contraindications	Infected or heavily contaminated wounds	Infected or heavily contaminated wounds Facial and neck tissue Over small mobile joints (hand and foot) Wounds that are macerated or infected	Infected or heavily contaminated wounds Wounds under high tension
Advantage	Provides superior cosmetic result Excellent tensile strength Low dehiscence rate	Fast Everts wound edges better and places less tension on edges as compared to suturing Less reactive than sutures leading to less inflammation and infection	Fastest method Less painful Does not require dressing changes No need for removal Possible lower rate of infection
Disadvantage	Takes longer Painful May leave suture marks Most reactive Requires removal	Painful Potential to provide inferior cosmetic result Leave larger puncture scars More expensive Requires removal	Lower tensile strength Susceptible to moisture More expensive

Table 40.4 SORT: key clinical recommendations

Clinical recommendation	Evidence rating
Directly applying topical anesthetics to the skin is an effective, noninvasive way of providing pain control during suturing or stapling of skin lacerations [9]	B
Tissue adhesives are an acceptable alternative to standard wound closure for repairing simple traumatic lacerations [17]	C

wound needs to be covered and dressed prior to return to participation [20]. For superficial lacerations, closure with skin tape and/or dressing will allow an athlete to return to activity. However, if the laceration is deeper, the provider must decide whether the athlete needs to be pulled from activity to provide definitive care. This becomes complicated in sports that do not allow for multiple player substitutions. Ultimately the provider must consider the risks of infection, bleeding, extension of the wound, and associated injury to the athlete prior to return [6].

Possible complications from repair of a laceration include wound infection, early wound dehiscence, and missed nerve or tendon injury. Irrigation of the wound is extremely important in decreasing the bacterial count in the wound. Infection rate is increased in those with diabetes, a contaminated wound, presence of foreign body, length greater than 5 cm, and location on the lower extremity [1, 2]. In general, prophylaxis with antibiotics is not recommended. Missing a nerve or tendon injury can lead to long-term problems with function. So, exploration of the wound under good light is very important (Fig. 40.4).

Friction Injuries

Chafing and Abrasions

Chafing is a superficial inflammatory dermatitis as a result of skin rubbing against skin or clothing. It is a painful, inflamed oozing lesion. Chafing can occur anywhere on the body, but the thighs, groin, axilla, and nipples (runner's nipples) are the most common locations. Chafing is common in endurance and ultra-endurance athletes.

Abrasions are superficial (no deeper than the epidermis) injuries to the skin that are often due to friction or a scrape caused by a fall. It is more severe than chafing; however bleeding, if present, is minimal (Fig. 40.5). Abrasion is common in sports with high risk of falls or trauma such as cycling, commonly referred to as road rash. Football players are susceptible to turf burn while wrestlers can get a mat burn.

Chafing and abrasions can be treated by cleaning with soap and water and drying the areas. Applying a drying powder, petroleum jelly, aquaphor, topical antibiotic, or topical steroids also may alleviate the pain. Wearing dry, well-fitting clothes are important for prevention of chafing [21]. Placing tape over the nipples may reduce short-term symptoms during a race by reducing friction. Risk of infection in healthy athletic population is very small.

Blisters

Blisters are the result of constant friction of the skin. Depending on the specific sport, blisters can happen in multiple anatomical locations. Palmar blisters are common in gymnasts. Foot blisters (bulla) are very common among marathon and ultramarathon runners. Blisters are the most common reported injuries in marathons with the incidence of 0.2–39% [21]. The most common locations include the distal aspects of the toes, the balls of the feet, and the posterior heel (Fig. 40.6).

Fig. 40.4 Exploration under adequate light and use of Q-tips revealed an extension of the laceration into the fascia and muscular layers in a 60-year-old skier as a result of ski blade accident (**a**, **b**). The laceration was closed in 3 separate layers (**c**, **d**)

Fig. 40.5 Abrasion as a result of a fall during a mountain bike race

Fig. 40.6 Foot blister in a finisher of an ultramarathon runner

Risk factors for the formation of blisters include heat, moisture, ill-fitting shoes, and increased reparative activity (e.g., running volume) [21]. Painful blisters can be punctured with a sharp sterile instrument, while trying to keep the blister roof intact. Preventive measures include wearing appropriate footwear, gloves, taping, moisture wicking synthetic socks, and dry socks. Applying drying powder or petroleum jelly also may help. Colloid-type dressings, such as DuoDERM, may minimize blister pain and help athletes complete long events.

Morel-Lavallée Lesion

A Morel-Lavallée lesion (MLL) in sports is typically caused by shearing forces due to a fall, and direct contact with the field or surface can cause separation of the superficial fascia from the underlying deep fascia. As a result, the potential space between these tissues is subsequently filled with serosanguinous fluid, blood, and necrotic fat [22, 23]. This fluid is susceptible to infection.

Patients with a MLL present with a palpable fluctuant mass that can be painful and hypoesthesia over the area due to shearing of cutaneous nerves during the initial injury. Depending on the site of injury, athletes may complain of

limited mobility. MLLs have been reported in various parts of the body including the greater trochanter/hip (30.4%), thigh (20.1%), pelvis (18.6%), and knee (15.7%) [24].

Diagnosis is made through history and physical exam. Standard radiographs are obtained initially to rule out bony involvement. Ultrasound, if available, can provide both diagnostic and therapeutic modalities. Imaging will show hypoechoic to anechoic fluid collections between the deep fat and the overlying fascia [24–28]. Acute and subacute lesions are usually heterogeneous with irregular margins and lobular shape, while chronic lesions are homogeneous with smooth margins and a flat or fusiform shape [22, 26, 28]. Magnetic resonance imaging (MRI) is considered the modality of choice due to its excellent soft tissue contrast. It not only identifies the lesion, but it also allows for determination of lesion characteristics and chronicity through the age and amount of blood, fat, and lymph tissue within the MLL [22, 23, 26, 27, 29–32]. Early diagnosis is significant because neglected lesions can result in recurrent fluid accumulation, infection, and skin necrosis [22, 28, 32, 33].

Treatment of acute ML lesions includes aspiration and compression. Aspiration has shown quicker return to play for athletes [34]. One proposed treatment algorithm recommends observation and compression bandages for small, asymptomatic lesions less than 50 mL and percutaneous aspiration for symptomatic lesions less than 50 mL without overlying skin changes [35]. Large and chronic lesions recur without surgical intervention [23]. Recent literature suggests that a minimally invasive approach through small incisions demonstrates superior outcomes [36]. Percutaneous treatment has allowed for a rapid return to sport in the appropriate patient [37, 38]. Adjuncts that may be helpful to surgical debridement includes sclerodesis and use of a drain [23].

Return to play is dependent on location of the lesion and its associated symptoms. There is no contraindication if range of motion remains intact and pain is well controlled.

Complications of a MLL can include infection, fluid reaccumulation, and neurovascular compromise leading to dermal necrosis, compartment syndrome, and tissue necrosis. It is common for the soft tissue to have long-term bulging or scar tissue formation [39–41].

Pressure Injuries

Subungual Hematoma

Subungual hematoma occurs in 2.5% of marathon runners [21]. The etiology is repetitive, forceful impact of the longest toe (first or second) into the toebox, especially during downhill running. It can also happen as a result of a direct trauma from being stepped on by another player in contact sports like football and soccer. Repetitive forces and terrain demands in ultramarathons may increase risk of hematoma. Treatment is usually conservative; however, painful hematomas can be drained using a sterile needle [21, 42, 43] (Fig. 40.7). Risk of infection is very low. With or without evacuation of the hematoma, the toe nail may fall off in 1–2 months. Return to sport is based on athletes' symptoms.

Environmental Injuries

Sunburn

Sunburn is common in outdoor sports and is caused by the sun's ultraviolet radiation especially during midday. Sunburn is a significant risk factor for further heat-related

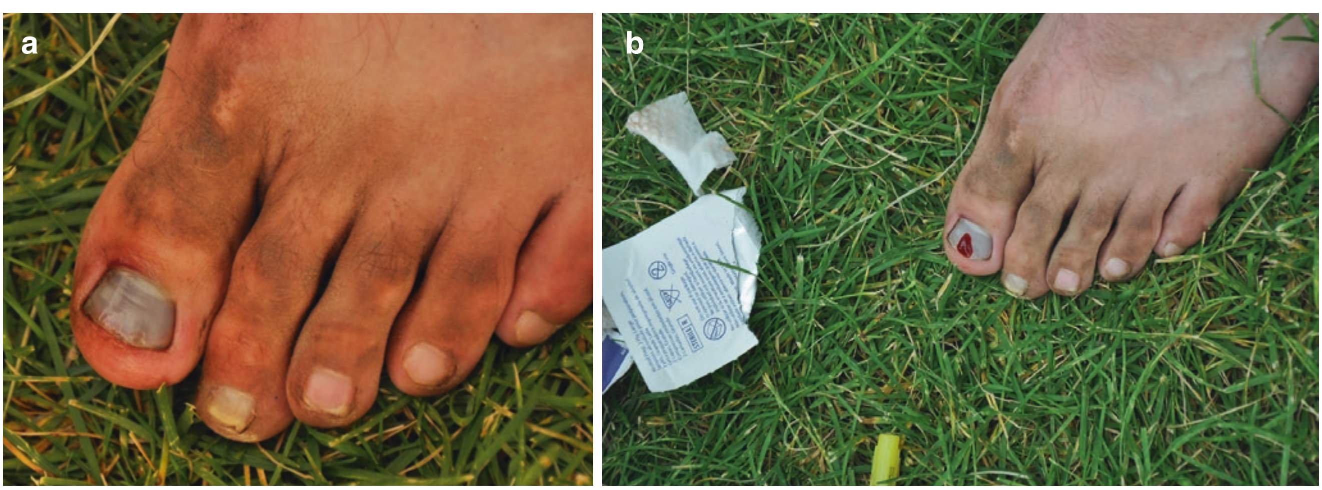

Fig. 40.7 Subungual hematoma of the right big toe in a 10-mile ultramarathon finisher (**a**). The hematoma was evacuated using a sterile 25-gauge needle (**b**)

illness. It is more common in people with fair skin. High altitude increases the ultraviolet intensity, so participants of events at high altitude should be aware of this risk [43]. Frequent use of a broad-spectrum sunscreen (preferably sweat-resistant sunscreen) with SPF of at least 30, strategically avoiding sun (e.g., running in the shade), and wearing protective clothing and a hat if possible are recommended [44]. Reapplication of sunscreen after significant sweating or rain exposure is important to prevent significant burns. An athlete with sunburn should be removed from direct sunlight with treatment involving use of cold compress or rinsing/bathing the area with cool water [6]. Mild sunburns can be treated with aloe vera, while more severe burns should receive silver sulfadiazine creams [6]. The presence of blisters, swelling, and exudates signifies increased severity and should be treated as direct contact burns [6].

Frostnip

Frostnip is the result of superficial cold-induced vasoconstriction in the skin usually occurring at freezing temperatures. As discussed earlier, the skin is commonly exposed during many sporting events. The superficial layers of the skin crystalize in the cold or wind, resulting in skin blanching and numb patches mostly in the nose, ears, chin, and cheeks. Since the tissue does not freeze, immediate rewarming can prevent tissue loss and long-term damage [45]. Frostnip is reversible and resolves rapidly with gentle rewarming without any sequelae, but it warns of risk of frostbite, and if conditions are ignored, significant injury may occur [44].

Frostbite

Frostbite is a freezing cold injury (FCI) to the skin and underlying tissues with formation of crystals in the extracellular space between cells (Fig. 40.8) [44, 45]. Frostbite occurs at a temperature below 0 °C (32 °F). Exposed areas (nose, ears, cheeks, and digits) are affected usually with more than half of the cases occurring in the toes [44]. Constrictive and wet clothes increase the risk of frostbite. A numb extremity in cold temperature should signal risk of frostbite and invite rapid rewarming. Care should be made not to rewarm the area through rubbing as it increases the risk of further tissue damage. The best treatment approach is rapid rewarming with warm water (37–40 °C) [44, 45]. Refreezing must be avoided as it can lead to vasoconstriction and further cellular injury typically leading to tissue death. Exercise on a frostbitten or rewarmed extremity increases risk of poor outcome.

Fig. 40.8 Second-degree frostbite

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Part VII

Other Sports-Related Trauma

Anna L. Watrerbrook

Chest Trauma

41

Jeffrey P. Feden

Key Points

- The spectrum of injury in blunt thoracic trauma varies widely, but athletic injuries are usually mild due to lower-energy mechanisms.
- Complications of thoracic trauma can be life-threatening and warrant a careful, structured approach to evaluation.
- Chest radiographs are the initial diagnostic imaging modality of choice, but computed tomography (CT) has greater sensitivity for many thoracic injuries.
- Established guidelines for returning to play following blunt thoracic or cardiac injury are lacking.

Introduction

The thorax is comprised of the thoracic wall and the thoracic cavity. The thoracic wall includes bony, cartilaginous, and muscular structures that protect the thoracic organs and other vital structures, such as the trachea and great vessels. The shoulder girdle, spine, and ribs provide much of the support and protection for the thoracic cavity. Injuries to the chest wall are common in sports. While life-threatening injuries to the thoracic organs and vital structures do not often result from athletic trauma, the serious nature of potential complications warrants careful evaluation of all thoracic injuries.

The initial assessment of all athletes with thoracic trauma follows the standard principles of Advanced Trauma Life Support (ATLS), with immediate attention to (A) airway with cervical spine immobilization, (B) breathing and ventilation, and (C) circulation with hemorrhage control. The goal of this primary survey is to simultaneously identify and manage life-threatening conditions before turning to a more comprehensive evaluation. Because of the lower-energy mechanisms associated with athletic trauma, thoracic injuries and their complications generally fall on the minor end of the spectrum. Furthermore, due to the infrequent nature of major thoracic injuries in sports, recommendations for management are commonly derived from the trauma literature, and return-to-play guidelines are often case-based or lacking altogether.

Rib Fractures

Mechanism of Injury in Sports and Epidemiology

Rib fractures are common in contact sports and include both osseous and cartilaginous rib injuries. Simple rib fractures are one of the most common injuries resulting from blunt chest trauma. Injuries to the costal cartilage, or chondral rib fractures, may also occur from blunt chest trauma and more often affect the sixth through eighth ribs based on anatomic vulnerability.

Clinical Presentation

Rib injuries may present with focal or generalized chest wall tenderness following blunt injury depending on the number of ribs involved. Rib and chondral fractures may be accompanied by palpable deformity. Pain is often moderate or severe immediately following injury and will usually render the athlete unable to continue with participation. Sideline evaluation must include immediate assessment of the airway, breathing, and circulation to identify or exclude the possibility of life-threatening injury.

J. P. Feden (✉)
Department of Emergency Medicine, Alpert Medical School of Brown University, Providence, RI, USA
e-mail: jfeden@lifespan.org

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Diagnosis

Plain radiography (anteroposterior and lateral chest radiographs) is the initial imaging modality of choice for blunt chest trauma. Sensitivity for diagnosing rib fractures is poor, but chest radiographs are important in evaluating for associated injuries, such as pneumothorax. The clinical utility of dedicated rib radiographs is controversial but may increase sensitivity for identification of rib fracture(s). When radiographs are negative and clinical suspicion remains, advanced imaging can be utilized to increase diagnostic sensitivity but may not change clinical management. CT is the preferred imaging modality for osseous and chondral rib fractures if plain films are negative. MRI may be useful for evaluating suspected chondral rib injury when CT is negative as it may identify nondisplaced chondral fractures [1]. Diagnostic point-of-care ultrasound is gaining popularity and shows promise for the detection of rib fractures that may not be evident on plain radiographs [2].

Initial Management

Sideline management of blunt chest injury should proceed in accordance with the standard principles of ATLS as described above. Initial management of suspected or confirmed rib fracture(s) often involves removal from competition and rest until acute pain subsides. NSAIDs may be used for analgesia, and opioids are sometimes necessary for severe pain. Lidocaine patches can be useful and effective adjuncts for pain management as well. Injection of local anesthetic to allow for expedited return to competition is controversial and usually reserved for elite or professional athletes.

Indications for Referral

Referral for evaluation in the emergency department must be considered in athletes with shortness of breath, diminished breath sounds, or any concerns for associated injury, such as pneumothorax. Additionally, athletes with abdominal pain and tenderness should be referred for emergency department evaluation of potential solid organ (e.g., liver and splenic) injuries which may require abdominal imaging.

Follow-Up Care

Evaluation of clinical healing based on serial examination is generally sufficient follow-up care. Serial imaging of rib fractures to evaluate or document radiographic healing is rarely necessary in uncomplicated cases.

Return to Sports

The return-to-play decision following simple rib fractures is often guided by symptoms. Progression of activity may be allowed as tolerated by pain. Athletes may be capable of returning to play in as little as 3–4 weeks [1]. Sport-specific functional abilities should be carefully evaluated prior to returning an athlete to competition. Rib protectors may be considered for contact athletes on an individualized basis.

Complications

Initial concerns with rib fractures are focused on associated thoracic and abdominal injuries, including pneumothorax, hemothorax, pulmonary contusion, and solid abdominal organ injuries. Delayed and long-term complications are uncommon.

Other Considerations

Chest wall contusions are evaluated and treated similar to confirmed rib fractures or suspected rib fractures without complication. Diagnostic imaging may be used to evaluate for rib fracture based on clinical concern. If initial imaging is negative, advanced imaging may be considered for diagnostic purposes, or the clinician may choose to proceed with conservative management as described above, with a focus on symptomatic therapy and return to activity as tolerated.

Sternal fractures are more common in high-energy trauma than athletic trauma and are rarely published in the sports medicine literature. Stress fractures of the sternum occur more commonly than traumatic sternal fractures which have been reported in rugby and ice hockey [3, 4]. Diagnosis of sternal fracture is made by plain radiographs or CT, and associated blunt cardiac and thoracic injuries must be considered. In addition to diagnostic imaging, an electrocardiogram should be obtained.

Similar to sternal fractures, fractures of the first rib occur much more commonly as stress injuries in athletes than traumatic injuries. Traumatic fracture of the first rib in contact athletes results from sudden, forceful contraction of the scalene muscles and may present as shoulder, scapular, or lower cervical pain [5]. While isolated, nondisplaced first rib fractures are typically uncomplicated, associated injury to the subclavian vessels, brachial plexus, lung, aortic arch, trachea, and esophagus must be considered based on anatomic proximity. It has been recommended that contact athletes with traumatic shoulder and/or neck pain should be evaluated for first rib fracture in the absence of an alternative diagnosis [5].

Pneumothorax

Mechanism of Injury in Sports and Epidemiology

Pneumothorax is defined as a collection of air within the pleural space and can occur spontaneously or as a result of pleural injury (with or without rib fracture) following blunt trauma to the chest. Traumatic pneumothorax can result from laceration of the visceral pleura by a fractured rib or from an abrupt increase in airway pressures and subsequent bronchial or alveolar rupture (e.g., barotrauma).

Traumatic pneumothorax is uncommon in athletics but has been reported in several contact and collision sports. Pneumothorax has also been described in relation to barotrauma from scuba diving and Valsalva maneuvers during weightlifting. The largest published series of sports-related pneumothorax details a majority of cases related to skiing, snowboarding, and other high-energy mechanisms of injury [6].

Classification

Primary pneumothorax is described as spontaneous or traumatic. Tension pneumothorax occurs with increasing pressure in the affected hemithorax and leads to hypoxia, mediastinal shift, decreased cardiac output, and cardiopulmonary collapse if left untreated.

Clinical Presentation

Dyspnea with pleuritic chest pain is the classic presentation of pneumothorax. Other clinical manifestations include tachypnea, tachycardia, anxiety, and hyperresonance to percussion and diminished lung sounds on the affected side. Palpation of subcutaneous air in the chest wall or rib fractures may also be associated with underlying pneumothorax. Development of tension pneumothorax can be accompanied by altered mental status, respiratory distress, hypoxia, hypotension, and distended neck veins.

Diagnosis

Conventional chest radiography is the initial screening test following thoracic trauma. Pneumothorax can be identified as the accumulation of a small apical or much larger air collection on upright chest radiographs (Fig. 41.1). Chest CT has superior sensitivity for the detection of thoracic injuries, including pneumothorax, and its widespread use in trauma centers allows for the identification of occult pneumothorax which is otherwise not visualized on plain films. Bedside thoracic ultrasound is also gaining popularity in the evaluation of thoracic trauma and has demonstrated improved sensitivity compared to supine radiographs (in which air accumulates in the anterior pleural space and may be hidden from view) [7]. It is important to understand that tension pneumothorax may be immediately life-threatening and should be diagnosed clinically. Delaying treatment for radiographic confirmation can result in hemodynamic collapse and death.

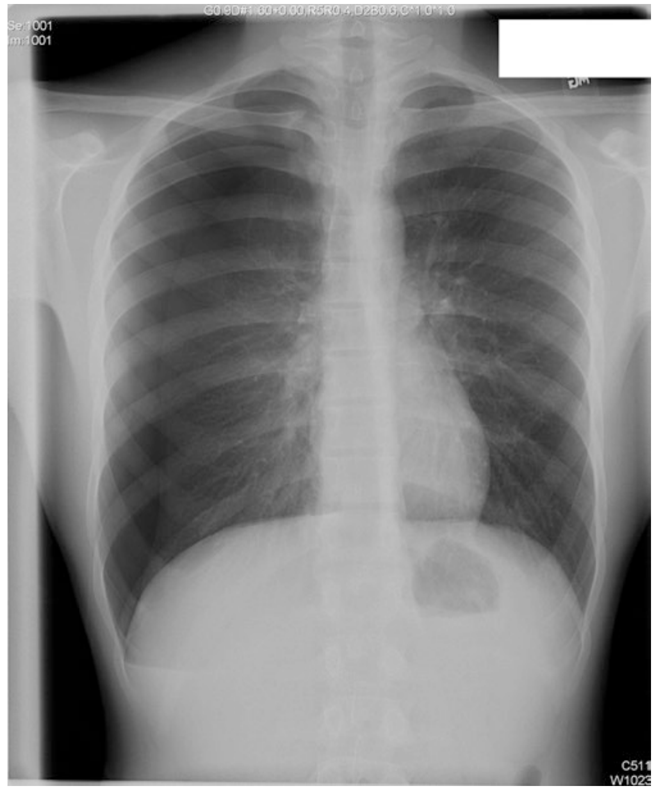

Fig. 41.1 Chest radiograph demonstrating large right-sided pneumothorax. (Courtesy of Robert Tubbs, MD, Providence, RI)

Initial Management

Initial management of pneumothorax focuses on the assessment of airway, breathing, and circulation. Supplemental 100% oxygen should be administered immediately. Suspicion of tension pneumothorax, such as respiratory distress and/or hemodynamic instability, must be addressed promptly and is treated with needle decompression. Decompression is performed with a 14G or 16G needle which is inserted perpendicular to the chest wall at the second intercostal space in the midclavicular line, resulting in a rush of air and rapid clinical improvement (Fig. 41.2). The needle catheter should be left in place while still recognizing that tension pneumothorax

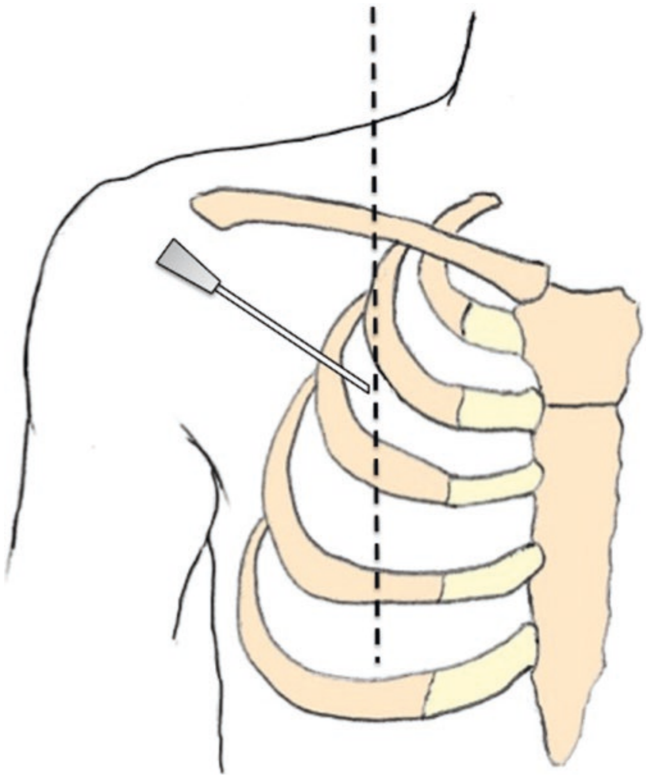

Fig. 41.2 Needle decompression of tension pneumothorax. (Illustration by Yvonne Chow, MD)

can recur if the catheter becomes dislodged. Definitive treatment is achieved by placement of a large-bore chest tube to allow lung re-expansion.

Occult pneumothorax may be safely managed with observation alone. Follow-up radiographs after several hours of observation are recommended to evaluate for progression. Stable patients with small simple traumatic pneumothorax may also be observed though most will be treated with chest tube thoracostomy. Small-bore chest tubes, also known as pigtail catheters, may be considered in uncomplicated cases. Larger pneumothoraces or unstable patients with traumatic pneumothorax require treatment with a large-bore chest tube.

Indications for Referral

Any athlete with suspected pneumothorax following blunt thoracic trauma should be referred to the emergency department for evaluation and emergent imaging.

Follow-Up Care

Patients with traumatic pneumothorax are often hospitalized and should have appropriate follow-up with a trauma or general surgeon following discharge.

Return to Sports

No specific guidelines exist for returning to sports after traumatic pneumothorax. The timeline for return ranges from 2 to 10 weeks based on case reports and expert opinion and should not be allowed prior to radiographic resolution [8, 9]. Progressive return to activity may be guided by symptoms as pain is often the limiting factor. Returning to play approximately 3–4 weeks after resolution of pneumothorax has been suggested [9].

Complications

Traumatic pneumothorax can be associated with and complicated by other life-threatening conditions such as hemothorax and tension pneumothorax. Pneumothoraces treated with chest tube may be complicated by persistent air leak, failure of lung re-expansion, and re-expansion pulmonary edema.

Other Considerations

With some athletes and teams traveling long distances by plane, the question of safe air travel following pneumothorax sometimes arises. The Medical Guidelines for Airline Travel indicate that pneumothorax is an absolute contraindication to air travel but should be safe by 2–3 weeks following successful treatment [10]. Available evidence is lacking, but a small study concluded that air travel 2 weeks following radiographic resolution of pneumothorax is safe [11].

Hemothorax

Mechanism of Injury in Sports and Epidemiology

Hemothorax represents a collection of blood within the pleural cavity and can result from aortic injury, myocardial rupture, or injuries to the hilum, intercostal vessels, or mammary vessels. It is more likely to occur with high-energy blunt or penetrating thoracic trauma and has been rarely reported in sports. Hemothorax is often accompanied by pneumothorax, leading to the term hemopneumothorax.

Clinical Presentation

Clinical presentation of hemothorax resembles that of pneumothorax with dyspnea, tachypnea, and hypoxia. Additionally, hypotension may result from hemorrhagic shock.

Diagnosis

A supine (Fig. 41.3) or upright (preferred) chest radiograph is the initial diagnostic test for blunt chest trauma and suspected hemothorax. Chest films can detect fluid collections greater than approximately 300 mL which will blunt the costophrenic angle. CT has superior sensitivity and complements the chest radiographs. Point-of-care ultrasound examination known as the extended focused assessment with sonography for trauma (eFAST) is also very useful for rapid diagnostic evaluation of hemothorax but requires a skilled operator.

Initial Management

Initial management of hemothorax focuses on the assessment of airway, breathing, and circulation. Occult hemothorax identified on CT imaging may be observed in some cases. However, hemothorax is frequently treated with large-bore tube thoracostomy to allow adequate drainage. Massive hemothorax defined by large-volume bleeding requires blood transfusion and emergent operative management.

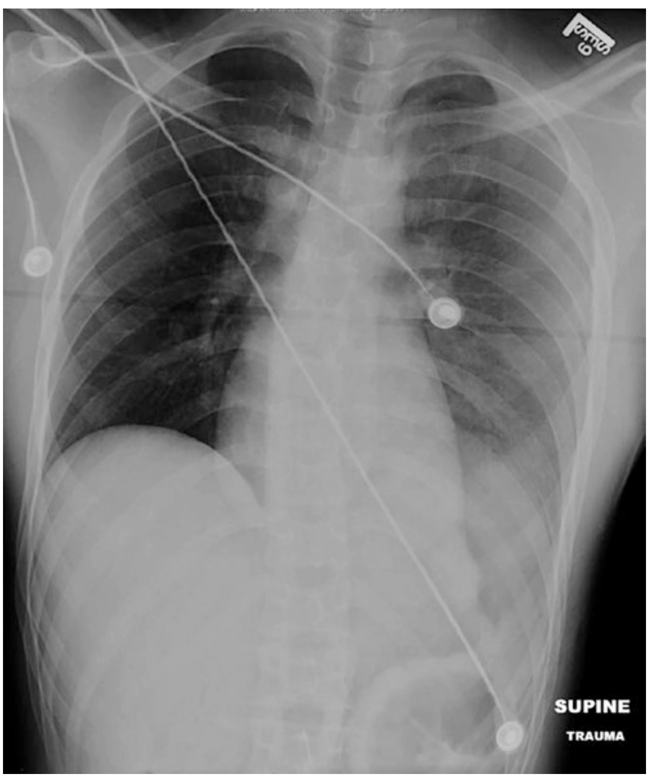

Fig. 41.3 Supine chest radiograph demonstrating left-sided hemopneumothorax. Hazy opacification of left hemithorax is consistent with hemothorax in a supine position. A "deep sulcus sign" on the left corresponds to an abnormally deepened left costophrenic angle due to the accumulation of air in the anterior and basal pleural space representative of pneumothorax. (Courtesy of Robert Tubbs, MD, Providence, RI)

Indications for Referral

Any athlete with suspected hemothorax following blunt thoracic trauma should be referred to the emergency department for evaluation and emergent imaging.

Return to Sports

There are no specific guidelines for returning to play following hemothorax. The decision to return to sports should be made in consultation with a trauma or general surgeon.

Complications

Massive hemothorax is a life-threatening condition and may cause hemorrhagic shock.

Pulmonary Contusion

Mechanism of Injury in Sports and Epidemiology

Pulmonary contusion is a common complication of blunt thoracic trauma characterized by parenchymal hemorrhage, interstitial edema, and alveolar collapse with the potential for respiratory compromise. It occurs uncommonly in sports, but case reports in football players exist in the literature [12, 13].

Clinical Presentation

Clinical findings of pulmonary contusion may occur immediately or in delayed fashion and include dyspnea, hypoxia, tachypnea, hemoptysis, chest pain, and wheezing. Lung auscultation may reveal crackles or diminished breath sounds.

Diagnosis

As with all instances of thoracic trauma, chest radiography is the initial test of choice but may underestimate the size of a pulmonary contusion. Radiographic findings may include focal or diffuse opacification extending beyond lung segments and may develop up to 48 h following injury (Fig. 41.4). CT has improved sensitivity for pulmonary contusion compared to chest X-ray and is often the preferred diagnostic imaging modality with the added advantage of quantifying the injury.

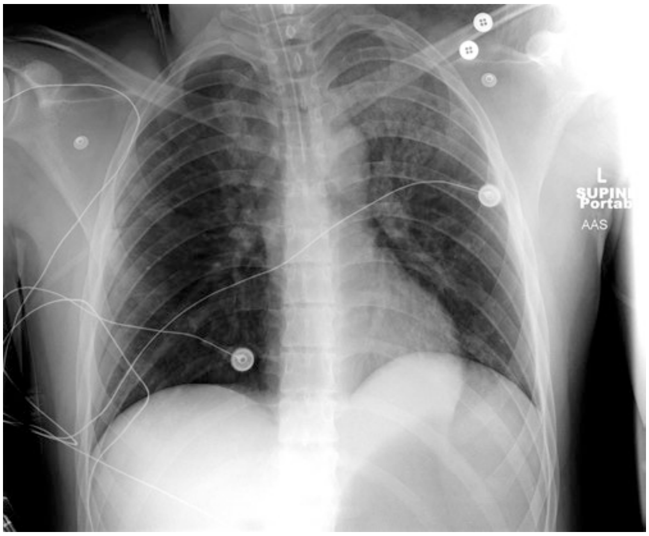

Fig. 41.4 Chest radiograph demonstrating pulmonary contusion. (Courtesy of Sarah Desoky, MD, Tucson, AZ)

Initial Management

Management of pulmonary contusion depends on the extent of injury. Occult injuries identified by CT and other minor injuries require little more than supportive care consisting of supplemental oxygen, pain management, and hemodynamic monitoring. Mechanical ventilation may be necessary for severe hypoxemia and respiratory failure.

Indications for Referral

Athletes with thoracic trauma significant enough to produce hypoxia, hemoptysis, or progressive or unresolving dyspnea should be referred for evaluation and imaging in the emergency department.

Return to Sports

There are no specific guidelines for returning to sports following pulmonary contusion. Case reports in football involving occult, mildly symptomatic contusions describe returning to exercise in 2–3 days with return to full athletic participation by 1 week [12, 13]. Athletes should not return to activity until respiratory symptoms have subsided.

Complications

Complications of pulmonary contusion range from pneumonia to acute respiratory distress syndrome (ARDS) in severe cases. Injuries associated with athletic trauma are generally mild and have a benign course with an uncomplicated recovery.

Pneumomediastinum

Mechanism of Injury in Sports and Epidemiology

Pneumomediastinum is defined by the presence of air in the mediastinal space and may be spontaneous or traumatic. Traumatic pneumomediastinum has been reported in collision sports, such as football and ice hockey [14, 15], and concern regarding injury to the tracheobronchial tree or aerodigestive tract often accompanies this injury. It has been proposed, however, that pneumomediastinum in collision sports is more closely related to the spontaneous form than that associated with major trauma [15]. Similar to spontaneous pneumomediastinum, it is believed that a Valsalva-type force occurs during collision and causes alveolar rupture rather than pleural or tracheobronchial injury. Air then dissects along the bronchovascular bundle into the mediastinum.

Classification

Pneumomediastinum may be spontaneous or traumatic.

Clinical Presentation

Chest pain, dyspnea, and neck pain are the most common symptoms of pneumomediastinum. Chest pain may radiate to the back or neck and may worsen with inspiration or swallowing. Air may track from the mediastinum into the cervical fascial planes producing cervical or supraclavicular subcutaneous emphysema which can be palpated as subcutaneous crepitation. One of the classic physical exam findings associated with pneumomediastinum is the auscultation of a crunching sound over the precordium known as the Hamman crunch.

Diagnosis

Pneumomediastinum can be diagnosed on a chest radiograph showing air streaks in the superior mediastinum, prominence of the left cardiac silhouette, or subcutaneous air in the neck (Fig. 41.5). Diagnosis of pneumomediastinum often prompts further evaluation for aerodigestive tract injury with water-soluble or barium contrast esophagram though such injuries are exceedingly rare in athletes.

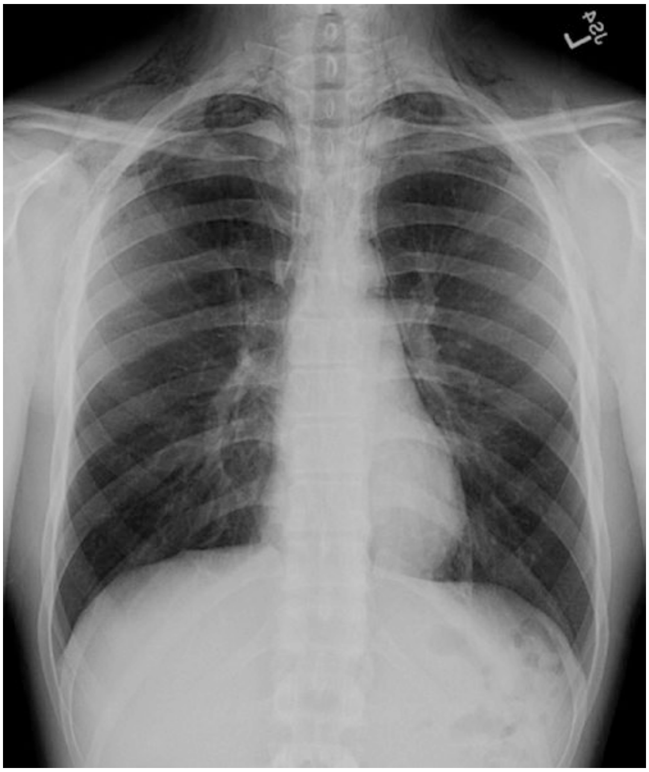

Fig. 41.5 Chest radiograph demonstrating pneumomediastinum with associated subcutaneous air extending into the neck

CT can also identify occult pneumomediastinum and proves useful in cases of suspected esophageal perforation as well.

Initial Management

Pneumomediastinum in athletes typically follows a benign course. A brief period of observation is often sufficient to ensure stability. Clinical deterioration requires additional diagnostic testing and consultation with a thoracic surgeon if necessary.

Indications for Referral

Athletes with characteristic symptoms of chest pain, neck pain, or dyspnea, or other clinical findings of pneumomediastinum, should be promptly referred for evaluation and imaging in the emergency department.

Follow-Up Care

Symptoms of pneumomediastinum should resolve within a few days. Radiographic resolution occurs between a few hours and 1–2 weeks. Follow-up care usually consists of primary care or sports medicine follow-up prior to returning to play. Repeat imaging is generally unnecessary.

Return to Sports

There are no evidence-based guidelines directing return-to-play decisions following pneumomediastinum. Return to activity often commences upon resolution of symptoms and may include documentation of radiographic resolution. Cases in the literature support return to full participation between 1 and 4 weeks but usually less than 2 weeks [14, 15]. Recommendations for air travel parallel those for pneumothorax with air travel presumed to be safe at 2–3 weeks assuming radiographic resolution [10].

Complications

Feared complications of pneumomediastinum relate to associated tracheobronchial or aerodigestive tract injury, such as esophageal perforation. Such injuries are rare but potentially fatal. Blunt aerodigestive injuries occur in less than 0.1% of patients with blunt trauma. Additionally, almost 80% of tracheobronchial injuries result in death prior to reaching the hospital [16].

Blunt Cardiac Injury

Mechanism of Injury in Sports and Epidemiology

Blunt cardiac injury can result from a direct precordial blow, compression injury, or deceleration mechanism. The right ventricle is most often injured because of its anterior anatomical position relative to the thoracic wall. The spectrum of injury ranges from myocardial contusion to rupture and can manifest as asymptomatic ECG changes, dysrhythmia, or cardiogenic shock and death. The incidence in sports is unknown, and there are no well-validated diagnostic or treatment algorithms [17].

Clinical Presentation

The clinical presentation of blunt cardiac injury can range from chest pain and dyspnea to sudden collapse and death. In rare cases, blunt precordial trauma can precipitate life-threatening dysrhythmias, such as ventricular fibrillation, which may present with collapse following a brief period of normal consciousness.

Diagnosis

The diagnosis of blunt cardiac injury is often challenging owing to its poor definition and varied clinical manifestations. History and physical exam may be nonspecific and unreliable. Athletes with suspected blunt cardiac injury should be evaluated with 12-lead ECG and chest radiographs evaluating for cardiac rhythm disturbance and associated thoracic injuries, respectively. ECG abnormalities associated with blunt cardiac injury include sinus tachycardia, dysrhythmias, and ST-segment changes. Despite poor sensitivity alone, cardiac enzymes such as troponin I are often used alongside ECG to rule out clinically significant blunt cardiac injury [18] but do not necessarily predict complications. Echocardiography should be performed early in cases of blunt cardiac injury with hemodynamic instability or shock but is not suitable as a screening test.

Initial Management

Ventricular dysrhythmia should be assumed in any athlete experiencing sudden collapse following blunt precordial trauma with initial resuscitative efforts focused on prompt defibrillation if possible. Resuscitation should otherwise proceed according to standard guidelines for advanced cardiac life support. All cases of suspected blunt cardiac injury should be transported to the emergency department for evaluation and diagnostic testing. A screening ECG is essential, and troponin I may also be used for screening purposes but remains controversial. Normal screening tests generally exclude clinically significant blunt cardiac injury. However, an abnormal ECG (e.g., persistent unexplained tachycardia, new bundle branch block, dysrhythmia) and/or cardiac biomarkers warrant admission and observation for 24–48 h to monitor for evolving injury. In addition to hemodynamic instability, echocardiography may be used when screening tests are abnormal. Most blunt cardiac injuries are self-limited. However, heart failure, heart block, or significant structural injuries should be managed accordingly.

Return to Sports

There are no specific guidelines for returning to sports following blunt cardiac injury [17]. The return-to-play decision may be aided by consultation with a cardiologist if uncertainty exists.

Complications

The clinical significance of blunt cardiac injury is related predominantly to its complications which can include myocardial rupture, coronary artery injury, ventricular dysrhythmias, fulminant cardiac failure, and death. Commotio cordis is defined by sudden cardiac death following blunt cardiac injury in individuals without pre-existing cardiac disease or abnormalities.

Pediatric Considerations

Commotio cordis is rare but remains a common cause of sudden death in young athletes occurring with a peak incidence between 11 and 19 years old [19]. It results from a precordial impact that occurs during ventricular repolarization which induces the R-on-T phenomenon (Fig. 41.6) and precipitates ventricular fibrillation. High-quality cardiopulmonary resuscitation and early defibrillation are critical to optimizing chances for survival. Prevention is possible by softening the impacting object (i.e., safety baseballs), but standard chest protectors are not universally effective.

Vascular Injury

Mechanism of Injury in Sports and Epidemiology

Blunt aortic injury and injury to the great vessels are rarely reported in sports as they more commonly result from high-energy traumatic mechanisms. However, such vascu-

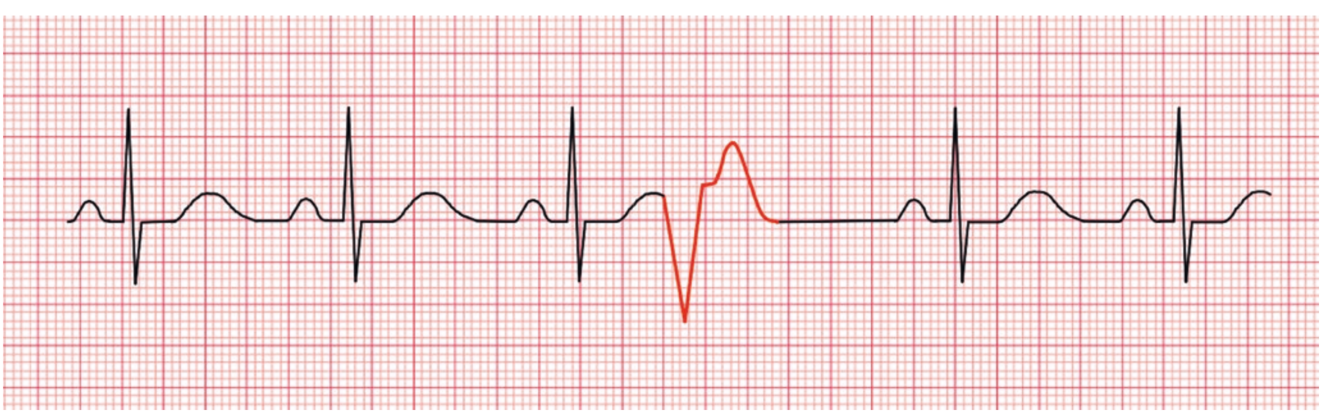

Fig. 41.6 Rhythm strip demonstrating the R-on-T phenomenon. (Illustration by Yvonne Chow, MD)

lar injuries should be considered as a potential complication in any athlete with posterior sternoclavicular dislocation (described elsewhere). Nontraumatic, exercise-related aortic rupture and aortic dissection are well-described complications and causes of sudden death in athletes with inherited aortic conditions, such as Marfan syndrome, but are outside the scope of this discussion [20]. Finally, inferior vena cava injury may occur in collision sports as demonstrated by a high-profile, near-fatal case in a college football player in 2012.

Clinical Presentation

Most aortic injuries surviving to the hospital are partial or contained injuries and may present along a spectrum of hemodynamic stability. Occasionally, these injuries might present without clinical signs initially, but any indication of hemorrhagic shock (i.e., hypotension, tachycardia, altered mental status, syncope) should be alarming. Fieldside evaluation follows standard ATLS protocols, and suspected vascular injury must be referred to the hospital for emergent evaluation.

Diagnosis

Screening chest radiographs may show mediastinal widening, but computed tomography (CT) is the diagnostic imaging modality of choice based on superior sensitivity and specificity.

Initial Management

Management of hemodynamically unstable injuries to the aorta or great vessels focuses on hemorrhage control and definitive operative repair. Blood products may be necessary in addition to intravenous fluids. Hemodynamically stable or contained injuries require blood pressure control to reduce the risk of impending rupture, in addition to surgical consultation.

Return to Sports

Returning to athletics, if possible, must be managed collaboratively with the surgical specialist.

Complications

Complications of aortic or other thoracic large vessel injury include hemorrhagic shock and death.

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42 Abdominal Trauma

Jaron Santelli, Jeremiah W. Ray, and Jonathan T. Finnoff

Key Points

- Sports providers should familiarize themselves with presentations of abdominal trauma.
- A primary and secondary survey should be completed on all athletes suspected of sustaining abdominal trauma to help identify life-threatening injuries.
- The spleen is the most common solid organ injured in blunt abdominal trauma.
- Any athlete that is suspected to have significant trauma to his or her abdomen, shows signs of worsening condition, or shows signs of shock should be transported to the nearest emergency department, ideally one with trauma capabilities.

Introduction

Abdominal injuries resulting from sports can be life-threatening; however, the majority of injuries are not severe enough to cause internal hemorrhage or death. Higher velocity and contact sports such as football, hockey, lacrosse, wrestling, rugby, and snowboarding have a higher rate of more significant abdominal injuries; however, the numbers are still small. Children are at an increased risk of injury secondary to their poorly defined musculature and smaller anteroposterior diameter. In addition, the pediatric athlete's ribs are more compliant and provide less overall protection to the abdominal organs, especially the liver and spleen.

There is limited data available on individual organ injuries. In addition, many abdominal injuries go unreported, making injury rates overall underreported. The National Collegiate Athletic Association (NCAA) injury surveillance data on collegiate sports from 2003 to 2004 reported that trunk or back injuries made up 13.2% of overall reported injuries during games and 10% of total reported injuries during practice [2]. The severity of these injuries is not further discussed. This same data set cites a high of 0.71 abdominal muscle injuries per 1000 player-hours in wrestling competition to a low of 0.01 injuries per 1000 player-hours in autumn football practices in 2004–2005. The British professional soccer clubs reported an incidence of "torso" injuries of up to 7% of all injuries [3].

Most abdominal injuries in sports are due to blunt trauma. There are three pathophysiologic mechanisms which lead to injury in blunt abdominal trauma: (1) Crush injury leading to compression of organs between the anterior abdominal wall and the posterior thoracic cage or vertebral column. This type of mechanism tends to put solid viscera at a higher risk. (2) Shearing forces transmitted across the abdominal wall due to a rapid acceleration or deceleration injury. These injuries tend to put both hollow and solid organs at risk. (3) Sudden and pronounced increase in intra-abdominal pressure caused by an outward force. This increase in pressure can cause a rupture or burst of hollow organs such as the intestine.

Imaging athletes with abdominal trauma will depend on several factors including the hemodynamic stability of the athlete, the mechanism of injury, the location of injury, and the providers' suspicion for injury. Focused assessment with sonography for trauma (FAST) exams should be the first-line imaging modality, especially in the unstable athlete. FAST or extended focused assessment with sonography for trauma

J. Santelli
Department of Emergency Medicine and Orthopedics, University of Maryland Medical Center, Baltimore, MD, USA
e-mail: jaron.santelli@som.umaryland.edu

J. W. Ray
Department of Orthopedic Surgery, University of California at Davis Medical Center, Davis, CA, USA
e-mail: jwray@ucdavis.edu

J. T. Finnoff (✉)
Department of Physical Medicine and Rehabilitation, Mayo Clinic Sports Medicine Center, Minneapolis, MN, USA
e-mail: finnoff.jonathan@mayo.edu

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(eFAST) exams may be performed on the sideline, training room, or emergency department as an adjunct to the physical exam in identifying athletes with acute intra-abdominal injury by quickly diagnosing intra-abdominal bleeding (see section below for more details). A computed tomography (CT) scan will be the gold standard for identifying most abdominal injuries as it can identify injuries in both the peritoneal and retroperitoneal spaces. CT scans should be done with contrast in the setting of trauma if there is no concern for renal dysfunction. A CT should only be done in a hemodynamically stable patient. Plain films are of limited utility but may identify free air under the diaphragm in intestinal rupture, lower rib fractures, or elevation of the hemidiaphragm after a diaphragm rupture.

All evaluations of athletes with abdominal trauma should follow Advanced Trauma Life Support (ATLS) guidelines. Initial evaluation should include a primary survey followed by a secondary survey. Repeat assessments are essential, including repeat vital signs, to identifying injuries with delayed presentation.

Extended Focused Assessment with Sonography for Trauma (eFAST)

The use of ultrasound in the setting of blunt thoracoabdominal trauma was first described in the literature by Dr. Kristensen et al. in 1971 [4]. However, it was not adopted for trauma in the United States of America until the 1990s. Dr. Rozycki and colleagues introduced the term FAST, or Focused Assessment with Sonography in Trauma, in 1995 [5]. It is a rapid four-view ultrasound examination to evaluate for the presence of hemoperitoneum and hemopericardium [5–9]. The FAST examination is now a component of the Advanced Trauma Life Support (ATLS algorithm [6]. Approximately 200 mL of free fluid is required to identify free intraperitoneal fluid. With this amount of free fluid, the sensitivity of the FAST examination for abdominal trauma ranges from 42% to 98% and the specificity from 95% to 100% [10–21]. The eFAST, or extended FAST examination, further evaluates for the presence of a pneumothorax or hemothorax. The sensitivity and specificity for pneumothorax with ultrasound are 86–98% and 97–100%, respectively, whereas the same for supine anteroposterior chest radiographs are 28–75% and 99–100%, respectively [22–25].

Although the eFAST is currently used predominantly in the emergency department setting, its use on the field is not unprecedented. A study by Strode et al. [26] in 2003 demonstrated that performing a FAST examination with a portable ultrasound unit in the prehospital setting was feasible. On the sidelines, the eFAST examination could be used to evaluate athletes with suspected thoracoabdominal trauma and improve the triage process. In particular, if a sideline eFAST examination reveals the presence of thoracoabdominal trauma in an athlete, it may facilitate earlier initiation of definitive treatment, thus reducing the morbidity and mortality associated with these injuries.

To perform the FAST examination, select a low-frequency (e.g., 7–1 MHz) phased array or curvilinear array transducer. Four areas are interrogated during a standard FAST examination [7]. The first is the hepatorenal recess (Morison's pouch) located in the right upper quadrant of the patient's abdomen (Fig. 42.1). The second are the splenorenal and splenodiaphragmatic recesses located in the patient's left upper quadrant (Fig. 42.2). The presence of free fluid (which appears hypo- or anechoic) in either of these two recesses suggests a hemoperitoneum.

The suprapubic region is evaluated next and is facilitated by a full bladder [7]. When free fluid is located in the

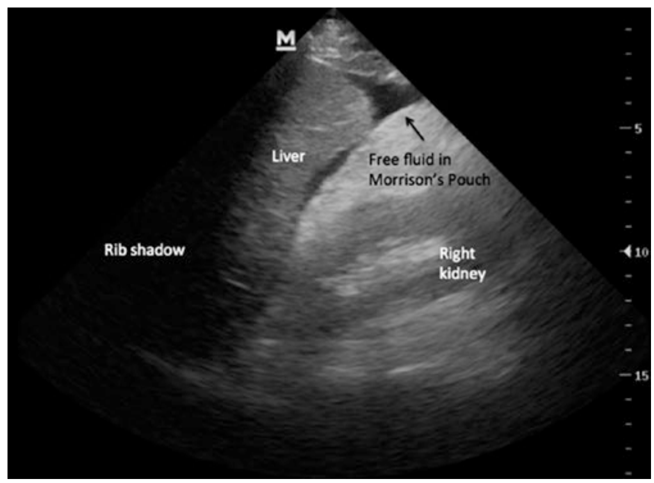


Fig. 42.1 Right upper quadrant: hepatorenal recess (Morison's pouch)

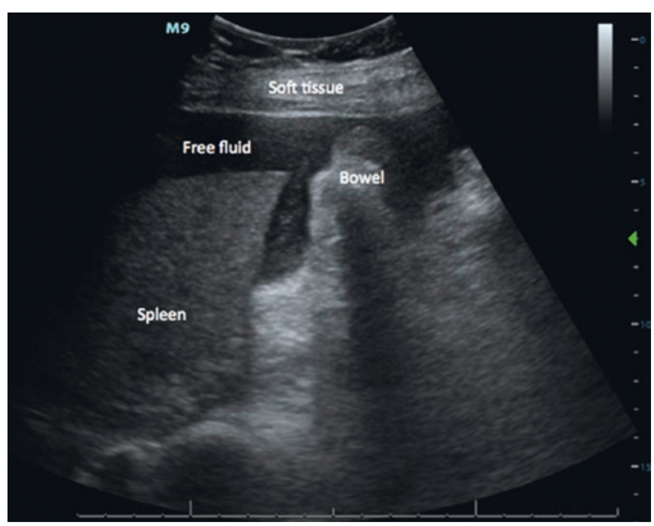


Fig. 42.2 Left upper quadrant: splenorenal and splenodiaphragmatic recesses

retro-vesicular (male) or retro-uterine (female) pouch, it presents as a hypo- or anechoic fluid collection posterior (deep) to the posterior bladder wall (Fig. 42.3). The posterior bladder wall has a hyperechoic appearance and is located between the anechoic urine contained within the bladder and the free fluid located in the retro-vesicular or retro-uterine pouch [7].

The subxiphoid region is the final area evaluated during the standard FAST examination [7]. The right ventricle is identified, and the ventricular wall is interrogated for the presence of a pericardial effusion. A pericardial effusion appears as a hypo- or anechoic fluid collection between the pericardium and myocardium (Fig. 42.4).

In addition to the previously mentioned four regions evaluated during the FAST examination, the eFAST examination includes an assessment for a pneumothorax [22]. Although a curvilinear or phased array transducer can be used, a high-frequency (e.g., 18–5 MHz) linear array transducer is ideal for this portion of the examination due to its ability to obtain a high-resolution image of superficial structures. The transducer should be placed between the ribs, and the pleura should be evaluated for the normal sliding movement that occurs between the visceral and parietal pleura with the associated hyperechoic comet-tail artifacts (Fig. 42.5) [22]. The absence of this sliding movement and comet-tail artifacts is highly suggestive of a pneumothorax (Fig. 42.6).

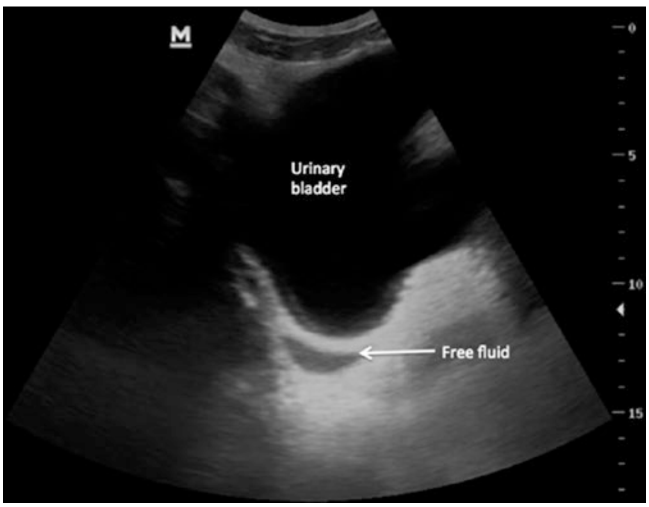


Fig. 42.3 Suprapubic region: retro-vesicular (male) or retro-uterine (female) pouch

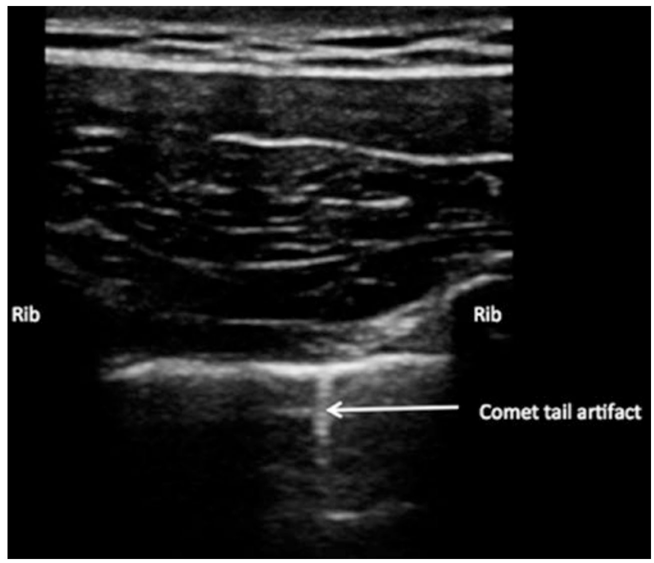


Fig. 42.5 Thorax: comet-tail artifacts

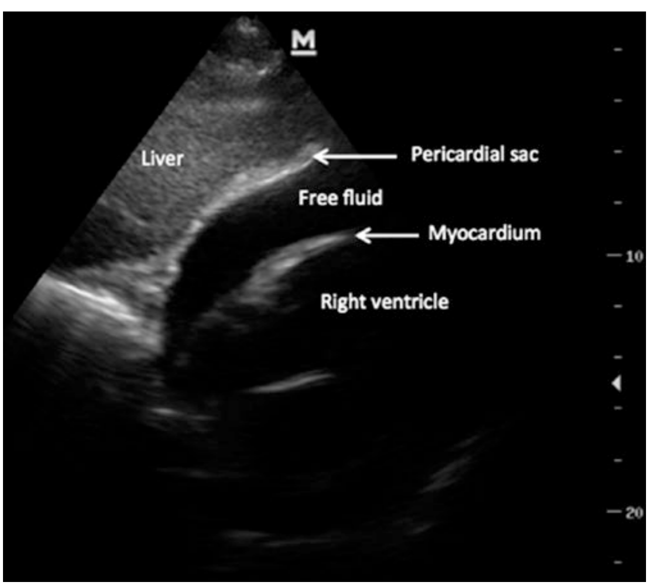


Fig. 42.4 Subxiphoid region: pericardial effusion

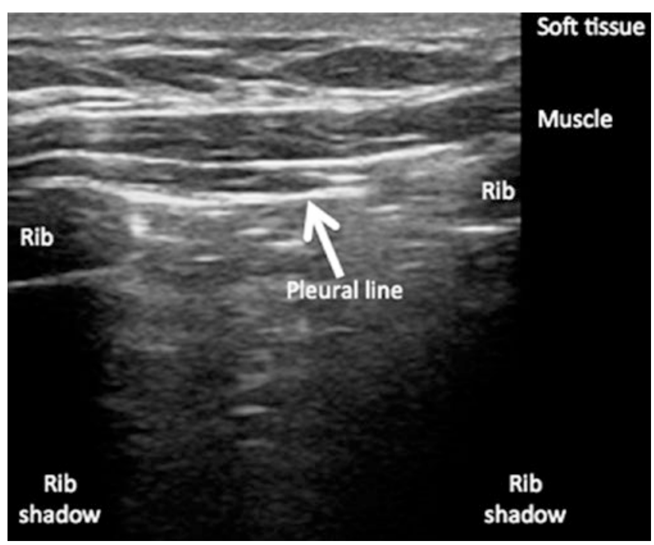


Fig. 42.6 Pneumothorax: absence of comet-tail artifacts

Abdominal Injuries by Organ

Liver

In all settings of blunt abdominal trauma, the liver is the second most commonly injured intra-abdominal organ, with an overall incidence of 1–8% [27]. Acute recognition of injuries to the liver can be difficult, but a good physical exam and eFAST or FAST examination can improve identification rates. When there is a high index of suspicion for injury to the liver, further imaging and workup will be needed.

Anatomy

The liver is the largest visceralorgan in the body located in the right hypochondrium and epigastric region. It has two distinct surfaces, the diaphragmatic surface and the visceral service. Anatomically, the liver is separated into two lobes, a larger right lobe and a smaller left lobe, by the falciform ligament (Fig. 42.7). From a surgical standpoint, the liver is divided into eight segments by the hepatic artery, the portal vein, and bile duct. The liver has a dual blood supply from the proper hepatic artery and the portal vein [28] (Fig. 42.8). The liver sees about 1500 mL per minute of blood. As a result of the multiple segments which receive individual blood supply and the sheer volume of blood through the liver per minute, controlling bleeding in a traumatic situation can be difficult.

Mechanism

Compression or crush injuries that may damage the liver are usually caused by collision sports such as football, soccer, or rugby. Compression injuries in the liver can lead to subscapular or intraparenchymal hematoma formation [1, 29]. In acceleration or deceleration injuries seen in sports such as

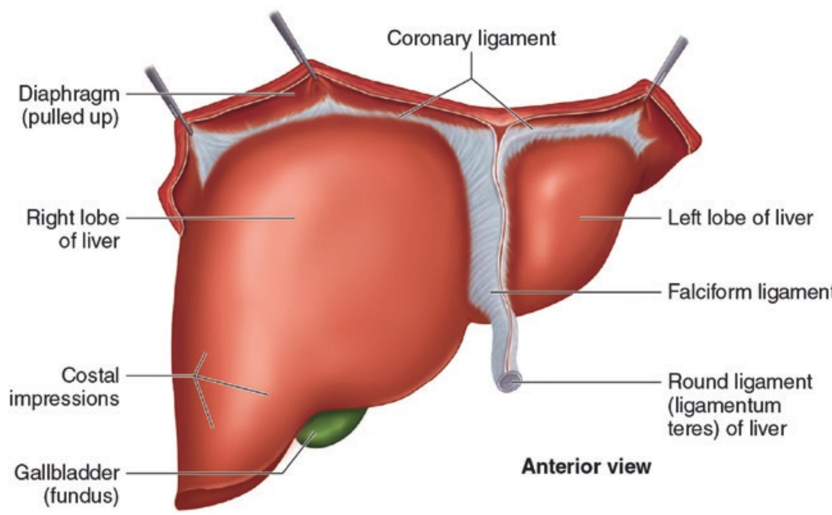


Fig. 42.7 Anatomy of the liver

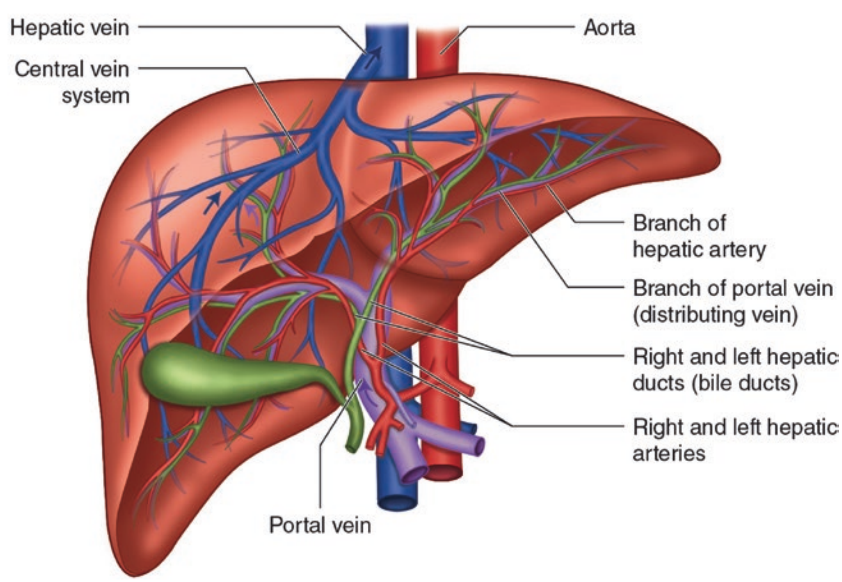


Fig. 42.8 Vasculature supply of the liver

Table 42.1 Liver injury scale [64]

Grade	Injury	Description
I	Hematoma	Subcapsular, nonexpanding, < 10% surface area
	Laceration	Capsular tear, nonbleeding, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, nonexpanding, 10–50% surface area
		Intraparenchymal, nonexpanding, < 2 cm in diameter
	Laceration	Capsular tear, active bleeding, 1–3 cm parenchymal depth, < 10 cm length
III	Hematoma	Subcapsular, > 50% surface area or expanding
		Ruptured subcapsular hematoma with active bleeding
		Intraparenchymal hematoma > 2 cm or expanding
	Laceration	> 3 cm parenchymal depth
IV	Hematoma	Ruptured intraparenchymal hematoma with active bleeding
	Laceration	Parenchymal disruption involving 25–50% of hepatic lobe
V	Laceration	Parenchymal disruption involving > 50% of hepatic
	Vascular	Juxtahepatic venous injuries, i.e., retrohepatic vena cava/major hepatic veins
VI	Vascular	Hepatic avulsion

The American Association for the Surgery of Trauma (AAST) presented a liver injury scale, advance one grade for multiple injuries to the same organ
Table from Moore et al. [64]

ski racing and motorsports, the liver is injured as a result of high velocity forces being transmitted across the organ. As a result of these forces, lacerations can occur within the capsule and at the underlying attachment point of the parenchyma. Injuries can also occur at the intima and media of nearby arteries [1, 29, 30].

Grading of Injury

Grading of liver injuries has been described by the American Association for the Surgery of Trauma (AAST) [31, 32]. The grading system of hepatic organ injury was developed to help determine intervention by injury severity. Injuries are described as grade I through grade VI where grade I is minor and grades III and above are considered severe. Grade VI is usually fatal. Luckily, the majority of injuries, ranging from 67% to 90% [31, 33], are considered minor or grades I–III. See Table 42.1.

Clinical Presentation and Physical Exam

The clinical presentation of athletes with closed liver injuries can vary depending on the severity of the injury. Initially, the hemodynamic stability of the athlete should be determined based on a primary and secondary survey. Athletes with severe injuries may present with hypotension and tachycardia consistent with shock. Athletes with less severe closed liver injuries will present with tenderness to palpation of the right upper quadrant. Peritoneal signs such as guarding, rebound tenderness, or rigidity may also be present; however, none of these are sensitive or specific for liver injury [29].

Additional physical exam findings that may suggest retroperitoneal hemorrhage or intra-abdominal hemorrhage include Grey Turner's sign and Cullen's sign [34]. Cullen's sign is described as superficial edema with bruising in the subcutaneous fatty tissue around the periumbilical region [35]**.** Grey Turner's sign refers to ecchymosis of the flanks and may co-occur with Cullen's sign especially in retroperitoneal hemorrhage [36]. Both signs however are delayed findings, are indicative of a significant amount of bleeding, and will likely not be present in an initial physical exam.

Evaluation

Sideline evaluation should begin with an understanding of the mechanism of trauma. A primary survey including assessment of vital signs should follow. Unstable patients should be stabilized according to ATLS guidelines [6] and the findings of the primary survey and transported without delay to the nearest emergency department with trauma capabilities (or the nearest emergency department if a trauma center is not available). Stable patients should have a more in-depth exam done looking for specific injury patterns suggestive of liver injury. Vital signs should be followed and repeated often to assess for signs of shock. Most hemorrhages of the liver occur within the first 24 h, and delayed bleeding is rare [33].

A sideline or training room FAST exam may be done to look for intraperitoneal hemorrhage. One may also see irregularities underneath the capsule of the liver suggesting hemorrhage or hematoma.

Any athlete with a suspected liver injury should also be evaluated in an emergency department with trauma capabilities (or the nearest emergency department if a trauma center is not available). Laboratory tests such as liver function tests (including aspartate aminotransferase (AST) and alanine aminotransferase (ALT)), serum electrolytes, complete blood count, coagulation studies, lipase, and blood type and screen or cross depending on the stability of the individual athlete should be performed. Indications for computed tomography (CT) scan with contrast, which is the gold standard for imaging diagnosis, include a high level of suspicion, right upper quadrant tenderness, elevated AST or ALT, gross hematuria, or a declining hemoglobin or hematocrit. Hemodynamically unstable patients should not undergo a CT scan but instead evaluated with bedside eFAST or FAST and by surgery. Emergent intervention by the surgical team may be required for findings such as free fluid found on a FAST, unstable vital signs, or worsening clinical condition.

In adults, AST values of greater than 400 IU/L or ALT values of 250 IU/L convey sensitivity and specificity of 92.9% and 100%, respectively, in the prediction of liver injuries [37]. One study done specifically in the pediatric population found that elevations in glucose and aspartate aminotransferase (AST) yielded the highest sensitivity with 75% and 65%, respectively [38, 39]; however, this same study, as well as others, notes that there is no laboratory value that is "adequately sensitive, specific or predictive so as to be clinically useful" [39]. In general, AST above 200 and ALT above 125 should prompt further evaluation either in the form of observation and repeat exam or by CT with IV contrast given the increased risk for intra-abdominal injury [40].

In athletes who have sustained a closed liver injury, providers should consider associated injuries. Up to 80% of patients with identifiable trauma to the hepatic system have at least one additional injury such as pulmonary injuries, pelvic fractures, or rib fractures [41].

Management

Management of closed liver injuries depends on the severity of the injury itself as put forth by the American Association for the Surgery of Trauma guidelines (Table 42.1). Athletes presenting with hypovolemic shock or with a significant drop in their hemoglobin and hematocrit will likely require blood transfusion. The hemodynamically unstable adult athlete will likely require intervention with embolization of bleeding vessels by interventional radiology or with an emergent laparotomy by a surgeon. There is limited data available on the management of hepatic injury using embolization in children. The decision to use embolization in the pediatric population should be made in conjunction with the pediatric surgeon and interventional radiologist [42, 43].

Athletes that require surgical intervention are likely to have grade III and above injuries. Hemodynamically stable athletes are more likely to be treated nonoperatively now as there has been a paradigm shift over the past several decades. Observation of stable athletes should occur in a facility that is able to provide emergent surgical intervention if deterioration of the athlete occurs.

Return to Play

Currently, there are no specific guidelines in regard to return to play for athletes who have sustained closed liver injuries. If an athlete is treated operatively, then return to play should be determined in coordination with the surgeon. For the athlete that was treated nonoperatively, he or she should only be cleared for return to play once normal anatomic function of the liver has returned. Specifically, the athlete's liver enzymes should have normalized. Serial imaging with CT scan or ultrasound is controversial and likely should not be done unless there is a specific deterioration in the athlete's status [29, 33]. Protective gear, such as flak jackets or increased padding around the liver, has not been studied for its effectiveness in protecting the organ.

Healing time in a closed liver injury is variable. A simple liver laceration or subscapular hematoma can heal in 2–4 months. A larger injury such as a complex laceration with vascular injury may take up to 6 months to heal [1]. Overall, resolution of the injury and return to play should be a coordinated decision between the surgeon who cared for the athlete initially, the team physician, the athlete, and his or her family.

Spleen

Although the liver is the most common cause of death due to intra-abdominal blunt trauma, the spleen is the most commonly injured organ in blunt trauma sustained in sports and the organ most likely to result in intra-abdominal hemorrhage.

Anatomy

This spleen is located in the left upper quadrant and lies against the left hemidiaphragm. It is protected in that area by ribs IX through XII. The spleen is connected to the curvature of the stomach by the gastrosplenic ligament and to the left kidney by the splenorenal ligament. Both of these ligaments contain blood vessels (Fig. 42.9).

The blood supply for the spleen comes from the splenic artery, which enters the organ through the hilum [28]. This organ filters about 15% of the body's blood supply every minute and is susceptible to significant bleeding with trauma [44]. The spleen also comprises about 25% of the total lymphoid mass of the body making it one of the largest lymphopoietic organs in the body [39]. In children, the spleen may not yet be covered by the rib cage, and the ribs that do cover the organ are more compliant providing less protection. Children also lack the adipose tissue and muscle tissue of adults, further contributing to the increased vulnerability to splenic injury in children [44].

Mechanism of Injury

As with the liver, the spleen can be injured by either direct compressive or crush injuries or by acceleration/deceleration forces. The spleen can be more susceptible to injury in the setting of infection, pregnancy, and portal hypertension as these factors can lead to splenic enlargement.

Delayed rupture and hemorrhage of the spleen is more common in adults than other organs such as the liver. The splenic capsule is able to contain bleeding rendering early signs of hemorrhage less reliable. Delayed bleeding has also been attributed to factors such as the development of pseudoaneurysms, splenic abscess, or secondary tearing of the parenchyma after adherence to the omentum [1, 33]. More

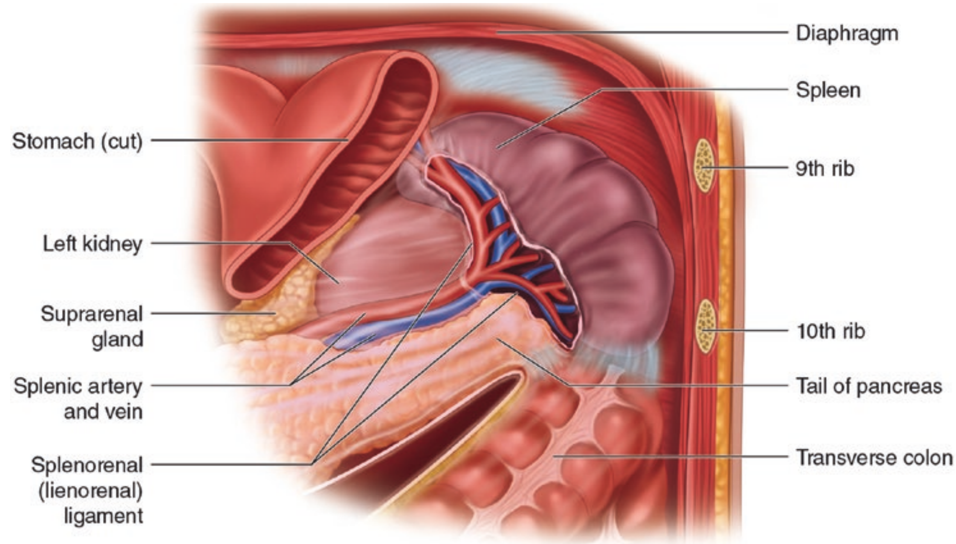


Fig. 42.9 Anatomy of the spleen

than 90% of secondary bleeds or ruptures will occur within 10 days, and the majority of the remainder will occur within 2 weeks [45, 46, 47]. Delayed rupture is rare in the pediatric population, and the majority of children with splenic injuries that require surgical intervention will usually declare themselves in the first 12 hours [47].

Grading of Injury

The American Association for the Surgery of Trauma (AAST) has presented a grading system for splenic injury as identified on computed tomography (CT). This grading system helps predict the success of nonoperative management. The grading system goes from grade I for minor injuries to grade V for severe injuries. See Table 42.2.

Clinical Presentation and Physical Exam

An athlete with a splenic injury may present immediately after trauma or may have a delayed presentation. The athlete will likely complain of sharp left-sided pain initially, followed by dull pain. Nausea, vomiting, and the urge to defecate have also been described but are not specific for splenic injury. Kehr's sign, referred pain to the left shoulder or proximal arm, may be present as a result of blood irritating the diaphragm and phrenic nerve. This is a delayed sign usually occurring about 30–60 minutes after the injury and so may not be present on initial evaluation [44].

On physical exam, the athletes should be evaluated for signs of hemorrhagic shock. Vitals signs may show tachycardia and hypotension; however, this may be delayed as 25% of circulating blood volume may need to be lost before hypotension occurs. The skin may be cool and clammy. The athlete will have tenderness to palpation in the left upper quadrant of the abdomen and may have Cullen's or Grey Turner's sign (see description in the "Liver Injury" section) if the injury is severe.

Table 42.2 Spleen injury scale [64]

Grade	Injury	Description
I	Hematoma	Subcapsular, nonexpanding, < 10% surface area
	Laceration	Capsular tear, nonbleeding, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, nonexpanding, 10–50% surface area
		Intraparenchymal, nonexpanding, < 2 cm in diameter
	Laceration	Capsular tear, active bleeding, 1–3 cm parenchymal depth which does not involve a trabecular vessel
III	Hematoma	Subcapsular, > 50% surface area or expanding
		Ruptured subcapsular hematoma with active bleeding
		Intraparenchymal hematoma > 2 cm or expanding
	Laceration	> 3 cm parenchymal depth or involving trabecular vessels
IV	Hematoma	Ruptured intraparenchymal hematoma with active bleeding
	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (> 25% of the spleen)
V	Hematoma	Completely shattered spleen
	Laceration	Hilar vascular injury which devascularizes spleen

The American Association for the Surgery of Trauma (AAST) has presented a grading system for splenic injury as identified on computed tomography (CT), advance one grade for multiple injuries to the same vessel
Table from Moore et al. [64]

Evaluation

The evaluation of a patient with a suspected splenic injury should begin on the sideline. Any athlete that sustains blunt abdominal trauma to the left upper quadrant should have a physical exam including vital signs that should be repeated frequently. As previously discussed, the splenic capsule can

contain initial bleeding masking the severity of the injury. As part of a primary and secondary survey on the sidelines or in the training room, a FAST exam may be done to look for intraperitoneal hemorrhage. The stable patient should be monitored for changes in exam, vital signs, pain, and mentation. Even in a stable patient, if the index of suspicion is high for splenic injury, the athlete should be referred for further evaluation in an emergency department with trauma capabilities (or the nearest emergency department if a trauma center is not available). The unstable athlete should be transferred to a trauma center and managed according to ATLS guidelines.

Once in the emergency department, a CT scan with contrast and laboratory testing including a complete blood count, electrolyte panel, liver function tests, lipase, possible coagulation studies, and blood type and screen should be done. Evaluation by a surgeon should also occur. In athletes that undergo CT scans, there are certain findings that are suggestive for injury. These include hemoperitoneum, hypodensities within the spleen, and a contrast blush or extravasation [45]. MRI can also be done but is not a practical choice to evaluate for splenic injuries given the length of time required for the test and the cost.

Management

Splenic injuries identified on imaging will need to be evaluated by a trauma surgeon for potential intervention versus observation. As in all athletes with blunt abdominal trauma, those who are hemodynamically unstable should be resuscitated per ATLS guidelines with crystalloid and potentially blood products and evaluated by a surgeon as soon as possible. Stable athletes should undergo a CT with treatment dictated by the grade or severity of the injury.

Surgical interventions will likely begin with spleen-salvaging procedures like embolization. If the injury is too severe or the prior has failed, a splenectomy should be performed. In the adult population, 50–70% of cases are managed nonoperatively [48, 49]. In children with splenic injuries, treatment course is dictated more by clinical condition than grading of injury. If the pediatric athlete remains hemodynamically stable (regardless of CT findings), he or she can be observed in a hospital equipped to transition the athlete to operative management if it becomes warranted. One criterion that is cited often for defining surgical intervention states that the need to replace more than half of the athlete's circulating blood is an indication for surgical management [50]. This can be estimated at about greater than or equal to 20 mL/kg of packed red blood cells. Initial surgical intervention may include embolization prior to splenectomy or other spleen-salvaging procedures. Attempts to avoid a splenectomy are driven by the fact that asplenic children are at a much higher risk of infection by encapsulated organisms and will require additional immunizations post-procedure [45].

Repeat imaging in the acute phase will be dictated by the severity of injury. For those athletes being observed, repeat imaging with CT scan is indicated if there is any clinical deterioration, in athletes with higher grade injuries with evolving neurologic injury, or if the initial injury was unclear. On average, athletes will be observed in the hospital for 1–3 days.

Return to Play

Time to return to play will vary according to whether the athlete underwent observation, embolization, or splenectomy. It is common practice to restrict participation of any sport or activity that could result in trauma to the area for up to 3 months. During this time, the athlete may make gradual progression in increasing physical activity while avoiding heavy lifting or strenuous activity. As there are no clinical studies to guide return to play, a recovery plan should be discussed with the surgeon, athletes, and family if applicable.

Diaphragm Injury

The diaphragm is one of the most commonly injured organs in the abdomen. Injuries can range from very minor and very common, as in the case of diaphragm spasm, to very severe and very uncommon, as in the case of diaphragm rupture. Diaphragm ruptures represent less than 1% of all traumatic injuries [51] and should be suspected in athletes presenting with symptoms similar to a diaphragm spasm that do not resolve or with any significant abdominal trauma. This injury can have life-threatening consequences and can be hard to diagnose, so physicians caring for athletes must include this in their working differential as it can be easily missed. Serious diaphragm injuries rarely occur independently. According to the National Trauma Database [51], at least 50% of patients with diaphragm injuries have additional injuries.

Anatomy

The diaphragm is a dome-shaped musculotendinous structure that separates the thoracic cavity from the abdominal cavity and plays a large role in respiratory mechanics. The aorta, inferior vena cava, esophagus, vagus nerve, and phrenic nerve transect the diaphragm in a horizontal plane (Fig. 42.10). It is composed of the costal and crural muscle groups, which are innervated by the phrenic nerve and vagus nerve. The vascular supply comes from the phrenic artery below the diaphragm and the pericardiacophrenic arteries above the diaphragm [28].

Mechanism

Any blunt force trauma to the abdomen can directly or indirectly cause injury to the diaphragm. This injury will usually

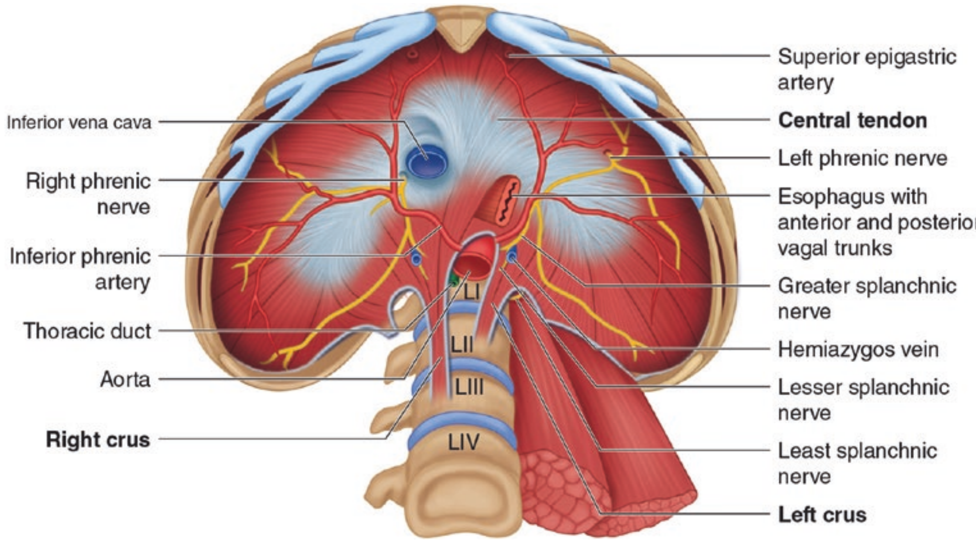


Fig. 42.10 Anatomy of the diagphragm

Table 42.3 Diaphragm injury scale [65]

Grade	Injury	Description
I	Contusion	
II	Laceration	</=2 cm
III	Laceration	2–10 cm
IV	Laceration	> 10 cm laceration with tissue loss </=25 cm^2
V	Laceration	Laceration with tissue loss > 25 cm^2

The American Association for the Surgery of Trauma (AAST) splenic injury grading system, advance one grade for bilateral injuries
Table from Moore et al. [65]

occur after direct contact where an athlete is hit in the epigastrium resulting in temporary spasm of the diaphragm muscle. Injury resulting in rupture may be caused by penetrating trauma or by a direct blow that causes a sudden increase in intra-abdominal pressure that is high enough to overcome diaphragm musculature. The left diaphragm is two to three times more likely to be injured with blunt trauma, which may be due to the fact that the right side is protected by the liver or as a result of a congenital weakness on the left side [52]. A rupture of the diaphragm can result in the acute herniation of abdominal contents into the chest.

Grading of Injury

Classification of diaphragm injuries has been established by the American Association for the Surgery of Trauma (AAST) which is very similar to grading for other organ injuries. And as with injury to other organs, the higher the degree of injury, the higher the associated mortality. See Table 42.3.

Clinical Presentation and Physical Exam

Athletes presenting with a diaphragm injury most commonly describe getting “the wind knocked out of me.” The athlete will complain of difficulty breathing until the spasm has resolved. This will usually last seconds to minutes in the setting of spasm and potentially longer with rupture and herniation. The athlete may also complain of chest pain or flank pain. On physical exam, the athlete will likely have tenderness to palpation over the site of impact and may even develop ecchymosis. The provider may hear decreased breath sounds on the side of injury if a pneumothorax has occurred secondary to herniation. With severe injuries or in delayed presentations, athletes may be hemodynamically unstable and require emergent treatment.

Evaluation

As with all injuries, evaluation should start with a primary and secondary survey. Given that an athlete with a diaphragm injury may complain of both shortness of breath and abdominal pain, a specific chest, pulmonary, and abdominal exam will need to be completed. If the athlete is hemodynamically unstable, he or she should be transported to the nearest trauma center (or the nearest emergency department if a trauma center is not available). In the case of herniation with pneumothorax that has progressed to tension pneumothorax, the athlete should have their chest decompressed with needle decompression as discussed in the thoracic trauma section and according to ATLS guidelines.

In the case of suspected diaphragm injury, sideline or training room bedside ultrasound will not likely be helpful. There is no standardized technique, and a negative study does not rule out a diaphragm injury. If ultrasound is attempted, the diaphragm will show up as a hyperechoic structure and may be evaluated for continuity. Disruption of the continuity is associated with rupture [53, 54]. A plain film of the chest is indicated and may show elevation of the diaphragm where the injury has occurred or even herniation of abdominal contents into the chest in severe injuries. In some cases, X-rays may also be negative. If clinical suspicion is high enough, a computed tomography (CT) scan of

the chest and abdomen should be performed; however, this also may be negative. Signs of diaphragm injury on CT include discontinuity of the diaphragm, herniation of abdominal contents into the chest, visualization of a nasogastric tube (if placed) within the chest, constriction of the bowel, or contiguous injuries from one side of the abdomen to the other (i.e., left pulmonary laceration and splenic laceration) [55]. MRI is also an option, but it is of limited utilization due to availability, cost, and time required to complete the test.

Once transported to a trauma center, the athlete should have basic labs ordered including a complete blood count, complete metabolic panel including liver function tests, lipase, and possibly a type and cross or screen for blood products.

A trauma surgeon should evaluate all hemodynamically unstable patients as well as all patients with a high index of suspicion for diaphragm injuries. When imaging studies have been inconclusive but injury is still suspected, more invasive procedures such as an exploratory laparotomy or laparoscopy may be required to diagnose and repair diaphragm injuries.

Management

Treatment for the minor diaphragm injuries can include hip flexion and loosening of restrictive equipment or clothing to ease breathing. If symptoms resolve quickly, no additional testing is required. If symptoms persist, the athlete should be evaluated for hemodynamic stability and for more significant injuries as above. Any athlete with concern for significant injury to the chest or abdomen should be transported to the trauma center (or the nearest emergency department if a trauma center is not available) for further care.

In the case of a suspected or identified diaphragm, rupture management by the surgical team will vary depending on stability, location of injury, and index of suspicion for injury severity. Hemodynamically stable athletes with negative imaging or those with small right-sided defects identified on imaging may be managed expectantly with frequent repeat exams. In unstable athletes or those with a larger injury identified on imaging, an exploratory laparoscopy or laparotomy will likely be required. Smaller diaphragm injuries will be repaired primarily by the surgeon, whereas larger injuries may require prosthetic material to close the diaphragm.

Mortality in diaphragm injuries is high, around 25% [51] due to both the injury itself and the associated complications. Complications vary depending on the type of trauma (penetrating vs. blunt) but include gastrointestinal herniation, altered respiratory mechanics, diaphragm paralysis due to nerve injury, pleural fistula, and ischemic bowel.

Return to Play

In the case of a diaphragm spasm, the athlete may return to the field of play once symptoms have resolved. In the case of more significant diaphragm injury such as rupture, the athlete will need to be cleared by the surgeon who repaired the injury. Overall rates of recurrence are low, but some sources suggest that athletes should have periodic chest X-rays and pulmonary function testing after a repair. No timeline is specified [56]; however, it is generally recommended that periodic testing is only indicated if the athlete has complaints such as chest pain or shortness of breath. Return to play will be influenced by the recovery time for the laparotomy as much as the repair of the diaphragm injury itself. An average timeline of return to strenuous activity will likely be 6–8 weeks but may be further dictated by the surgeon who repaired the defect.

Abdominal Wall Contusion

Contusion of the abdominal wall musculature is another common sports-related injury. A subsequent hematoma may form especially when injuries occur to the epigastric arteries and cause bleeding within the rectus abdominis sheath.

Anatomy

The abdominal wall musculature is composed of the external obliques, the internal obliques, the rectus abdominis, and the transversus muscles (Fig. 42.11). The majority of the blood supply comes from the inferior and superior epigastric arteries (Fig. 42.12). These vessels can be damaged during trauma and contribute to hematoma formation.

Mechanism

Injury to the abdominal wall musculature occurs as a result of either direct trauma or forceful contraction of the musculature. A contusion and hematoma may subsequently form. The rectus abdominis musculature is at a higher risk for hematoma formation given the anatomical location of the epigastric arteries.

Clinical Presentation and Physical Exam

In completing an assessment and physical exam of an athlete with a suspected abdominal wall contusion or hematoma, the athlete will likely complain of acute-onset pain occasionally with nausea and vomiting. If there is hematoma formation, rapid swelling in the area will occur. In the case of a rectus sheath hematoma, a mass below the umbilicus may be palpable. This injury can mimic an acute abdomen.

Evaluation

Any injured athlete should be screened for hemodynamic stability and injury by a primary and secondary survey. The injured area should have overlying clothing or equipment removed for full visualization. Inspection, auscultation, palpation, and percussion of the area should be done.

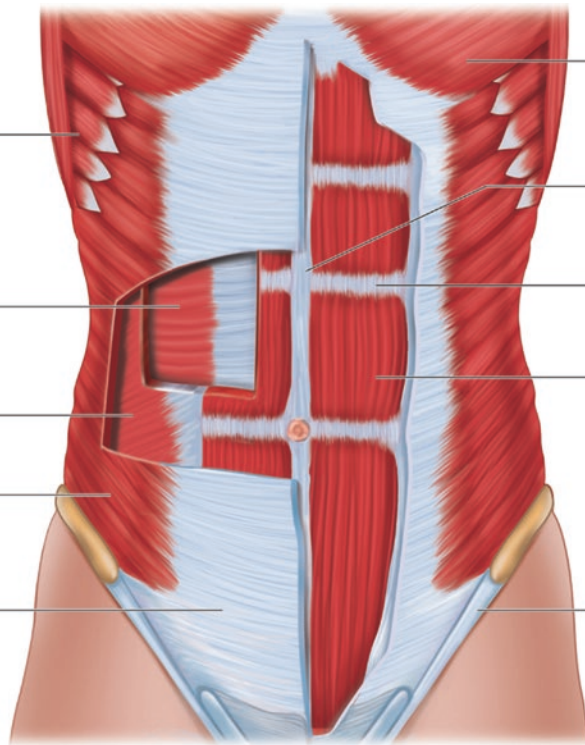
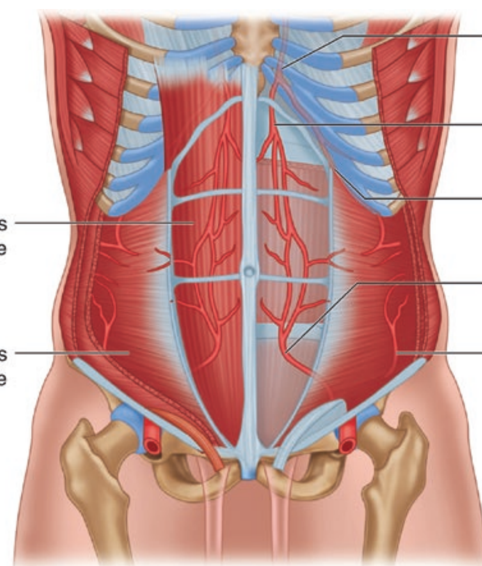


Fig. 42.11 Musculature of the abdominal wall

Fig. 42.12 Arterial supply on the abdominal wall



Abnormalities on exam should prompt further evaluation, and any hemodynamic instability should prompt transportation to a trauma center.

Ultrasound can be used both on the sideline and in the training room to screen for hematomas of the abdominal wall. On ultrasound imaging, the area of injury will reveal a hypoechoic area. The physician can also use ultrasound to follow the injury for improvement and eventually resolution of the hematoma. Computed tomography (CT) scan can also be used to confirm hematoma size and location and evaluate for additional injuries. Repeat CT is often not necessary.

Management

Athletes with a suspected abdominal wall contusion or hematoma are unlikely to be unstable if the injury occurs independently of other injuries. However, as with any other abdominal injury, a primary and secondary survey should be performed to screen for additional injury. The hematoma can

be monitored for progression of size by repeat physical exam and ultrasound. Movement that increases pain should be avoided initially.

Treatment

Treatment of an abdominal wall contusion or hematoma initially includes ice, rest, and analgesics. Activities that include flexion or rotation of the core as well as stretching of the core should be avoided until the pain is resolved. In the case of a large hematoma, surgical evacuation and potentially ligation of the epigastric artery may be required. After the initial pain has resolved, rehabilitation should include regaining flexibility and core strength.

Return to Play

An athlete may return to play after an abdominal wall contusion or hematoma once he or she is able to complete functional testing. Goals should include returning to normal range of motion, preinjury strength, and preinjury flexibility. Returning an athlete to the field of play before these goals have been achieved may put him or her at risk for further injury.

Kidney, Ureter, and Bladder Injuries

Genitourinary injuries seldom occur in isolation and are rarely life-threatening with few exceptions. Injuries to the genitourinary system often occur in the setting of a pelvic fracture or significant abdominal trauma. Providers should be aware of possible coexisting injuries and manage the athlete accordingly. Injuries to be discussed in this section include those to the kidneys, ureters, and bladder.

Anatomy

The genitourinary system can be divided up into the upper and lower tracts with the upper tract consisting of the kidneys and ureters and the lower tract consisting of the bladder, urethra, and external genitalia (Fig. 42.13).

The kidneys and the ureter lie within the retroperitoneal space. These organs are protected by the surrounding tissue which includes the lower ribs specifically T10 through T12, the musculature of the spine including the paraspinal musculature and the psoas muscle, and the perinephric fat. Due to the location and size of the liver, the right kidney extends lower than the left.

The ureters course distally after they exit the kidney pole and enter the bladder posteriorly and inferiorly at the trigone.

The bladder's location within the pelvis varies depending on how distended it is. When it is empty, the bladder will sit along the pelvis floor, but when fully distended, it may extend to the level of the umbilicus. The peritoneal surface of the dome is the weakest point and most mobile point of the bladder.

Mechanism

As with all abdominal injuries in sports, the mechanisms that are most commonly associated with injury are blunt trauma and deceleration. Occasionally penetrating trauma can occur

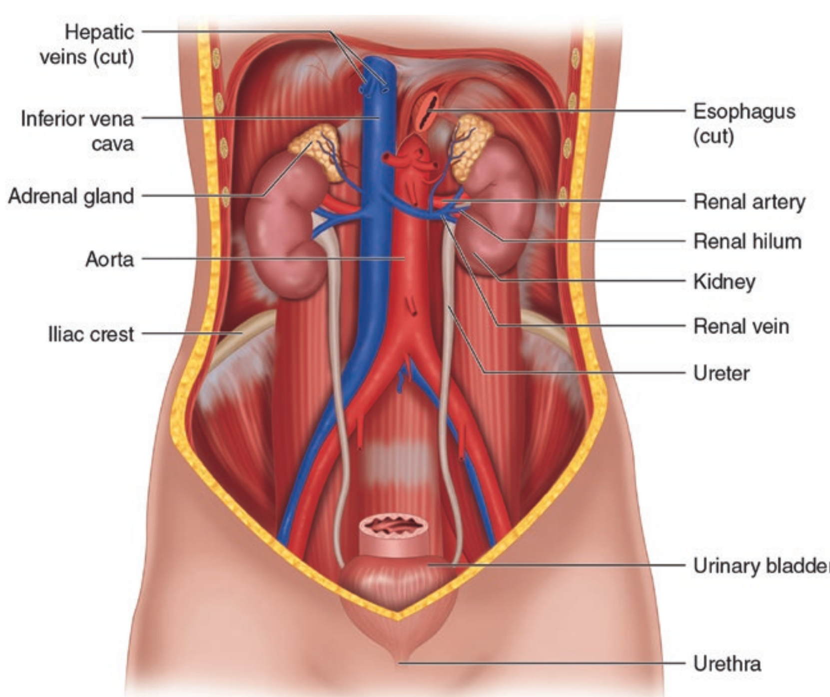


Fig. 42.13 Anatomy of the genitourinary system

although this is rare in sports. In the case of deceleration injuries seen in falls from great heights or motorsports, the athlete may sustain avulsion of renal pedicle, renal artery dissection, or avulsion of the ureter at the ureteropelvic junction. Blunt force injuries, which tend to occur in contact sports athletes or falls from lower heights, can result in a shattered kidney, bladder rupture, or injury to the external genitalia. Injuries to the lower genitourinary tract can also be caused by or in conjunction with pelvic fractures.

Grading of Injury

The American Association for the Surgery of Trauma Organ Injury Severity Score for the Kidney, Ureter, and Bladder [57, 58] has stratified injury according to a scale that correlates needs for surgical intervention. All of the organs listed have their own scoring scale. See Tables 42.4, 42.5, and 42.6.

Table 42.4 Kidney injury scale [64]

Grade	Injury	Description
I	Contusion	Microscopic or gross hematuria, urologic studies normal
	Hematoma	Subcapsular, nonexpanding without parenchymal laceration
II	Hematoma	Nonexpanding perirenal hematoma confirmed to renal retroperitoneum
	Laceration	< 1.0 cm parenchymal depth of renal cortex without urinary extravasation
III	Laceration	> 1.0 cm parenchymal depth of renal cortex without collecting system rupture or urinary extravasation
IV	Laceration	Parenchymal laceration extending through renal cortex, medulla, and collecting system
	Vascular	Main renal artery or vein injury with contained hemorrhage
V	Laceration	Completely shattered kidney
	Vascular	Avulsion of renal hilum which devascularizes kidney

The American Association for the Surgery of Trauma (AAST) Organ Injury Severity Score for the kidney, advance one grade for bilateral injuries, advance one grade for multiple injuries to the same vessel
Table from Moore et al. [64]

Table 42.5 Ureter injury scale [66]

Grade	Injury	Description
I	Hematoma	Contusion or hematoma without devascularization
II	Laceration	< 50% transection
III	Laceration	> 50% transection
IV	Laceration	Complete transection with 2 cm devascularization
V	Laceration	Avulsion with > 2 cm of devascularization

The American Association for the Surgery of Trauma (AAST) Organ Injury Severity Score for the ureter, advance one grade for bilateral injuries, advance one grade for multiple injuries to the same vessel
Table from Moore et al. [66]

Table 42.6 Bladder injury scale [66]

Grade	Injury	Description
I	Hematoma	Contusion, intramural hematoma
	Laceration	Partial thickness
II	Laceration	Extraperitoneal bladder wall laceration < 2 cm
III	Laceration	Extraperitoneal (≥ 2 cm) or intraperitoneal (< 2 cm) bladder wall lacerations
IV	Laceration	Intraperitoneal bladder wall laceration ≥ 2 cm
V	Laceration	Intraperitoneal or extraperitoneal bladder wall laceration extending into the bladder neck or ureteral orifice (trigone)

The American Association for the Surgery of Trauma (AAST) Organ Injury Severity Score for the bladder, advance one grade for bilateral injuries, advance one grade if multiple lesions exist
Table from Moore et al. [66]

Clinical Presentation and Physical Exam

An athlete with an injury to the genitourinary system will have complaints and a physical exam that varies by the location of the injury. In general, athletes with upper tract injuries may present with complaints of flank pain or hematuria. Athletes with lower tract injuries and specifically bladder injuries will present with complaints of suprapubic pain, gross hematuria, and difficulty or inability to void.

While conducting a physical exam, the provider should be aware that injuries to the genitourinary tract rarely occur in isolation, and so a thorough physical exam should be performed. The provider should look for bruising to the flank and abdomen such as Grey Turner's sign and Cullen's sign. He or she may also need to perform a visual inspection of the genitals for signs of bleeding or trauma. The provider should then palpate for costovertebral angle tenderness and flank pain as well as palpate the abdomen for distention, guarding, and rebound tenderness. As ureteral injuries are often missed on initial evaluation, delayed findings include persistent flank pain, fever, and a palpable flank mass that may represent a urinoma. The pelvis should be assessed for possible fractures as two-thirds of significant bladder injuries have co-occurring pelvic fractures [59, 60]. This can be done through palpation and compression as well and evaluation of the hips.

Special consideration needs to be given to pediatric athletes. Injuries to the renal system are more common in children as their kidneys are larger in comparison to their abdomen and pelvis so the surrounding organs and rib cage provide less protection.

Evaluation

As in previous discussions, sideline evaluation should begin with a thorough primary and secondary survey according to ATLS protocols. In the case of genitourinary injuries, complaints from athletes may be nonspecific so providers need to take into consideration the mechanism and type of trauma to have an appropriate level of suspicion for injuries to these

organs. Vital signs should be taken and repeated looking for signs of shock. All unstable patients should be urgently transported to the nearest trauma center (or the nearest emergency department if a trauma center is not available).

Any athlete with a suspected genitourinary injury should have an evaluation of his or her urine for the presence of hematuria. The quantitative measurement of hematuria can be accomplished by a chamber count or the number of red blood cells per milliliter of excreted urine, sediment count or the direct examination of a centrifuged sample, or indirect examination with a dipstick which is the simplest and least specific way to evaluate hematuria. Hematuria found by dipstick should always be confirmed by microscopic evaluation of the sediment [61].

Hematuria is then categorized into gross hematuria and microhematuria. In general, gross hematuria can be seen by the naked eye and is urine that is not clear or yellow. Although the definitions vary, microscopic hematuria can be defined as greater than or equal to three to five red blood cells (RBC) per high-powered field (hpf) [60]. Assessment for hematuria is extremely important in athletes with possible genitourinary injuries as gross hematuria is a cardinal sign of bladder injury and is reportedly present in greater than 66% [61] to 95% [30] of bladder injuries. Microhematuria will be present in almost all the remaining cases. Neither gross nor microhematuria is a sensitive marker of ureteral injuries as approximately 25% of individuals with ureteral injuries have a normal urinalysis [60]. That being said, hematuria should raise concern for genitourinary injury.

In addition to urinalysis, a complete blood count and complete metabolic panel including liver function tests should be ordered. Providers may also consider lipase, blood type, and coagulation studies depending on the presentation of the athlete.

The diagnostic test of choice for suspected renal injury is a CT scan with IV contrast. It is indicated in blunt abdominal trauma when gross hematuria or microhematuria with shock is present (only once the patient has been stabilized). Other indications for imaging include a fall from height, significant deceleration, or other clinical features of renal trauma (flank pain or contusion). If there is concern for injury to the collecting system, delayed CT images will be needed. The indications listed above are the same for both pediatric and adult athletes.

CT scans have largely replaced intravenous pyelography for renal trauma [62, 63]. A CT cystogram with retrograde filling of the bladder is the test of choice in suspected bladder injuries [62]. Specific indications to perform this test include gross hematuria or the combination of a pelvic fracture with microhematuria.

Ultrasound can be used to visualize free fluid surrounding the intraperitoneal bladder as part of the FAST exam; however, this modality cannot differentiate blood from urine. Ultrasound can also be used to evaluate the kidneys; however, there is a lack of sensitivity in the setting of trauma, and therefore it cannot be used to exclude injury [63].

X-rays do not have a role in identifying genitourinary injuries but may have a role in trauma evaluations. Fractures of the posterior ribs or spine fractures should raise the clinician's suspicion for upper tract injuries (kidney, ureter). Any pelvic fracture seen on imaging should raise the providers' suspicion for bladder and urethra injury in the athlete. Fractures that include the anterior arch or all four rami should increase suspicion for bladder injury. In addition, fractures of ipsilateral rami that co-occur with posterior displacement of the sacrum, sacroiliac joint, or the ilium should also raise concern for urethral injuries.

Management

Treatment for blunt abdominal trauma that results in injuries to the genitourinary system depends on the type and severity of the injury according to the American Association for the Surgery of Trauma Organ Injury Severity Score [57].

Any unstable athlete should be emergently evaluated by a trauma surgeon. Athletes in whom an injury to the kidney, ureter, or bladder is identified will also need urgent evaluation and possible intervention by urology. Foley placement is advised to track urine output, except in the setting of urethral injury.

Grade I and II renal injuries are usually managed nonoperatively, while grade V injuries usually require a nephrectomy. Renovascular injuries can be life-threatening and will require immediate evaluation and intervention. If such an injury is suspected, a renal arteriogram may be obtained, but this is a specialized test that may not always be available. Treatment of a renovascular injury may include operative revascularization, nephrectomy, endovascular stenting (in the case of arterial dissection), and observation [60].

Ureteral injuries may require a nephrostomy tube or possible stenting in the setting of transection by urology. Management of bladder injuries also varies by severity and location. Operative repair is often required for intraperitoneal bladder ruptures, but most retroperitoneal injuries are managed nonoperatively. In such injuries, a suprapubic catheter or Foley catheter can be placed to provide drainage of urine to keep the bladder decompressed while the injury heals [60].

In both adult athletes without gross hematuria and pediatric athletes who have microhematuria of 50 RBC/hpf or fewer who do not exhibit signs of shock, no intervention is required. These athletes should be followed by a urologist as outpatients until the microhematuria has cleared [30].

Return to Play

In athletes who have no major injury identified but have simple microhematuria, return to play and return to training are

determined by the resolution of his or her hematuria and clearance from urology. In athletes with more significant injuries, they may not return to play until they have been cleared by urology. Prior to return to play, all athletes must exhibit normal renal function and voiding. There is some literature that suggests that significant renal trauma may result in chronic hypertension. At this time, there are no specific recommendations outside of normal, routine blood pressure screening in athletes once he or she has been cleared. If the athlete is found to have persistent hypertension, appropriate treatment should occur based on the level of elevation.

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43 Head Injury

Christopher Guyer and Jeremiah W. Ray

Key Points
- Physicians should consider using clinical decision rules when determining the need for CT in athletes with traumatic head injuries.
- There are multiple methods of diagnosing a concussion but no universally accepted standard. Neuroimaging is utilized to detect a skull fracture and/or intracranial hemorrhage but has no role in concussion diagnosis.
- Return-to-play guidelines for athletes having sustained intracranial hemorrhage are based on expert opinion.

Introduction

Head injury is a frequent cause of death in sport [1]. Head injuries occur as a result of athletes colliding with other players and objects. These impacts produce translational (linear) or rotational (angular) forces that lead to a variety of clinical pathology [2]. The brain is a particularly important organ because it is incapable of regeneration and transplantation is not an option. Head injuries can lead to dementia, seizure, paralysis, and death.

Historically, football participation has caused the highest number of fatalities due to the number of athletes participating. Other high-risk sports include but are not limited to horseback riding, skydiving, motorcycle and car racing, gymnastics, ice hockey, boxing, martial arts, and rugby. Football is a high-profile sport and receives a significant amount of media attention and reports in medical literature. Rule changes, along with improvements in helmet technology and research into head injury biomechanics, have led to fewer catastrophic sports-related head injuries [3, 4]. A recent review estimates the number of catastrophic sports-related injuries to be 0.60 per 100,000 participants per year among high school athletes [5].

C. Guyer (✉)
Divisions of Emergency Medicine and of Sports Medicine, Henry Ford Health System, Detroit, MI, USA
e-mail: cguyer@wayne.edu

J. W. Ray
Department of Orthopedic Surgery, University of California at Davis Medical Center, Davis, CA, USA
e-mail: jwray@ucdavis.edu

Anatomy

The scalp is the outermost layer of the head and has five layers: the skin, subcutaneous tissue, galea, areolar tissue, and pericranium. The scalp has a significant blood supply, and injury can result in extensive blood loss. The skull is rigid and made up of eight bones: two temporal bones, two parietal bones, the sphenoid, ethmoid, and frontal bones. In adults, expandability is limited. Cranial nerves and blood vessels enter the basilar skull (Fig. 43.1).

The brain is made up of two cerebral hemispheres, the cerebellum, and the brainstem. Each hemisphere is divided into lobes: frontal, temporal, parietal, and occipital. The outermost layer covering the brain is the dura mater. Underneath this is the arachnoid mater, a thinner connective tissue layer. The innermost layer is called the pia mater. Cerebrospinal fluid is contained in the subarachnoid space that separates the arachnoid and the pia. Several subarachnoid spaces surround the brain. Within the brain are two lateral ventricles, a third ventricle, and a fourth ventricle. The ventricles communicate by several foramina (Fig. 43.2).

Spectrum of Injury

Head injuries can be classified based on their anatomic location (epidural, subdural, subarachnoid, and intraparenchymal), the mechanism of injury (coup, contrecoup, linear, rotational), the distribution (focal or diffuse), and clinical presentation. Catastrophic injuries are defined by the

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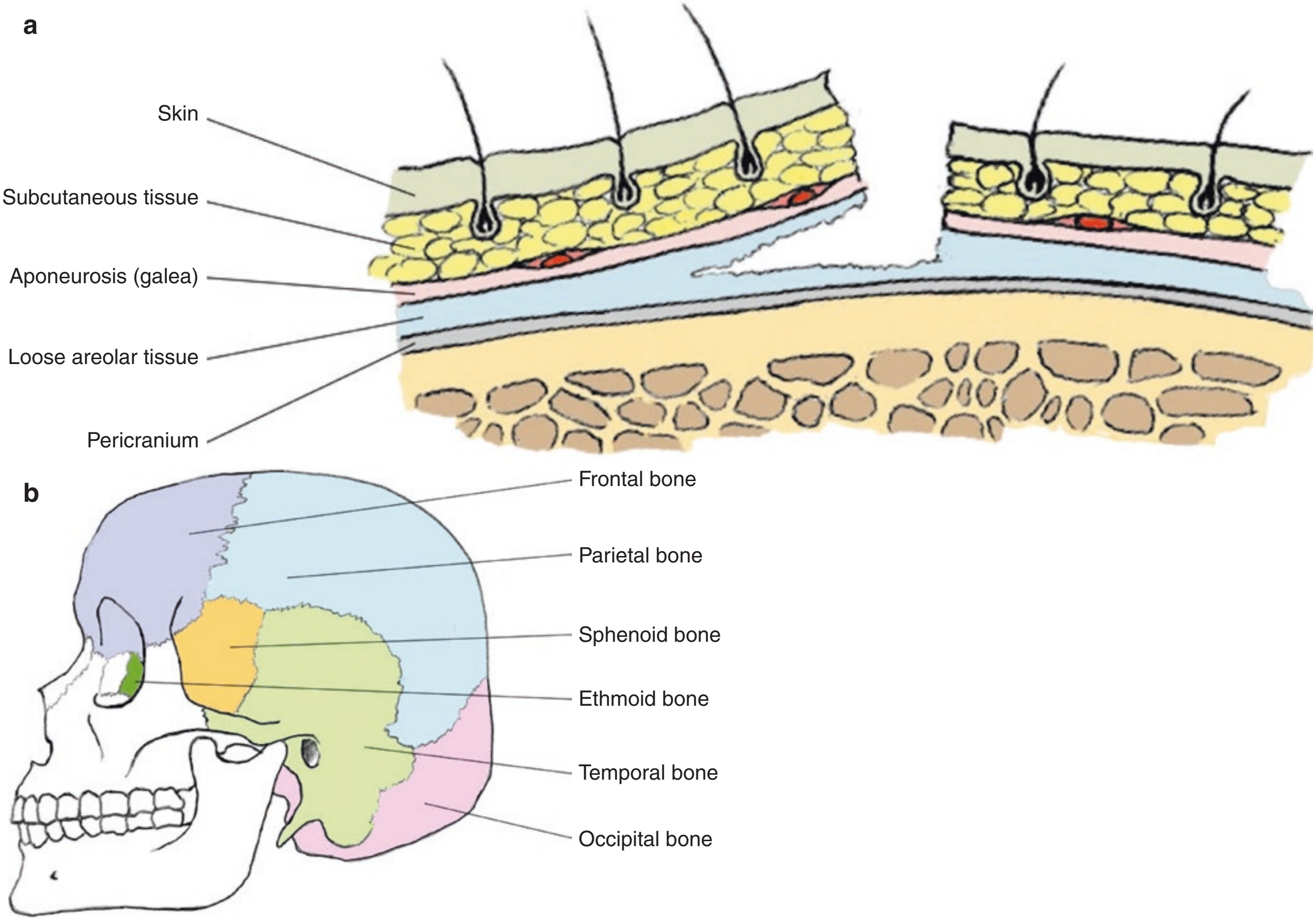
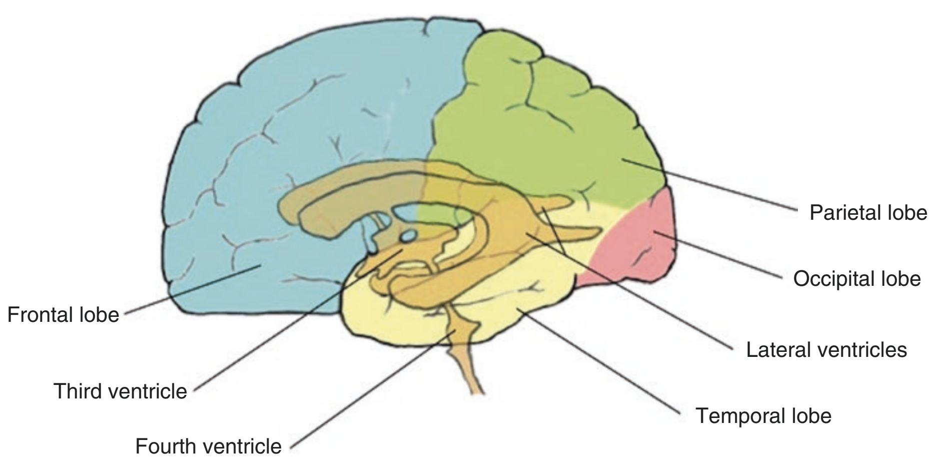


Fig. 43.1 Scalp and skull anatomy

Fig. 43.2 Brain anatomy



National Center for Catastrophic Sports Injury Research as either direct (injury experienced as a result of participating in skills of the sport) or indirect (systemic failure secondary to exertion while participating in a sport). Injuries are further classified as fatal (injury causes death to the athlete), nonfatal (injury causes a permanent neurological functional disability), or serious (no permanent functional disability) [2].

Pathophysiology

Factors that affect cerebral blood flow include autoregulation, cerebral perfusion pressure (CPP), mean arterial pressure (MAP), and intracranial pressure (ICP). Autoregulation normally regulates cerebral blood flow to achieve equilibrium between oxygen delivery and metabolism. Autoregulation

adjusts CPP from 50 to 150 mmHg for maintenance of oxygen demands and regional blood flow. CPP < 60 mmHg is the lower limit of autoregulation. At this low of a pressure, local control of cerebral blood flow cannot be adjusted to maintain adequate flow for function. In head-injured athletes, it is important to maintain a MAP of ≥ 80 mmHg because low blood pressure with elevated ICP results in low CPP and brain injury.

The cranium is a closed space with a fixed volume. Volume changes such as bleeding increase the ICP and decrease the CPP. The volume of the brain parenchyma, cerebrospinal fluid, and intravascular blood determines the ICP. If one of these compartments expands, a reduction in another must occur or ICP will increase. ICP elevations can be life-threatening and can lead to a response called the Cushing reflex, a triad of hypertension, bradycardia, and respiratory irregularity [6].

Acute traumatic brain injuries can be divided into two phases: primary and secondary. In the primary phase, cellular injury occurs as a result of the force of injury. Primary injuries include contusions, hematomas, diffuse axonal injury, direct cellular damage, loss of the blood-brain barrier, disruption of neurochemical homeostasis, and loss of the electrochemical function. Injury prevention and mitigation are the only ways to reduce primary cell death.

Secondary brain injury occurs as a result of the massive release of neurotransmitters, also known as the secondary neurotoxic cascade. Cytoplasmic and nuclear enzymes are activated by ionic shifts and cause mitochondrial damage. This in turn leads to cell death and necrosis. Proinflammatory cytokines and additional enzymes are released as the body tries to repair damaged cells. Secondary injury is nonselective, and extensive neuron loss can occur. Many cells will also undergo apoptosis, and this can occur even longer than a year after the initial injury [6].

Secondary insult is a clinical term used to describe conditions and circumstances that speed up neurotoxic damage and worsen outcomes. Examples include hypotension, hypoxemia, and hyperglycemia. Prevention of hypoxia and ischemia is the major treatment goal [6].

Brain edema occurs from two processes. Cytotoxic edema is caused by large ionic shifts as a result of loss of cellular membrane integrity that occurs from damage to mitochondria. Extracellular edema occurs from the breakdown of, or direct trauma to, the blood-brain barrier, ionic shifts, and changes to water exchange mechanisms. The brain swells and ICP increases in response to increased intracellular and extracellular water content. This leads to direct compression of tissues, vascular compression ischemia, herniation, and brain death.

Initial Evaluation and Management on the Field

When an athlete is "down" after sustaining a head injury, they require immediate assessment of their level of consciousness. Treatment for an unconscious athlete should follow Advanced Trauma Life Support protocols. If the athlete is facedown or on their side, they can be cautiously logrolled into a supine position while maintaining in-line stabilization of the cervical spine. Evaluation of their airway and respiratory effort is a primary concern.

For athletes wearing a helmet, if airway intervention is required or there is need for respiratory support, the facemask can be removed to allow for access. The helmet should be removed if facemask removal is inadequate for airway access. A minimum of three trained rescuers is needed, and they should follow a team approach for removal of the helmet. The neck must be manually immobilized, and sequential removal of other protective gear, such as shoulder pads, should occur at the same time as helmet removal.

Glasgow Coma Scale is a widely accepted and useful scoring tool for rapid assessment of trauma patients (Table 43.1). The use of this scale allows providers to communicate the condition of the patient by rating their eye, verbal, and motor responses. Injuries can be classified as severe (GCS score of 3–8), moderate (GCS score of 9–13), or mild (GCS score of 14 or 15).

The next step in assessing the athlete is to perform a pupillary exam to evaluate for size and symmetry. Responses to verbal and noxious stimuli should be noted. Athletes with prolonged loss of consciousness will need to be prepared for immediate transport to a hospital with neurosurgical services. CT scanning will need to be performed to evaluate for intracranial hemorrhage.

Equipment removal should be performed by those with the highest level of training. This may occur on the field in a prehospital setting or in the emergency department. In a recent statement from the National Athletic Trainers' Association, they recommended that when appropriate, protective equipment may be removed prior to transport of the

Table 43.1 GCS score

Glasgow Coma Scale	
Eye opening	
4	Spontaneous
3	To speech
2	To pain
1	No response
Verbal response	
5	Alert and oriented
4	Disoriented conversation
3	Speaking but nonsensical
2	Moans or unintelligible sounds
1	No response
Motor response	
6	Follows commands
5	Localizes pain
4	Moves or withdraws to pain
3	Decorticate flexion
2	Decerebrate extension
1	No response

athlete. Often the sideline sports medicine team has the most experience with equipment removal than other medical team members or hospital emergency staff [7]. If the helmet is left on, it can be taped to a rigid immobilization device. CT scanning can still be performed with a helmet, facemask, and shoulder pads in place.

Brain contusion or intracerebral hematoma will usually cause headache. Neurologic deficits can also occur. These injuries may lead to a seizure. If an athlete is seizing, they can be logrolled onto their side to allow for blood and saliva to escape from the side of the mouth and the nose and to prevent the tongue from obstructing the airway. An oral airway or nasal trumpet can be placed if needed, but providers should not place objects or fingers into the athlete's mouth as amputation can occur. Traumatic seizures will usually last 1–2 minutes. These athletes will need transportation to the hospital for further evaluation.

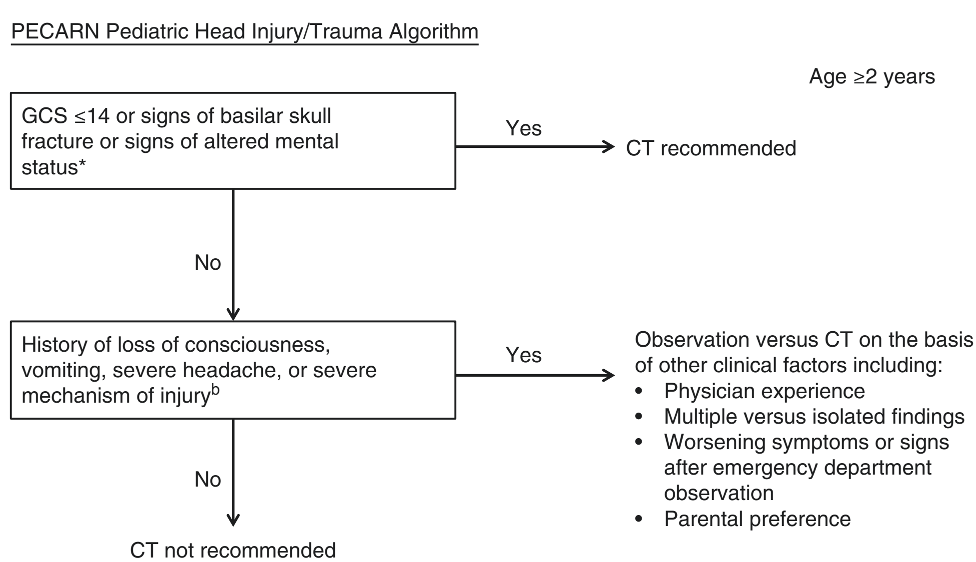
Athletes who are awake can be evaluated on the sidelines for signs and symptoms of head injury. Providers should note level of consciousness, memory, speech, coordination, reflexes, vision, concentration, and gait. Symptoms of head injury include headache, nausea, vomiting, confusion, dizziness, photophobia, irritability, and amnesia. Athletes with symptoms should not return to play. Those with worsening headache, nausea and vomiting, or decreased level of consciousness will need to be transported to the hospital [8]. Any athlete that sustained loss of consciousness should be observed closely for worsening symptoms for at least 24 hours.

Athletes with suspected skull fracture or intracranial hemorrhage will need CT scanning. CT allows for quick diagnosis of fractures and the location, type, and extent of bleeding. This modality can help with making decisions for operative intervention. Reliable clinical decision tools have been developed to assist providers in determining the need for CT scan in head-injured patients. For athletes 16 years of age and older, the Canadian CT Head Rule and New Orleans Criteria (Table 43.2) were developed. Prospective validation studies demonstrate 100% sensitivity and 60–76% specificity for the Canadian CT Head Rule. The New Orleans Criteria has been validated at 82–100% sensitivity and 12–26% specificity. The PECARN Pediatric Head Injury/Trauma Algorithm (Table 43.3) is useful for determining the need for CT in athletes younger than 16 years old. This algorithm has a validated sensitivity of 100% and 53–60% specificity for pediatric patients 2 years or older.

Table 43.2 Canadian CT Head Rule and New Orleans Criteria

Canadian CT Head Rule
Can be used to clear head injury without imaging in patients with GCS of 13-15 and at least one of the following: witnessed loss of consciousness, amnesia to the head injury event, or witnessed disorientation
CT of the head is indicated if any of the criteria below are present **Medium-risk criteria** Retrograde amnesia >30 min Dangerous mechanism of injury (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, or fall from >3 feet or >5 stairs) **High-risk criteria** GCS score <15 for >2 hours Suspected open or depressed skull fracture Suspected basilar skull fracture Emesis ≥2 times >65 years of age
If any of the following exclusion criteria are present, then the Canadian CT Head Rule does not apply: age <16 years, use of blood thinners, seizure after injury
New Orleans CT Head Criteria
Can be used in patients with head injury and loss of consciousness who are neurologically normal (GCS of 15 and normal brief neurological exam)
CT of the head is indicated if any of the criteria below are present Age >60 years Headache Vomiting Drug or alcohol intoxication Seizures Visible trauma above the clavicle Persistent anterograde amnesia

Table 43.3 PECARN pediatric head injury/trauma algorithm
[a]Agitation, somnolence, repetitive questioning, or slow response to verbal communication
[b]Motor vehicle crash with patient ejection, death of another passenger, or rollover; pedestrian or bicyclist without helmet struck by a motorized vehicle; falls of more than 1.5 m/5 ft; head struck by a high impact object



Concussion

Mechanism of Injury in Sports

A concussive event results from acceleration, deceleration, or rotational forces transmitted to the brain, which do not necessitate direct trauma to the head [9]. This mechanism induces diffuse axonal stretching, which mechanically disrupts cell membranes and causes an unregulated efflux of ions [10]. It is this rapid depolarization that results in the release of myriad neurotransmitters, predominately excitatory amino acids such as glutamate [11]. The Na^+/K^+ ATP-dependent pump activates in order to reestablish proper ion homeostasis but quickly depletes intracellular glucose [12, 13]. The glucose depletion leads to mitochondrial dysfunction and inadequate oxidative metabolism, resulting in subsequent anaerobic metabolism and lactate accumulation [14–16]. Subsequent cerebral hypoperfusion, followed by hyperperfusion and edema, can occur in severe cases of traumatic brain injury [17]. There have been attempts to measure the force in newtons applied to the cranium to better identify high-risk events; however, accelerometers (in helmets and mouthpieces) for the assessment of concussion that report peak linear and rotational acceleration are not ready for clinical use and are an area of active research [18].

Epidemiology

In the United States, concussion in sports affects approximately 1.6–3.8 million athletes a year [19–21]. A concussion is also called a mild traumatic brain injury. It is important to note that although not all traumatic brain injuries are concussions, all concussions are mild traumatic brain injuries [22]. The diagnosis and management of concussion in sports is an evolving field of medicine and not without controversy. Several governing bodies have provided position statements and high-quality reviews on the topic [9, 22–27]. The American Medical Society for Sports Medicine (AMSSM) defines a concussion as “a traumatically induced transient disturbance of brain function” [22]. Various definitions for concussion exist, and to date there is no standardized objective manner for diagnosis.

Classification

There is no classification system for concussions, and experts no longer grade concussions.

Clinical Presentation

A significant challenge in making the diagnosis of concussion is the widely variable time of symptom onset post-impact. Some athletes will have symptoms immediately, whereas other athletes may have gradual onset of symptoms over the course of 24–48 hours. It is possible that immediately post injury, the athlete may experience some degree of headache, head pressure, confusion, fogginess, and/or difficulty concentrating [28]. Memory difficulties, a vacant stare, and repetitive questioning are common. Athletes may experience blurred vision, double vision, photophobia, balance, and coordination difficulty. A loss of consciousness occurs in less than 10% of concussions, leaving the majority of concussions to occur without a loss of consciousness [27]. In the event of head trauma, a loss of consciousness requires transient electrochemical dysfunction of the reticular activating system caused by rotational forces exerted upon the midbrain [9]. A loss of consciousness was historically considered a marker of concussion severity but more recently was shown not to strictly correlate with severity but rather point to a specific region of the brain that received the forces [29]. Minutes to hours after the injury, the athlete may report photophobia, nausea, vomiting, and somnolence. In the hours to days after the injury, the athlete may describe irritability, difficulty sleeping, loss of appetite, fatigue, mood lability, and anxiety.

Diagnosis (Table 43.4)

Table 43.4 Sport concussion signs and symptoms

Signs
Loss of consciousness or moment of lying motionless on the playing surface
Balance difficulties
Gait difficulties
Motor incoordination: stumbling, slow, or labored movements
Disorientation or confusion or an inability to respond appropriately to questions
Blank or vacant look
Symptoms
Headache
“Pressure in head”
Neck pain
Nausea or vomiting
Dizziness
Blurred vision
Balance problems
Sensitivity to light
Sensitivity to noise
Feeling slowed down
Feeling like “in a fog”
“Don’t feel right”
Difficulty concentrating
Difficulty remembering
Fatigue or low energy
Confusion
Drowsiness
More emotional
Irritability
Sadness
Nervous or anxious
Difficulty sleeping

Initial Management

It is imperative that if there is any concern for a concussive event, the athlete is pulled from competition immediately. Prior to concussion assessment, evaluate the athlete for the presence of adequate cardiopulmonary function and a patent airway. Next, evaluate for cervical spine injury, skull fracture, intracranial hemorrhage, and ocular globe rupture. These conditions necessitate immediate emergency medical services. Once life- and limb-threatening injuries have been excluded, proceed to your assessment for a concussion. There are multiple concussion assessment tools available, each with benefits and downsides. A well-validated and commonly utilized concussion assessment tool by sports practitioners across the world is the Sport Concussion Assessment Tool or SCAT, which is currently in its fifth edition as of 2017 [30]. The SCAT-5 can be downloaded for free from the *British Journal of Sports Medicine* website (http://bjsm.bmj.com/content/bjsports/early/2017/04/26/bjsports-2017-097506SCAT5.full.pdf). The SCAT-5 can be utilized independently or in tandem with other sport concussion diagnostic modalities such as eye tracking, vestibular-ocular motor screen (VOMS), and neuropsychiatric testing (with or without computerized administration).

Indications for Emergency Department Referral

There are well-validated clinical decision tools to help the provider make evidence-informed decisions on whether or not imaging is clinically indicated. In children, we recommend using the PECARN [31] clinical decision rule, and for adults both the Canadian CT Head Rules and the New Orleans CT Head Rules are well validated [32]. It is important to mention that CT scans and magnetic resonance images of the brain do not diagnose concussion and are only clinically indicated when there is concern for intracranial hemorrhage (subdural hematoma, subarachnoid hemorrhage) or skull fracture. Magnetic resonance imaging (MRI), specifically, MRI diffusion tensor imaging (DTI), is a useful tool in concussion research but at this time has no clinical indication in the concussion setting [33, 34]. Any focal neurologic deficits require immediate neurological imaging. Any concern for cervical spine injury, intracranial hemorrhage, skull fracture, or ocular globe rupture mandates immediate referral for emergency services.

Follow-Up Care

To date, there are no standard return-to-play algorithms. Most return-to-play strategies are based on historical practice patterns and not well informed by evidence. This is due to a paucity of data to guide these strategies. Table 43.5 is an example of what a return-to-play strategy might look like. Every athlete should have an individualized, graduated return protocol. The general rule is that the return to activity is gradual and an athlete may not advance to the next stage of activity until he/she is asymptomatic at that activity load. There is some data to suggest that early nonimpact cardiovascular exercise, such as stationary cycling, improves recovery when compared to controls with strict rest [35]. Further supporting early cardiovascular activity is a growing body of observational data that prolonged activity restriction delays recovery [36].

Table 43.5 Graduated return-to-sport strategy

Stage	Goal
Light nonimpact aerobic activity such as a stationary bike for 30–45 minutes	Mild increase in heart rate
Dynamic activity such as plyometrics or weights	Add vestibular-ocular motor challenge
Noncontact training drills	Sports-specific exercise, coordination, and cognitive load
Full-contact practice	Assess functional ability in full sport
Return to play	Play sport

Return to Sports

As previously noted, there is no universally agreed upon return-to-sport protocol. Return-to-sport protocols are highly variable and dependent on local practice patterns. The longstanding cornerstone of concussion management has been rest, encompassing both physical and cognitive rests [27]. More recently, the data supporting this stance has been called into question as this practice is only supported by animal studies, two small observational studies in humans, and biochemical/physiologic theory [37]. The concept of mild physical noncontact physical activity to augment the recovery of a concussion was introduced in 2008 by Majerske et al. who advocate for prompt light cardiovascular activity, such as stationary bike [38]. In 2016, a prospective multicenter trial out of Ontario, Canada, demonstrated that early return to activity within 7 days of concussion halved the rate of post-concussive symptoms when reexamined on day 28 [35]. Although there is broad variation in return-to-play guidelines, the general rule is that a concussed athlete is to be held from activity until asymptomatic at which point they gradually reintroduce activity [28] (Table 43.6).

In the majority of concussed athletes, balance disturbances normalize by about 3 days post injury [39, 40]. Approximately 90% of athletes experience symptom resolution by post injury days 7–10 [41]. Acetaminophen is recommended for headache control if needed as there is a

Table 43.6 Graduated return-to-academics strategy

Stage	Goal	Stage duration
1	Brain rest with no school. Some advocate for complete cognitive rest and do not allow the use of electronic devices, while others allow for these activities strictly as tolerated. The goal of this stage is to allow recovery without any symptom aggravation. This stage may require as little as a few days and a *maximum* of 2 weeks	Advance to Stage 2 when asymptomatic or at 2 weeks regardless of symptoms
2	Initiate gradual return to activity and academic work as tolerated. Begin with 15-minute reading/studying sessions a couple times a day and progress as tolerated	Advance to Stage 3 when asymptomatic or at 2 weeks regardless of symptoms
3	Modified school work. Return to school but with modified curriculum to accommodate needs of the patient such as a modified attendance schedule, modified curriculum, and modified environment as required by the patient	Advance to Stage 4 when asymptomatic or at 2 weeks regardless of symptoms
4	Full school work with minimal adjustments such as the ability to take a rest day if required or spacing out examinations as needed	Advance to Stage 5 when asymptomatic or at 2 weeks regardless of symptoms
5	Full school curriculum without modifications	Indefinite

theoretical risk of bleeding with aspirin and other nonsteroidal anti-inflammatories [22].

The old adage of waking a concussed athlete on the first night of the concussion no longer stands. Observe a concussed athlete for 4 hours after injury, and if she/he remains with a stable neurologic examination and without increasingly severe symptoms, then that athlete may be permitted to sleep without mid-sleep evaluations [31].

Complications

There is a growing body of evidence that links repetitive head trauma with chronic traumatic encephalopathy (CTE) [42, 43]. The neurobehavioral changes noted in contact sports were documented as early as the 1920s when boxers were deemed "punch drunk" and found to have increased rates of parkinsonism, dysarthria, and psychiatric disturbances [44]. There is a growing evidence to suggest that the "sloshing phenomenon" [45], in which multiple subconcussive episodes contribute to the formation of neocortical neurofibrillary tangles and tauopathy, can ultimately result in CTE [46]. The clinical presentation of CTE begins with a latent period of approximately 8 years between the last traumatic event and onset of symptoms. Athletes begin to manifest symptoms between the ages of 35 and 45 years (range 24–65 years) [42]. CTE has been described as "a progressive deterioration in social and cognitive functioning, mood and behavioral disorders, progressive deterioration in interpersonal relationships, violent behavior, substance abuse, headaches and/or body aches, and increasing religiosity" [47]. It is of increasing concern that this long-term sequelae of concussion may develop even in individuals where there was little, if any, evidence of significant prior history. The diagnosis of CTE is ultimately made at autopsy, but ongoing research is focused on more specific diagnostic testing to determine both such risk and more precise methods of monitoring early-phase injury as well.

The concept of "second-impact syndrome" is one of the wide debates, with some arguing that this clinical entity does not exist [48]. Second-impact syndrome has also been termed "diffuse cerebral edema" and is the result of sustaining a second concussive event prior to symptom resolution of the first event. The proposed mechanism is loss of autoregulation of cerebral blood supply, which results in vascular engorgement and subsequent catastrophic cerebral edema [49]. Epidemiologically, it appears that some of the clinical presentations of "second-impact syndrome" were intracranial hemorrhages that massively expanded and caused death [50]. It is worth highlighting that the available body of literature clearly supports the fact that sustaining a second concussion shortly after an initial concussion prolongs the recovery and worsens symptom severity [51, 52].

Pediatric Considerations

Younger athletes have been shown to have longer recovery times when compared to older athletes [53, 54]. It is suggested that persons with mood disorders [20], learning disabilities or ADD/ADHD [55], or migraine headaches [56] might be more susceptible to concussion as well as experience prolonged symptoms from a concussion.

Intracranial Hemorrhage

Intracranial hemorrhage is the leading cause of death from sports-related head injuries. Four types of hemorrhage exist: epidural hematoma, subdural hematoma, subarachnoid hemorrhage, and intraparenchymal hemorrhage. Sideline providers should be familiar with all four types and be able to provide quick assessment for patients with suspected injuries.

Epidural Hematoma

Mechanism of Injury in Sports

Epidural hematomas occur when blood collects in the potential space between the skull and dura mater. The mechanism

is direct trauma to the head when the athlete is not wearing a helmet, for example, a baseball player struck in the head by a ball. An epidural hematoma can also occur when injury is sustained to the portions of the skull not protected by a helmet. Hematomas can occur in the infratentorial and supratentorial compartments but are most common in the temporal region. Fractures of the squamous portion of the temporal bone are usually associated with laceration of the middle meningeal artery.

Epidemiology

Overall, epidural hematoma is an uncommon head injury found in 1–4% of traumatic head injury patients. Observational studies have found that the mean age of patients with epidural hematoma is between 20 and 30 years of age [57]. Epidural hematomas are most often seen in the temporoparietal and temporal regions with a slight right-sided predominance. Bilateral epidural hematomas are seen in 2–5% of patients [58]. The majority of epidural hematomas are caused by traffic accidents, falls, and assaults with only a small number being a result of trauma sustained from participation in sports.

Clinical Presentation

In classic descriptions of athletes with epidural hematomas, there is an initial loss of consciousness following the injury. This is followed by a lucid interval and subsequent deterioration. Athletes may report headache symptoms. In cases where the hematoma is rapidly expanding secondary to arterial bleeding, the patient may never regain consciousness. Abnormal pupil reaction, abnormal posturing, and focal weakness may be present on physical examination.

Diagnosis

The most useful imaging modality for diagnosing epidural hematoma is a noncontrast CT of the head and brain. The CT will demonstrate a biconvex (football-shaped) mass in a focal location, usually in the temporal region. CT also allows for diagnosis of associated skull fractures. Small epidural hematomas may be missed due to volume averaging with adjacent bone. MRI and angiography are also imaging options but are used infrequently as CT is widely available (Fig. 43.3).

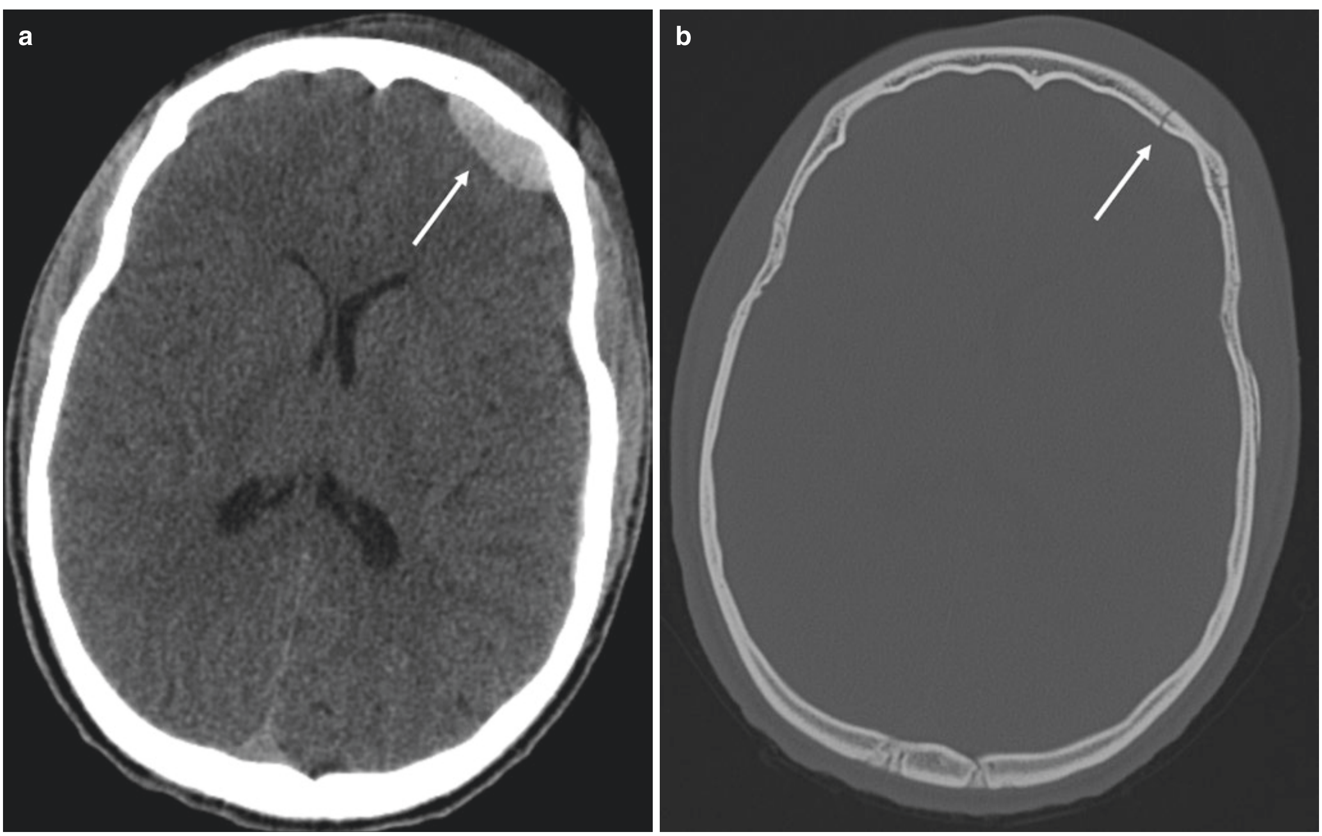


Fig. 43.3 Noncontrast head CT demonstrating (**a**) epidural hematoma (arrow) and (**b**) overlying skull fracture (arrow)

Initial Management

Initial sideline management of epidural hematoma should follow ATLS protocol as outlined earlier in this chapter. Spinal injury precautions should be initiated based on the mechanism of injury and level of suspicion for injury. Athletes with initial loss of consciousness should be reexamined frequently on the sideline. Anisocoria is a sign of oculomotor nerve compression. Athletes with suspected epidural hematoma should be immediately transported to a hospital with CT capability and neurosurgeon availability.

Indications for Neurosurgery Referral

All athletes with an epidural hematoma will require neurosurgical consultation. Arterial bleeding can lead to herniation within a few hours. Rapid identification and treatment is necessary. With epidural hematoma, the brain is usually spared of any direct injury, and these patients should be expected to do well. Surgical intervention is indicated if the clot is $\geq$ 30 cm^3 or 15 mm thick, if there is $\geq$ 5 mm of midline shift, if focal deficits are present, or if the GCS is < 8. Surgical treatment with craniotomy and evacuation of the clot is usually effective. In patients with severe decompensation, ICP monitoring with or without hemicraniectomy may be indicated. Conservative treatment can be considered in athletes without criteria for surgical intervention and includes serial CT scanning and close neurological observation.

Follow-Up Care

Athletes with epidural hematoma will require inpatient hospital management and neurosurgical consultation as indicated previously. Further care after hospitalization may include subacute rehabilitation. Athletes discharged from the hospital will need outpatient follow-up based on the interventions performed during hospitalization. This might include ambulatory clinic visits, physical therapy, and occupational therapy.

Return to Sports

There is no consensus regarding return to play in athletes who have sustained an epidural hematoma. Evidence in this area is based on expert opinion. After any intracranial hemorrhage, return to collision sports is discouraged. Return to non-collision sports can be considered if recovery is complete. It has been suggested that return to sport be considered 1 year after injury in patients where neurologic recovery is complete and imaging, neurocognitive, balance, and other clinical measures are at baseline for the individual athlete. These decisions should be made with the treating neurosurgeon. Informed discussion must take place with the athlete and their family if they are a minor. Athletes should not be allowed to return to play if they have persistent symptoms, permanent neurologic sequelae (e.g., organic dementia, hemiplegia, homonymous hemianopsia), hydrocephalus, or symptomatic neurologic or pain-producing abnormalities about the foramen magnum [59].

Complications

Complications of epidural hematomas include cerebral herniation, death, permanent neurological disability, seizures, and post-concussion syndrome.

Pediatric Considerations

Clinical features in pediatric patients with epidural hematomas are similar to those in adults, including the classic description of a lucid interval. In children, skull fractures are less common because their skull is more elastic than that of adults. Providers should aim to make the diagnosis before neurologic deterioration occurs. CT is the diagnostic modality of choice in pediatric patients with head injury and signs and symptoms of an epidural hematoma. Priorities in assessment and stabilization of children with head injury are the same as in adult trauma patients. Children with epidural hematoma require emergent evaluation by a neurosurgeon. Emergent craniotomy and hematoma evacuation is required if acute epidural hematoma is present with altered mental status, signs of increased intracranial pressure, pupillary abnormalities, focal neurologic deficits, or cerebellar signs. Pediatric patients with small epidural hematomas and no neurologic deficits may be managed nonoperatively by a neurosurgeon.

Subdural Hematoma

Mechanism of Injury in Sports

Subdural hematoma occurs when bridging dural veins tear from sudden acceleration-deceleration of the brain parenchyma. They can also result from accumulation of blood from cortical lacerations and rupture of intraparenchymal hemorrhage into the subdural space [2]. This can occur from shearing forces or direct trauma sustained in contact sports like being tackled or tackling in football. Trauma from boxing, mixed martial arts, and rugby also can produce these injuries. Subdural hematomas can occur in noncontact sports

where an athlete sustains head trauma, for example, a baseball player hit in the head with a bat or ball, a soccer player kicked in the head, or a cyclist thrown from a bicycle.

Epidemiology

Subdural hematoma is the most frequent type of hematoma occurring from trauma sustained in football. It is also the most common fatal injury from sports participation. Studies have shown that brain injuries account for 69% of deaths related to football [60], with 86% of these being diagnosed as subdural hematoma [61].

Clinical Presentation

Signs and symptoms of subdural hematoma are often delayed because veins bleed slower than arteries. Subdural hematomas can present acutely or chronically and can present with a variety of signs and symptoms. Patients with an acute subdural hematoma will typically have loss of consciousness and a poor initial neurologic examination. Those with either a small initial subdural hematoma or previously unrecognized subdural hematoma may have delayed symptoms including persistent headaches, altered mentation, blurred vision, nausea, focal weakness, and seizures. A complete list of the signs and symptoms of subdural hematoma can be found in Table 43.7.

Diagnosis

The diagnosis of subdural hematoma is made using noncontrast CT scanning. Acute subdural hematomas will appear as hyperdense, crescent-shaped lesions between the calvarium and cortex. They can extend beyond the suture lines and may follow the contour of the calvarium. They may also be seen in the interhemispheric fissure. Subacute or chronic subdural hematomas may appear isodense or hypodense. IV contrast may enhance these findings. MRI may also be a useful imaging modality. Indirect evidence may be present including midline shift, effacement of the ipsilateral cortical sulci, and compression of the ventricles. Subdural hematomas involving the posterior fossa do not cross the midline and do not extend above the tentorium (Fig. 43.4).

Table 43.7 Signs and symptoms of subdural hematoma

History of recent head injury	Numbness
Loss of consciousness or fluctuating levels of consciousness	Headache (constant or fluctuating)
Irritability	Dizziness
Seizures	Disorientation
Pain	Amnesia
Loss of muscle control	Weakness or lethargy
Altered breathing patterns	Loss of appetite
Hearing loss, ringing or tinnitus	Personality changes
Ataxia or difficulty walking	Inability to speak or slurred speech
Blurred vision	Deviated gaze or abnormal movements

Fig. 43.4 Noncontrast head CT of an 18-year-old football player with right-sided subdural hematoma, effacement of the ventricles, and midline shift. (Image courtesy of Ross Mathiasen, MD)

Initial Management

Sideline management of athletes with subdural hematomas should follow ATLS protocols. Providers will need to ensure that airway, breathing, and circulation are maintained. Airway assessment should include evaluation for patency and the athlete's ability to protect himself from aspiration of oral secretions, blood, or gastric contents. Athletes with impaired respiration may be breathing spontaneously, but tidal volumes may be low. A rapid rate of breathing may also be present and can exhaust the accessory muscles of respiration, leading to hypoventilation. Breathing should be supported with supplemental oxygen and bag valve mask ventilations. Airway support may be necessary and includes

oral or nasal airways and oral or nasotracheal intubation. Spinal injury precautions must be taken if spinal cord injury cannot be immediately excluded. Fluid resuscitation may be required to treat shock. Athletes will require evaluation for additional injuries during the secondary survey. Acute seizures may need treatment with benzodiazepines. Immediate transport to a hospital with CT scanning and neurosurgical capabilities will be necessary.

Indications for Neurosurgical Referral

Any athlete with subdural hematoma will require evaluation by a neurosurgeon. Indications for surgical management include hematoma thickness ≥10 mm, midline shift ≥5 mm, if GCS score decreases two or more points between injury and hospital admission, if pupils are asymmetric or fixed and dilated, or if intracranial pressure is greater than 20 mm Hg. Surgical management usually consists of craniotomy or burr holes. Patients can be managed conservatively if GCS is greater than 9, and the hematoma is small with no mass effect seen on CT. They will require monitoring of the intracranial pressure.

Follow-Up Care

As with epidural hematoma, athletes with a subdural hematoma require inpatient hospital management and neurosurgical consultation. Aftercare may include subacute rehabilitation, outpatient follow-up visits, physical therapy, and occupational therapy.

Return to Sports

Similar to epidural hematoma and any intracranial hemorrhage, return to contact sports after resolution of subdural hematoma is discouraged. No level I evidence exists to guide decisions. Current return-to-play recommendations are based on expert opinion. Athletes might consider return to play in noncontact sports after 1 year. This time is suggested to allow for reabsorption of the hematoma. Confirmation of resolution of subdural hematoma will need to be documented on repeat imaging, and the brain must be re-expanded to fill the subdural space with no residual hygroma.

Complications

Complications of subdural hematoma include cerebral herniation, death, persistent post-concussive symptoms (memory loss, dizziness, headache, anxiety, difficulty concentrating), seizures, and neurologic deficits.

Pediatric Considerations

Shaken baby syndrome and subdural hematoma in very young children are beyond the scope of this discussion. Child athletes with subdural hematomas usually have a history of major head trauma. Predisposing factors may be present but are less frequent. These include bleeding tendency, cerebral atrophy, and arachnoid cyst. Subdural hematoma should be considered in adolescent athletes with neurologic deterioration following head injury. Anisocoria, hemiparesis, and hemiplegia are all lateralizing neurologic signs and indicate cranial nerve compression or brainstem compression and signify progression to cerebral herniation. Initial assessment and management of these patients follows that for adult trauma patients. Pediatric patients with GCS ≤ 12 require emergent neurosurgical evaluation, and those with GCS ≤ 8 will require ICP monitoring. As with epidural hematoma, CT is the imaging modality of choice. No definitive criteria exist for surgical decision-making in pediatric patients with subdural hematoma. Surgical evacuation is performed in most children with acute subdural hematoma, neurologic deficit, and midline shift seen on CT. Comatose patients with severe edema and signs of infarct are poor candidates for surgical intervention. This contrasts with the management approach of epidural hematoma. Pediatric patients with subdural hematoma should be managed in a facility with advanced imaging and neurosurgical capabilities. Initial neurologic status is the best predictor of outcome regardless of age or injury mechanism.

Subarachnoid Hemorrhage

Mechanism of Injury in Sports

Traumatic subarachnoid hemorrhage is the result of disruption of the parenchyma and subarachnoid vessels. In subarachnoid hemorrhage, bleeding is confined to the cerebrospinal fluid space between the pia mater and the arachnoid membranes. These injuries can occur spontaneously from rupture of an aneurysm or arteriovenous malformation or from trauma sustained while participating in sports. Additional causes include cavernous angiomas, mycotic aneurysms, neoplasms, and blood dyscrasias. Subarachnoid hemorrhage has been reported from participation in swimming, diving, gymnastics, golf, weight training, and other sports [62]. The increase in risk of subarachnoid hemorrhage with physical activity or sports participation is thought

to be due to activation of the sympathetic nervous system, with increased heart rate and blood pressure and/or Valsalva maneuvers.

Epidemiology

Current literature suggests that subarachnoid hemorrhages occur in association with physical activity in up to 58% of cases. Only 2.1% of these cases occurred in athletes participating in sport. The majority of athletes with subarachnoid hemorrhage are male. Aneurysms were found in one-third of athletes with subarachnoid hemorrhage [62].

Clinical Presentation

Athletes with traumatic subarachnoid hemorrhage can present with sentinel headache, photophobia, and meningeal signs. Classically, the headache is described as the worst headache of their life. There may be a lucid period after the injury. Other symptoms include altered mental status, nausea and vomiting, and seizure. Physical exam may reveal signs of cranial nerve palsy, hypertension, retinal hemorrhages, papilledema, and motor deficits. Neurologic deterioration may be rapid, even to the point of death.

Fig. 43.5 Noncontrast head CT showing acute subarachnoid hemorrhage involving the right frontal and temporoparietal lobes (arrow) in addition to a large left posterior parietal scalp hematoma (arrowhead). (Image courtesy of Brent Griffith, MD)

Diagnosis

CT scanning is the imaging modality of choice in detecting traumatic subarachnoid hemorrhage (Figs. 43.5 and 43.6). Some subarachnoid hemorrhages may be missed on delayed CT, but typically in the setting of traumatic injury, CT is obtained immediately following the injury. CT scanning within 6 hours after injury or onset of headache is typically sensitive (93–100% in the first 24 hours) for diagnosing subarachnoid hemorrhage. If suspicion is high for subarachnoid hemorrhage, cognitively intact patients may require lumbar puncture to exclude aneurysm or arteriovenous malformation-related subarachnoid hemorrhage. CT angiography is the test of choice for detecting aneurysms. Providers may also consider the use of magnetic resonance angiography, but this test is limited by technical considerations in unstable patients [63].

Initial Management

As with other types of intracranial hemorrhage, the initial management of athletes with subarachnoid hemorrhage should follow ATLS protocols. In an unconscious athlete, the primary evaluation will focus on assessment of airway, breathing, and circulation. As with any unconscious athlete, cervical spine instability should be assumed, and their spine should be immobilized. If airway, breathing, and circulation have been adequately secured, a secondary survey can begin which includes evaluation of the head, maxillofacial structures, cervical spine and neck, chest, abdomen, genitourinary system, musculoskeletal system, and neurologic system. Unconscious athletes will require immediate transport to a hospital where CT scanning and neurosurgical services are available. If the patient is awake and cooperative, knowing the athlete's neuropsychological baseline is important for short- and long-term management of their head injury. Physical examination includes neurological, musculoskeletal and spine, maxillofacial, and ophthalmologic exams. Athletes will need to be observed for signs of neurologic deterioration. If intracranial hemorrhage is suspected, athletes must be transferred for imaging with CT and further evaluation.

Indications for Neurosurgeon Referral

Neurosurgical consultation will be necessary for any athlete with subarachnoid hemorrhage. Athletes with a GCS score from 13 to 15 can typically be managed conserva-

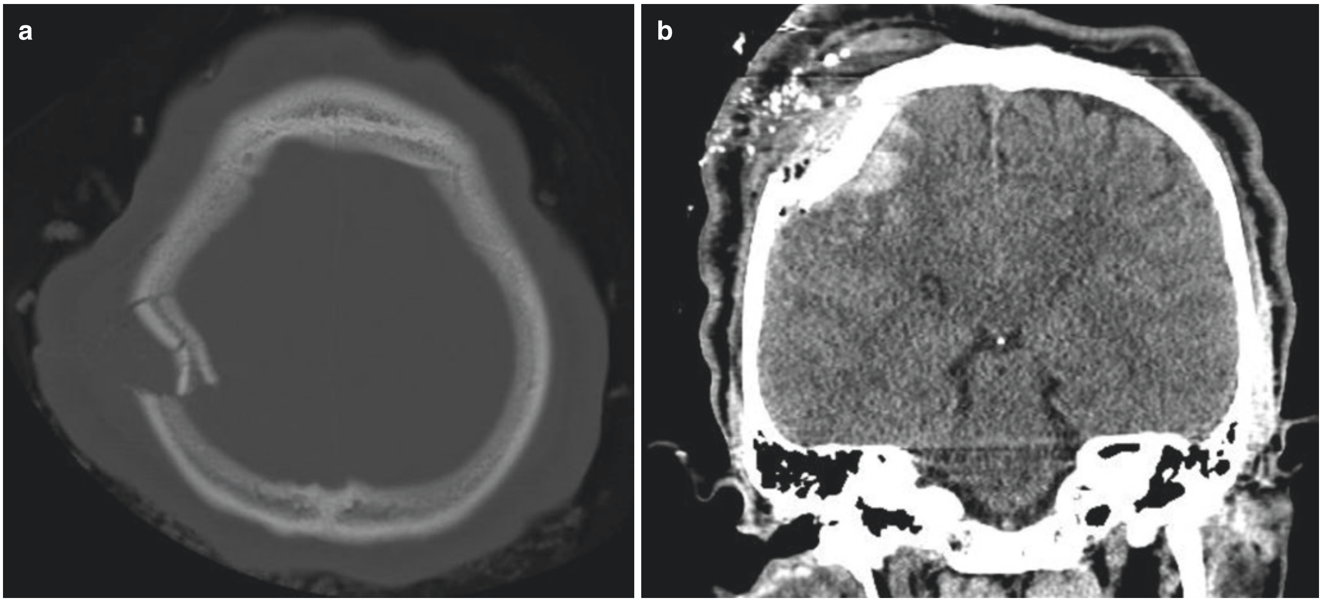


Fig. 43.6 Noncontrast head CT demonstrating (**a**) a depressed skull fracture involving the right parietal bone near the vertex (axial, bone window) and (**b**) associated hemorrhagic parenchymal contusion (coronal, brain window)

tively. They will require monitoring with follow-up CT scanning to ensure that bleeding is resolving. They will also require observation of blood pressure and possible intracranial pressure monitoring. Surgical management is rarely necessary in isolated traumatic subarachnoid hemorrhage. If an underlying aneurysm is found, it will often require treatment.

Follow-Up Care

Athletes who have sustained subarachnoid hemorrhage will require further outpatient care after hospital discharge. This may include subacute rehabilitation and outpatient physical and occupational therapy.

Return to Sports

Return to contact sports is contraindicated in athletes who have sustained spontaneous subarachnoid hemorrhage from any cause. Return to contact sports is also contraindicated in any athlete who requires operative intervention of their intracranial hemorrhage. No evidence-based guidelines exist, and return-to-play decisions are typically based on expert opinion. As with other types of intracranial hemorrhage, return to noncontact sports could be considered in athletes with subarachnoid hemorrhage 1 year after injury if they have complete recovery, have returned to baseline, have normal neuroimaging, and have no vascular abnormalities or coagulopathy.

Complications

Complications of subarachnoid hemorrhage include vasospasm, rebleeding, thromboembolism, cerebral infarction, cerebral edema, hydrocephalus, intracranial hypertension, electrolyte abnormalities, respiratory failure, myocardial dysfunction, and sepsis.

Pediatric Considerations

Aneurysms are uncommon in children and adolescents. Subarachnoid hemorrhage usually occurs secondary to arteriovenous malformation. Risk factors that may be present in the pediatric population include polycystic kidney disease, Marfan syndrome, Ehlers-Danlos syndrome, and coarctation of the aorta. In children, the reported incidence of subarachnoid hemorrhage is 0.18–2/100,000.

Intracerebral Hemorrhage (Brain Contusions)

Mechanism of Injury in Sports

Traumatic intracerebral hemorrhage in athletes can result from sudden acceleration and deceleration of the orbitofrontal lobes and temporal poles along the skull base as well as from direct head trauma sustained during sports participation. Bleeding occurs directly into the brain parenchyma, typically from rupture of a torn artery. Bleeding can also

occur from rupture of congenital vascular lesions like aneurysms or arteriovenous malformations. Traumatic intracerebral hemorrhages will typically expand over time.

Clinical Presentation

Athletes with intracerebral hemorrhage will often have stroke-like symptoms. They may not have a lucid interval, and their presentation can progress rapidly and lead to death. Intraparenchymal hemorrhage may have been unavoidable in cases where a congenital abnormality exists. Additional signs and symptoms of intracerebral hemorrhage include decreased mental status, nausea and vomiting, headache, seizures, and a focal or evolving neurological exam. See Table 43.8 for common focal neurological findings.

Signs of increased intracranial pressure or herniation include lethargy, hypertension, papilledema, emesis, and the Cushing reflex (bradycardia, decreased respiratory rate, and hypertension).

Diagnosis

The imaging modality of choice for diagnosing intracerebral hemorrhage is a noncontrast CT of the head and brain. A positive test will often reveal hyperdense areas that are well demarcated. They are usually located at the frontal, temporal, and occipital poles. Surrounding edema and mass effect may be present with midline shift and herniation. Serial CT scanning in the first 48 hours is usually required as hemorrhagic transformation can occur.

Table 43.8 Focal neurological findings in intracerebral hemorrhage

Structure	Findings
Putamen	Contralateral hemiparesis, sensory loss, conjugate gaze paresis
	Homonymous hemianopia
	Aphasia, neglect, apraxia
Thalamus	Contralateral sensory loss, hemiparesis, conjugate gaze paresis
	Homonymous hemianopia
	Miosis, aphasia, confusion
Lobar	Contralateral hemiparesis, sensory loss, conjugate gaze paresis
	Homonymous hemianopia
	Abulia, aphasia, neglect, apraxia
Caudate nucleus	Contralateral hemiparesis, conjugate gaze paresis
	Confusion
Brainstem	Quadraparesis, facial weakness
	Decreased level of consciousness
	Gaze paresis, ocular bobbing
	Miosis, autonomic instability
Cerebellum	Truncal ataxia
	Ipsilateral facial weakness, sensory loss
	Gaze paresis, skew deviation
	Miosis
	Decreased level of consciousness

Initial Management

Initial treatment of athletes with intracerebral hemorrhage is consistent with the other types of hemorrhage previously discussed. In unconscious athletes, the focus should be on evaluating and securing the athlete's airway, breathing, and circulation while maintaining spinal immobilization precautions until spinal injury can be safely excluded. Following the primary survey, a secondary survey can be completed, and the athlete can be transferred to a facility with CT scanning and neurosurgical capabilities. Awake athletes with head injuries will require neurological, ophthalmologic, maxillofacial, musculoskeletal, and spine exams as appropriate based on their injury. If intracerebral hemorrhage is suspected, the athlete will require transfer to an appropriate facility where CT imaging can be performed and neurosurgical consultation is available.

Indications for Neurosurgical Referral

Any athlete with intracerebral hemorrhage will require admission to the hospital and consultation with a neurosurgeon. Operative management should be considered if neurologic deterioration is caused by the contusion, if the contusion volume is ≥ 50 cm^3, with frontal and temporal contusion volume ≥ 20 cm^3, in those with ≥ 5 mm of midline shift or effaced basal cisterns, and if the GCS score is 6–8 [57, 59].

Follow-Up Care

Similar to previously described types of hemorrhage, follow-up care after hospitalization for intracerebral hemorrhage is based on the type of interventions required and the deficits sustained as a result of the injury. Rehabilitation in subacute and ambulatory settings may be required.

Return to Sports

Athletes with intracerebral hemorrhage should be discouraged from returning to contact sports. Return to noncontact sports can be considered after 1 year if recovery is complete. If return to sports is contemplated, imaging studies should be normal. Physical exam, neurocognitive testing, and balance

testing should be at the athlete's baseline. Contraindications to return to play include persistent edema or hemorrhage. Return to noncontact sports after craniotomy can be considered in athletes who have returned to baseline as above and if CT demonstrates complete bony union and MRI shows no underlying brain or meningeal injury.

Complications

Athletes with intracerebral hemorrhage are at risk of hematoma expansion, perihematomal edema with increased intracranial pressure, intraventricular extension of hemorrhage with hydrocephalus, seizures, venous thrombotic events, hyperglycemia, hypertension, fever, and infections [64].

Pediatric Considerations

In the pediatric population, intracerebral hemorrhage can be caused by trauma, vascular malformations, hematologic causes like thrombocytopenia, platelet dysfunction, hemophilia, congenital and acquired coagulopathies, hemoglobinopathies, cancer, moyamoya disease, and cryptogenic causes. Similarly to adults, pediatric patients can present with symptoms of nausea and vomiting, focal or generalized seizure, focal neurologic deficits, neck pain, and altered level of consciousness. Initial evaluation follows that for adults neuroimaging with CT or MRI is required for diagnosis. MRI is a preferred imaging modality in stable patients because of the lack of radiation and better resolution. CT should be performed if the patient is unstable or if obtaining MRI might delay the diagnosis. Management is similar to adults with initial evaluation focusing on stabilization of the athlete and treating elevated intracranial pressure if present with close monitoring for herniation. Neurosurgery consultation is required, and hematology consultation may be necessary if hematologic abnormalities are suspected.

Posttraumatic Seizure

Mechanism of Injury in Sports

Seizures can occur as a result of sports-related head trauma and can manifest as early seizures or late seizures. The exact pathophysiology of early seizures and concussive convulsions is unknown. The current theory suggests that rapid, nondiscriminatory release of excitatory neurotransmitters occurs at the time of impact, followed by spreading depression [65]. This is thought to result in loss of cortical inhibition and transient functional decerebration.

Posttraumatic seizures are thought to occur as a result of pathological changes and include reactive gliosis, axon retraction balls, Wallerian degeneration, microglial scar formation, and cystic white matter lesions. Changes to intrinsic membrane properties of pyramidal neurons and enhanced N-methyl-D-aspartate synaptic conductances are also thought to cause posttraumatic seizures. Hippocampal injury may also be a contributor.

In patients with brain contusion or cortical laceration, iron is released as a result of breakdown of hemoglobin. Iron may increase intracellular calcium oscillation and free radical formation. This results in excitotoxic damage, neuronal death, and glial scarring, leading to epileptiform activity [66].

Epidemiology

Up to 1 in 70 patients with concussion will experience a concussive convulsion after impact. Early posttraumatic seizures occur in 6–10% of individuals with head injury and in up to 20% in those with depressed skull fractures and intracerebral hematomas. Status epilepticus can develop in about 10% of patients with acute head injury. Focal motor status epilepticus is more common with subdural hematomas and depressed skull fractures.

The incidence of posttraumatic epilepsy at 10 years is approximately 2%. This risk is correlated with severity of the injury. Patients are considered to be at higher risk if they have early seizures, intracranial hemorrhage, cerebral contusion, or depressed skull fracture. Seizures begin within 2 years for the majority of patients with traumatic brain injury.

Classification

Posttraumatic seizures are classified based on the time of their occurrence following injury. Seizures occurring within seconds after injury were previously called impact seizures but are now referred to as concussive convulsions to better describe the non-epileptic nature of these events.

Early posttraumatic seizures occur within 1 week of head trauma. Similar to concussive convulsions, early seizures are not thought to represent epilepsy.

Posttraumatic epilepsy occurs more than 1 week after head injury. These occur as the result of more permanent structural and physiologic changes to the brain.

Clinical Presentation

Concussive convulsion will present with immediate tonic activity, followed by a clonic or myoclonic phase, and can last up to several minutes. If present, a postictal state is very

brief. Early posttraumatic seizures typically occur during the first 24 hours. The majority of these are generalized tonic-clonic. Seizures that occur later are more likely to be focal in onset, and more than half of those occurring after the first day are simple partial (pure motor) or focal with secondary generalization.

The majority of early posttraumatic seizures occurring in the first 24 hours are generalized tonic-clonic. Focal-onset seizures occur more often after the first day with more than half being either simple partial (pure motor) seizures or focal with secondary generalization.

In posttraumatic epilepsy, the seizures are not typically secondarily generalized. A smaller number are simple or complex.

About one in ten patients with an acute head injury will develop status epilepticus. Focal motor status epilepticus is seen most often in those with subdural hematoma or depressed skull fracture.

Diagnosis

The diagnosis of concussive convulsion is made clinically based on the athlete's presentation, and neuroimaging is not required. A noncontrast head CT is required in athletes with early posttraumatic seizures. MRI and EEG are not required during the acute phase of head injury.

Evaluation of athletes with posttraumatic seizure should follow that for any patient with first-time seizure. In patients with known traumatic brain injury within the last 2 years, diagnostic testing may be limited to neuroimaging, serum chemistries, and toxicology screen. If the patient is febrile and has other findings on exam or if the seizure is not consistent with the site of previous brain injury, further diagnostic evaluation may be required.

Initial Management

For athletes who sustain a concussive convulsion, immediate treatment includes supportive care and airway protection. No correlation has been shown with these injuries and abnormal imaging with CT and MRI. Athletes with early posttraumatic seizure will require imaging with a head CT. Treatment with antiepileptic drugs may be required if an athlete has significant risk factors such as a depressed skull fracture, intracerebral hematoma, or penetrating head injury and can reduce the incidence of early seizures in these patients. Treatment may also be required if there is concern for risk of status epilepticus or aggravating a systemic injury. Recurrent seizures can also increase cerebral blood flow and increase intracranial pressure. Treatment is typically started during hospitalization and withdrawn within the first few weeks.

Anticonvulsant treatment is recommended for athletes who have a late seizure. The risk of recurrence is up to 86% in untreated individuals [67]. Prophylactic treatment for posttraumatic epilepsy is not routinely recommended.

Indications for Neurology or Neurosurgical Referral

Neurology consultation should be considered for confirmation of the diagnosis in athletes with early or late posttraumatic seizures and for assistance with antiepileptic medication management in athletes with posttraumatic seizures. Neurosurgical consultation will be necessary if the athlete has a depressed skull fracture, intracerebral hematoma, or penetrating head injury.

Follow-Up Care

Athletes with concussive convulsion will not require treatment with anticonvulsant therapy. Those patients started on antiepileptic treatment will require regular outpatient follow-up for monitoring their seizure frequency, neurologic status and medication, observation for development of adverse medication effects, and measurement of drug levels.

Return to Sports

Concussive convulsions are considered benign and by themselves will not prevent an athlete from returning to play. If an underlying skull fracture or intracranial hemorrhage precipitated the onset of seizure, return-to-play decisions would be based on the underlying injury.

For athletes with recently diagnosed early or late posttraumatic seizures, return to sports may need to be delayed until treatment and control of seizures is established. Sports-specific restrictions are outlined in Table 43.9.

Participation in high-risk sports is not typically recommended for athletes with a seizure disorder. Helmet use is recommended for athletes participating in sports like cycling, equestrian sports, football, skateboarding, and skiing. A partner should be present for equestrian sports, ice skating, rollerblading, skateboarding, and boating. If an athlete with a seizure disorder is participating in sports that involve water, a lifeguard should be present and aware that the athlete has epilepsy. Activities should not be performed in the ocean or

Table 43.9 Risk stratification for sports participation in athletes with seizures

High risk	Bungee jumping
	Gymnastics
	Hang gliding
	Motor sports
	Mountain and rock climbing
	Scuba diving
	Sky diving
	Surfing
	Cycling
	Equestrian sports
	Ice skating
Moderate risk	Rollerblading
	Skateboarding
	Sailing/canoeing
	Swimming
	Water skiing/wakeboarding/ wakesurfing
	Aerobics
	Bowling
	Cross-country running or skiing
Low or no risk	Dancing
	Field hockey
	Golf
	Hiking
	Track
Contact sports with no known risk	Baseball
	Football
	Basketball
	Soccer

in water where visibility is limited. Athletes should also wear a life jacket if appropriate [8].

Complications

Athletes with concussive convulsion rarely have significant complications. Those with early posttraumatic seizures and posttraumatic epilepsy may be at risk for aspiration pneumonia if food or saliva is inhaled during a seizure episode, injuries that occur during a fall (fall, contusion, tongue biting, dislocation, fracture, etc.). Athletes can also experience side effects from using anticonvulsant medications.

Pediatric Considerations

Children under 7 years of age are more likely to have early posttraumatic seizures when compared to adolescents or adults. Early seizures and depressed skull fractures are risk factors for late posttraumatic seizures [68].

Skull Fracture

Mechanism of Injury in Sports

Skull fractures result from direct trauma. This occurs in contact sports and in sports with a high risk of collision or other trauma to the head.

Epidemiology

Sports-specific epidemiologic data on skull fractures is lacking. Simple linear fractures are the most common. Sports with the highest distribution of reported skull fractures include baseball, skateboarding, soccer, skiing and snowboarding, horseback riding, golf, street hockey, football, rollerblading and roller hockey, and skating [69]. In the general population, skull fractures are reported to occur between 2% and 20% of all cases of head trauma with an incidence of 35–45/100,000 people annually. Skull fractures occur most commonly between the ages of 20 and 50 years. Males are affected more predominantly than females. Falls are the most common mechanism and account for up to 35% of cases [70].

Fracture Classification

Skull fractures are classified by location (skull convexity or basilar), pattern (linear, depressed, or comminuted), and if they are open or closed. Linear fractures are the most common type and involve a single full-thickness fracture line. They account for 60–90% of skull fractures. Intracranial injury occurs in 15–30% of these patients [71]. If there is no associated intracranial injury, they are considered isolated. Comminuted fractures occur when the bone is broken into multiple pieces. The evaluation and management of simple comminuted skull fractures follows that of linear fractures.

A depressed fracture is one where a palpable bony abnormality is present. The risk of intracranial injury is up to 30%. Depressed fractures have a higher risk than linear fractures of dural tear and brain laceration. They also carry a higher risk of posttraumatic seizure.

Basilar skull fractures commonly involve the temporal bone, external auditory canal, and the tympanic membrane.

Fractures are considered as open if they communicate with the skin, sinuses, or middle ear. Open fractures, depressed fractures, fractures that involve a sinus, and those that are associated with pneumocephalus are all considered complicated fractures.

Clinical Presentation

The mechanism of injury is particularly important when considering a diagnosis of skull fracture. Aside from localized pain, other high-risk features to be aware of include loss of consciousness, seizure, amnesia, persistent headache, vomiting, and changes in behavior or confusion. Patients may present with signs of intracranial injury, such as altered mental status with GCS < 15 and abnormal neurologic examination. Additional physical exam findings include bony step-offs of the skull, clear rhinorrhea or otorrhea, hemotympanum, and periorbital or retroauricular ecchymosis.

Diagnosis

Athletes diagnosed with or suspected of having a skull fracture will require noncontrast CT of the head and brain to characterize the fracture pattern and to evaluate for associated injuries. Suture lines can be confused with fractures on imaging. Suture lines are typically < 2 mm wide and of uniform appearance. They can also be distinguished from fractures because they will appear lighter, are seen at specific anatomic areas, do not follow a straight line, and are relatively symmetric [6].

Initial Management

The priorities in sideline management of an athlete with suspected skull fracture are the same as in athlete with head trauma. The primary survey includes assessment and stabilization of the athlete's airway, breathing, and circulation. Spinal immobilization is necessary as these injuries are usually caused by high-energy mechanisms. Bleeding from wounds associated with skull fractures can be significant. Initial treatment should be with direct pressure. Large scalp lacerations may require placement of large sutures or the use of commercially available Raney clips or hemostatic agents like QuikClot. If scalp wounds are identified on the secondary survey, they should not be probed and examiners should be careful to avoid injury from bone edges or fragments. If skull fracture is suspected, athletes will require transfer for imaging and neurosurgical consultation.

Indications for Neurosurgical Referral

Isolated linear skull fractures can be treated conservatively and do not require consultation with a neurosurgeon unless they have associated intracranial hemorrhage. Athletes with depressed skull fractures will require hospital admission and consultation with a neurosurgeon. Those without dural penetration or significant complications can be managed conservatively. Surgical intervention is required for open skull fractures that are depressed > 5 mm or if there is associated dural tear, pneumocephalus, underlying hematoma, or gross contamination. All patients with basilar skull fractures will require admission and neurosurgery consultation. Skull fractures caused by penetrating injuries are considered open fractures and require consultation with a neurosurgeon.

Follow-Up Care

Athletes with skull fractures will require outpatient follow-up based on the type of injury and any associated injuries such as intracranial hemorrhage or concussion. For other types of fractures, the type of follow-up care is based on management and interventions required during hospitalization and associated injuries.

Return to Sports

No evidence-based guidelines exist for return to play. The majority of skull fractures heal within 3–6 months. Athletes with simple linear fractures can return to exercise as soon as symptoms allow. Return to high-risk sports can be considered at 12 weeks if CT demonstrates fracture union. Athletes with comminuted fractures, basilar skull fractures, and those requiring surgical treatment may consider return to play after fracture union is demonstrated. This may take 6 months or longer. Special consideration should be given for return to play when there are other associated injuries including intracranial hemorrhage.

Complications

Complications from skull fractures include cerebrospinal fluid leak, intracranial hemorrhage, venous sinus stenosis or thrombosis, meningitis, cranial nerve deficits, motor deficits, posttraumatic seizure, and postsurgical anosmia [70].

Pediatric Considerations

Signs and symptoms of skull fractures in the pediatric population are similar to those in adults. History may include traumatic injury, loss of consciousness, and localized pain and swelling. Physical examination features include soft tissue swelling of the scalp, hematoma, palpable fracture, skull defect, crepitus, and signs of basilar skull fracture (e.g., raccoon eyes, Battle's sign, hemotympanum, cerebrospinal fluid rhinorrhea, or otorrhea). As in adult patients,

pediatric patients will need to be evaluated for traumatic brain injury. CT is the imaging modality of choice. Pediatric patients with depressed skull fractures, basilar skull fractures, and linear skull fractures with greater than 3 mm of separation will all require evaluation by a neurosurgeon with pediatric expertise. If basilar skull fracture is present with CSF leakage, evaluation by a pediatric otolaryngologist is also required. Pediatric patients with no neurological findings and isolated linear skull fractures with no depression and narrow margins (< 3 mm separation) or isolated basilar skull fractures who have an otherwise normal CT can be treated conservatively without neurosurgical consultation [72].

Soft Tissue Injuries

Soft tissue injuries of the scalp are not uncommon from head trauma sustained during sports participation. High-risk sports include boxing, martial arts, and baseball. The use of a helmet is effective at reducing the risk of soft tissue injury to the head.

Abrasions of the scalp are common in sports. Local wound care is usually all that is necessary for treatment. Athlete can be instructed on keeping the wound clean, using antibiotic ointment to prevent infection, and covering the wound prior to participation.

Scalp lacerations can cause significant blood loss. Immediate management should focus on hemostasis and can often be achieved with direct pressure with sterile gauze pads and compression. If this is not effective, infiltration of lidocaine with epinephrine can be performed, and bleeding vessels can be clamped or ligated. Wounds should be explored for fractures and lacerations of the galea. Typically, anesthesia can be provided with local infiltration or by performing a scalp block. Copious irrigation can be performed with a high-pressure irrigation device. Debridement should be performed conservatively. Caution should be taken to ensure that hair is not buried in the wound. Superficial wounds can be closed using nonabsorbable sutures or staples. Large or gaping wounds can be closed using a two-layer technique, with absorbable sutures used subcutaneously for approximation of the galea. Avulsed tissue from the scalp can usually be reimplanted because of the scalp's excellent blood supply. Risk of infection and scalp wounds is low but is increased in multilayer closures.

Scalp hematomas can occur as a result of direct contact. Patients should be evaluated for underlying skull fracture or intracranial hemorrhage. Localized hematoma can be treated conservatively. Initial management includes cold therapy and compression. Aspiration and incision and drainage are not normally required (Table 43.10).

Table 43.10 SORT: key clinical recommendations

Clinical recommendation	Evidence rating
The need for neuroimaging in a head-injured athlete should be determined by using clinical decision rules	A
Concussion is a clinical diagnosis; there are no objective diagnostic criterion for concussion	C
It is common for concussed athletes to remain asymptomatic or minimally symptomatic in the initial 24 hours post injury	C
90% of athletes with concussion are asymptomatic by day 10 post injury; however, it is still considered normal to have symptoms for up to 28 days	C
Return to collision sports is discouraged after any intracranial hemorrhage	C
Return to non-collision sports can be considered after intracranial hemorrhage if recovery is complete	C
Participation in high-risk sports is not typically recommended for athletes with a seizure disorder	C
Athletes with simple linear skull fractures can return to exercise as soon as symptoms allow	C

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44 Facial Trauma

Christopher Hogrefe

Key Points

- Up to 42% of facial fractures are secondary to sports injuries (Fig. 44.1) [1].
- There is a high rate of associated injuries with facial fractures. Be certain to assess for concomitant injuries, particularly airway, intracranial, and cervical spine trauma.
- The size of the fracture does not always portend the treatment. Readily apparent orbital fractures may not necessitate an operative intervention, while subtle mandibular fractures may require surgical fixation.
- Protective equipment (e.g., face shields, mouthguards, etc.) can help reduce the risk of these injuries in addition to safeguarding a healing fracture.

Fig. 44.1 A maxillofacial CT 3-D reconstruction of a mixed martial arts fighter with multiple facial fractures following a knee to the head

Facial Fractures and Dislocations

Introduction

Over the years the advent and advancement of protective equipment (e.g., helmets, visors, glasses, etc.) have helped to decrease the incidence of facial fractures [2]. However, increased involvement in sports with higher risk for injury and/or more contact has diminished some of the progress in this regard [3]. In the United States, baseball and softball have been associated with the highest incidence of facial fractures (44.3%). Throwing, catching, and hitting a ball have been correlated with these injuries (34.1%), while collisions are the next most common etiology (24.5%) [4]. On the other hand, in countries like Italy, Chile, and Germany, soccer accounts for the highest frequency of sports-related facial fractures (up to 59.2%) [5]. Regardless, it appears that adolescents and young adults are at the greatest risk for such fractures [6].

Table 44.1 Incidence of sports-related facial fractures* [9]

Facial bone	Incidence (%)
Nasal	35.9
Orbital	33.5
Maxillary	12.6
Mandibular	7.2
Zygomatic	4.2
Naso-orbitoethmoid	1.2

*Skull fractures represented 30.5% of overall fractures in this study

The facial bone anatomy is complex and contains at least 14 bones (not including the frontal bone). The frequency with which these bones fracture varies, but Table 44.1 provides insight into the relative incidence of certain facial bone frac-

C. Hogrefe (✉)
Department of Emergency Medicine, University of Iowa Hospitals and Clinics, Iowa City, IA, USA
e-mail: christopher-hogrefe@uiowa.edu

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tures [7]. Some of the pertinent nuances to the bony anatomy are discussed in the sections that follow. In general, it is worth noting that the amount of force needed to fracture a facial bone is quite significant (30 grams of force for the nose and up to 200 grams of force for the superior orbital rim) [3]. The design of the face serves many purposes, including to protect the intracranial contents by functioning as a crumple zone and dispersing forces across the facial bones. Additionally, due to the rich vascularity of the face, edema and ecchymosis often promptly follow these injuries, complicating the physical examination. Consequently, it is important that the sports medicine physician has a high index of suspicion for these fractures and other accompanying injuries.

Orbital Fractures

Athletic competition can precipitate direct trauma to the orbital rim and orbital walls, resulting in fractures to any of the associated bones. The orbit itself consists of seven bones: frontal, sphenoid, maxilla, palatine, zygomatic, ethmoid, and lacrimal bones (Fig. 44.2). The medial and inferior orbital walls are the most frequently affected, which often result in "blow-out" fractures. In fact, the posteromedial portion of the orbital floor and the lamina papyracea of the medial wall are the weakest components of the orbit. It is important to note that orbital fractures can result in concomitant ocular injuries as well.

Mechanism of Injury in Sports

There are generally thought to be two broad mechanisms by which orbital fractures transpire. A direct blow to the orbital rim can cause one type of fracture, such as when a baseball or tennis ball collides with the orbital rim. Conversely, direct trauma to the eye itself, for instance, by an (in)advertent fist or elbow, can precipitate an orbital "blow-out" fracture. There is another, less common injury pattern referred to as a "blow-in" fracture, which can occur if fracture fragments are displaced into the orbital space, often secondary to trauma to the frontal and/or maxillary bones [8].

Epidemiology

It is reported that roughly one-third of orbital fractures transpire in sport. The actual number of cases that this represents is not known [9]. "Blow-out" fractures are the most common type of orbital fractures, with only 5% of these injuries occurring bilaterally [10].

Clinical Presentation

First and foremost, patients should have normal (or baseline) visual acuity. If this is not the case, it should significantly raise one's suspicion for ocular injury. Roughly 25% of orbital fractures involve the globe, with "blow-out" fractures carrying an associated ocular injury rate of up to 40% [11]. Especially noteworthy, 5–10% of "blow-out" fractures result in a ruptured globe [12]. One should assess for bony tenderness, swelling, and/or ecchymosis. Enophthalmos (depression of the globe due to orbital fat sinking through an orbital fracture) may be appreciated. Periorbital emphysema can be present, attributed to communication with either the ethmoid or maxillary sinus. Impaired ocular mobility may be seen as well due to entrapment of the extraocular muscles within the fracture fragments. For instance, if the medial rectus is entrapped, there will be a defect with lateral gaze (Fig. 44.3). Athletes may also note decreased sensation in the upper lip region due to an injury to the infraorbital nerve.

Diagnosis

While the physical examination can certainly provide evidence to suggest an orbital fracture, further confirmation in the form of imaging is warranted. Plain films of the orbits (including Waters view or exaggerated Waters view) should primarily be considered if there is no accessibility to further

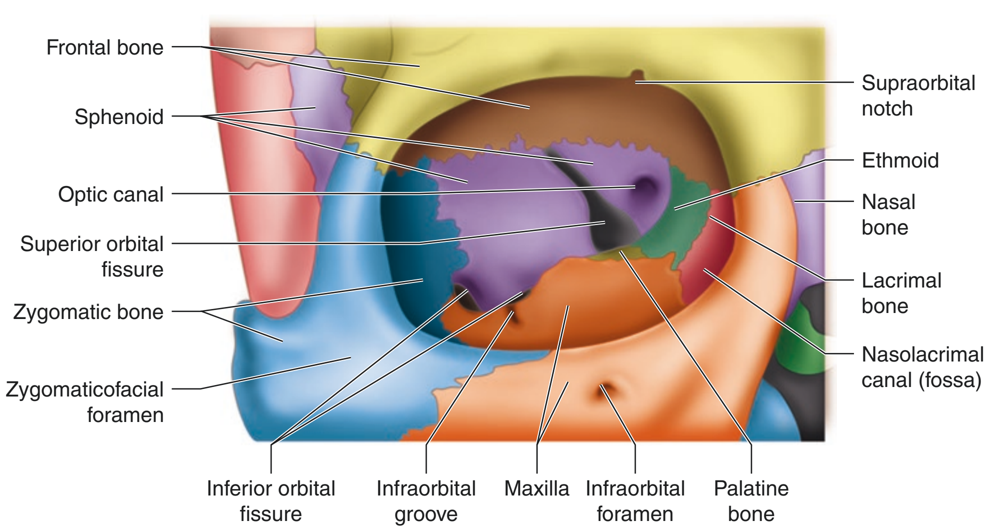

Fig. 44.2 The bones comprising the orbit

Fig. 44.3 A patient with a right medial orbital fracture causing medial rectus entrapment and a lateral gaze defect (i.e. decreased abduction of the right eye) (Image from Joseph and Glavas [114])

advanced imaging. A CT scan of the orbits is the preferred imaging modality. Thin-sliced (1.5 mm) helical scans are recommended, although axial and coronal CT scans can suffice [11]. The sensitivity of an orbital CT scan is between 79% and 96% [8]. Ultrasound and MRI are of limited utility, and ultrasound in particular should be avoided if there is suspicion for a concomitant globe rupture.

Initial Management

The most important initial management step in the treatment of orbital fractures is to assess for physical examination findings that should prompt an immediate Emergency Department, Ophthalmology, and/or Facial Trauma evaluation (see below under the section "Indications for Referral"). Otherwise, the current recommendations for treatment include the aggressive use of cold packs or ice for the first 48 hours [13]. Sneezing with one's mouth open, the avoidance of nose blowing, and refraining from coughing or other Valsalva maneuvers should be recommended. Sleeping with the head of the bed elevated has been advocated as well. Provide the athlete with nasal decongestants and pain control. The utilization of prophylactic antibiotics is debated; however, it is generally recommended if there is communication of the fracture with a sinus [14]. Amoxicillin-clavulanate and azithromycin are considered appropriate selections in this context with the duration of treatment lasting 10–14 days.

Indications for Referral

An immediate evaluation is warranted if there is concern for a ruptured globe, concomitant facial fracture, visual changes, or impaired ocular motion. If these entities have been ruled out on physical examination, athletes can be further evaluated (including the procurement of imaging) 1–2 days after the initial injury.

Follow-up Care

If a fracture is diagnosed, follow-up with an Ophthalmologist and an Otolaryngologist/Facial Surgeon is warranted within 1 week. The timing of such follow-up is designed to allow for the swelling to resolve, which may further improve the athlete's symptoms. Keep in mind that only approximately 15% of patients will require a surgical intervention [15]. Typical reasons for a surgical intervention include persistent diplopia, impaired ocular movement, and/or significant enophthalmos.

Fig. 44.4 A protective face mask for use after an orbital fracture

Return to Sports

In the setting of an isolated orbital fracture, return to noncontact sports typically occurs in approximately 2 weeks. In contact sports the return is closer to 4–6 weeks [8]. If there is an associated ocular injury or surgery is pursued, the return will be longer and is dependent upon the extent of the ocular injury and/or complexity of the surgical intervention. Regardless, proper eye protection is recommended upon an athlete's return, generally for up to 8 weeks after the injury [4] (Fig. 44.4).

Complications

There are several potential complications associated with orbital fractures. The more common complications include sinusitis, permanent extraocular movement restriction, orbital infection, and/or enophthalmos. Depending upon the severity of the injury, athletes can suffer permanent vision loss and/or cosmetic defects, particularly if there is a delay in treatment [16].

Pediatric Considerations

While orbital wall fractures can occur in children, they are less common, especially under the ages of 6 or 7. The bones are more malleable, allowing them to bow or bend more prior to fracturing. This can, however, increase the risk for ocular entrapment. Recall that given the lack of pneumatization of the paranasal sinuses in younger athletes, the orbital floor is

not as weak in children as it is in adults. With that said, orbital roof fractures are slightly more common in this age group and associated neurologic involvement can follow.

Mandibular Fractures

Given its prominence on the face and lack of surrounding support, the mandible is a common site of fractures in sports. There is some variability in the literature as to the most common sites of fracture within the mandible. Broadly speaking, the condyle, angle, and the body of the mandible are the most common fracture sites (typically in that order), followed by the molar and mental regions, respectively (Fig. 44.5). It is estimated that 20–40% of patients with mandible fractures will have another injury. Thus, the sideline physician should be ever mindful of the ABCs of patient stabilization and advanced cardiac life support (ACLS) protocols.

Mechanism of Injury in Sports

Mandibular fractures in the setting of sports are most commonly found in contact sports (e.g., boxing, wrestling, and football), although sports account for only 4% of all such fractures [17]. They are typically the result of direct trauma to the mandible, which is a ring structure (similar to the pelvis). Thus, contralateral fractures occur with some frequency and should be considered. It is not uncommon for these fractures to be associated with concomitant intracranial and/or cervical spine injuries.

Epidemiology

Among all of the facial fractures, mandibular fractures are the third most common (trailing only nasal and zygomatic fractures). The incidence of these injuries peaks between the ages of 16 and 20 years old [8]. Sport-specific mandibular fractures do not appear to show a gender predilection, although in the overall population young males have a higher prevalence of such injuries [2, 18].

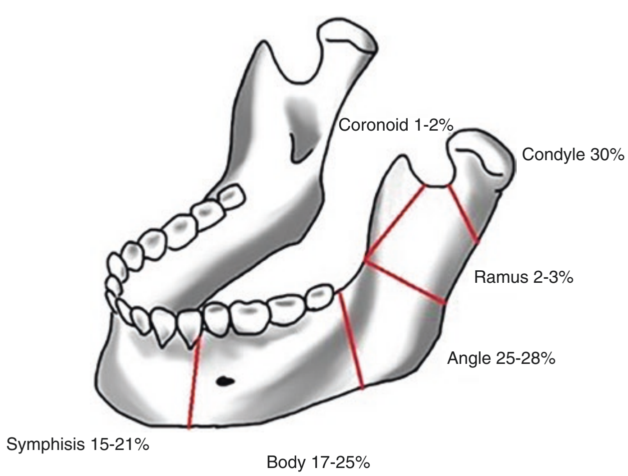

Fig. 44.5 Distribution of mandibular fractures (Image courtesy of Steve Chukwulebe, MD)

Clinical Presentation

Athletes with mandible fractures often report pain exacerbated by jaw movement, dysphagia, and/or an abnormal bite in the setting of a traumatic episode. On physical examination, one should be certain to thoroughly inspect the jaw. In addition to a visible step-off and/or facial asymmetry, mandible fractures can be associated with lacerations, particularly under the chin. A valuable clinical test is the tongue blade bite test, which is performed by inserting a tongue depressor into the patient's mouth between the maxillary and mandibular teeth. The patient should bite down on the depressor, and the physician should then twist the blade. If the tongue depressor can be broken without causing the athlete pain, the test is considered negative. This test has been shown to have a sensitivity of 88.5% with a specificity of 95% for identifying mandible fractures (with roughly the same utility in isolated pediatric patients) [19]. Additional valuable physical examination findings commonly associated with mandible fractures are noted in Table 44.2.

Diagnosis

As opposed to some of its facial fracture counterparts, plain films can be quite useful in diagnosing mandible fractures. An orthopantomogram (or Panorex) is generally the view of choice, facilitating both a diagnosis in addition to surgical planning when necessary (Fig. 44.6). In athletes where there

Table 44.2 Clinical findings associated with mandible fractures [3]

Loose, fractured, or missing teeth
Trismus (mouth opening <2–3 finger widths)
Inability to chew
Intraoral/gingival lacerations
Ecchymosis/hematoma (at the fracture site or the sublingual space)
Paresthesias of the lip/chin
Lack of motion of the mandibular condyles with palpation through the external auditory canal

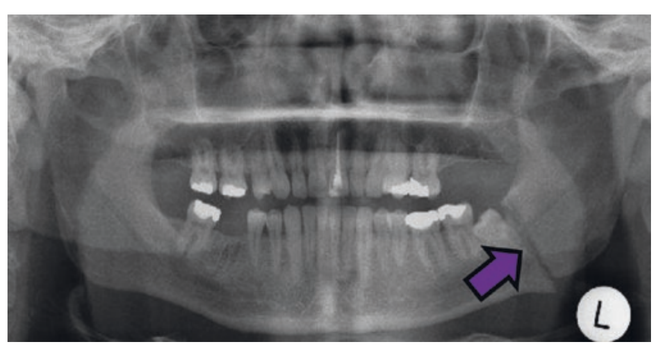

Fig. 44.6 An orthopantomogram depicting a fracture at the left angle of the mandible

is a high clinical suspicion for a fracture in the setting of a negative orthopantomogram, a maxillofacial CT should be considered. This is especially the case if a ramus or condyle fracture is suspected as the degree of displacement in these areas can be subtle [3]. Of note, some studies have suggested that ultrasound also has a high sensitivity (97.4%) and specificity (100%) for identifying mandible fractures, along with boasting a 100% positive predictive value [20]. Lastly, in the event of a newly missing tooth, consider securing a chest radiograph to ensure that the tooth was not aspirated.

Initial Management

The initial management of these injuries is primarily centered on ensuring that the athlete's airway is intact and that there are no cervical spine abnormalities. Any bleeding should be controlled with gentle pressure such as biting on gauze and/or closing the mouth. No wound closure should be immediately attempted. Keeping the mouth closed may also decrease the risk for (further) fracture displacement and/or subsequent malunion or nonunion [8]. Any suspected mandible fracture associated with a dental disruption, alveolar ridge fracture, and/or a laceration should be considered an open fracture. Athletes should be evaluated by a specialist immediately in the setting of such fractures, as definitive treatment often entails a surgical intervention.

Indications for Referral

Suspicion for a mandible fracture warrants immediate assessment in an Emergency Department or by an Oral Maxillofacial Surgeon. If there are concomitant injuries (e.g., oral lacerations, airway compromise, cervical spine injuries, dental avulsions, etc.), the Emergency Department may represent the optimal place to facilitate further evaluation and treatment. Delays of more than 14 days can lead to early bony healing and thus more complicated subsequent management.

Follow-up Care

In relatively infrequent cases of non-displaced, closed fractures resulting in minimal pain, athletes may be discharged from the Emergency Department. They will typically be placed on a soft diet and scheduled for follow-up with an Otolaryngologist or Oral Maxillofacial Surgeon.

Return to Sports

In nonoperative fractures or following a surgical intervention, it has been argued that a return to aerobic activities and strength training is reasonable at 10–14 days. No activities that place the athlete at risk for a blow to the face should be performed. Otherwise, full contact activity has been advocated as quickly as day 41, while others prefer a minimum of 8 weeks [3]. In combat sports a minimum of 3 months of recovery from the time of injury is recommended to enable adequate healing [21]. When returning to contact activities, it is reasonable to consider a custom mouthguard and/or modified headgear (e.g., the protective flap on a baseball helmet as in Fig. 44.7) to protect the surgical repair and guard against a subsequent reinjury. There is no clear consensus regarding the duration that such protective equipment should be utilized moving forward. The athlete's preference and comfort (e.g., with the equipment itself and with the participation in sport) should be considered when making this determination.

Fig. 44.7 A baseball helmet modified with a protective facial flap

Complications

Complications from mandibular fractures and the treatment of such injuries are rare, particularly if the initial injury is promptly identified and treated [22]. Infections can occur, particularly in the setting of an open fracture. Other potential complications include disruption of the dental roots with subsequent pulpal necrosis and/or abscess, malocclusion, and paresthesias to the face due to a sensory nerve injury [3]. For some patients, physical therapy may be beneficial after a prolonged period of immobilization, which often accompanies mandibular fracture treatment.

Pediatric Considerations

Mandibular fractures are rare in children younger than six and are often of the greenstick variety. However, these injuries can result in damage to permanent teeth and/or facial deformities. Thus, it is important for these athletes to secure follow-up with a Dentist and/or Oral Surgeon to avoid potential long-term complications. Prolonged immobilization of the mandible can also lead to fibro-osseous or osseous ankyloses of the temporomandibular joint [3].

Maxillofacial Fractures

The maxillofacial region of the face, or the midface, includes the entire face excluding the mandible.[1] These high-energy injuries carry with them a higher rate of concomitant injuries. It is paramount that the team physician also considers airway compromise, intracranial pathology, and cervical spine injuries, among others, when evaluating a possible maxillofacial fracture. Fractures of the maxilla and zygoma will be the primary focus of this section.

Mechanism of Injury in Sports

Direct, blunt trauma to the face constitutes the bulk of the injuries that result in midface fractures. This can range from a fall (e.g., a wrestling takedown) to a wayward elbow (e.g., when boxing out an opposing rebounder in basketball) to a ball directly to the face (e.g., a softball batter).

Epidemiology

It has been estimated that up to 30% of facial fractures involve the midface [8]. Fractures of the zygoma rival mandibular fractures with regard to the incidence in sport [23, 24]. However, isolated maxillary fractures tend to be uncommon [3]. Overall, there is a higher incidence of these fractures in younger males as well, although this may not be isolated to athletic participation alone.

Fracture Classification

The majority of midface fractures are complex and do not lend themselves well to strict classification systems. However, the Le Fort classification is commonly utilized to distinguish midface fractures involving the maxilla based on the plane of injury (Table 44.3 and Fig. 44.8). It is worth noting that athletes may have more than one Le Fort fracture on the same side and/or different classifications of fractures on each side of the midface.

Table 44.3 Le Fort fracture definitions

Le Fort type	Description
I (Horizontal)	Fracture of the maxilla below the nose but above the teeth; extends through the lateral maxillary sinus to the lateral pterygoid plate
II (Pyramidal)	Pyramid-shaped fracture from the nasal and ethmoid bones through the zygmomaticomaxillary suture and maxilla
III (Transverse)	Craniofacial disjunction causing movement of the entire midface, including the orbital rims and base of the nose
IV	A Le Fort III that also involves the frontal bone

Clinical Presentation

In addition to the presence of direct trauma to the face, there are other useful findings on clinical examination that should raise a sideline physician's suspicion for a midface fracture. These findings are included in Fig. 44.9. Cerebrospinal fluid (clear) rhinorrhea is a rare but concerning physical examination feature for which sideline physicians should assess.

Le Fort fractures can be identified with specific movements of the midface. The team physician should place the thumb and index finger of one hand on the nasal bridge and utilize the other hand to pull the maxillary teeth. If the hand on the nasal bridge does not move while the hard palate and upper teeth move forward with traction, the athlete has a Le Fort I fracture. If both structures move, the patient has a Le Fort II fracture. And if the entire midface shifts (including the orbital rims) with the aforementioned traction, the athlete has a Le Fort III injury.

Diagnosis

The definitive diagnosis of a maxillofacial fracture is made via a maxillofacial CT (2 mm axial and coronal cuts are recommended). Plain radiography of the face is not sufficient to rule out such injuries. Consideration should be given to obtain additional imaging (e.g., a CT of the brain and/or cervical spine) as well due to the high rate of associated injuries.

Initial Management

The most important initial management steps for suspected midface fractures include ensuring that the athlete is able to protect his/her airway and that care is taken to stabilize the cervical spine when necessary. Next, any bleeding should be controlled via gentle pressure with care taken to avoid potentially shifting or displacing any existing fractures.

Indications for Referral

All athletes with suspected midface fractures warrant immediate evaluation in an Emergency Department. Further advanced imaging in the form of a maxillofacial CT should be pursued. If a fracture is identified, an evaluation by an Oral Maxillofacial Surgeon should occur. Due to the high risk of ocular injuries as well, consultation with an Ophthalmologist is also advised [25].

Follow-up Care

There are instances when athletes may be discharged from the Emergency Department following maxillofacial fractures that do not require immediate intervention. For instance, closed zygoma fractures or displaced zygoma fractures that are stable for outpatient reduction will require follow-up.

[1] From a technical standpoint, orbital and nasal fractures are also included in the category of maxillofacial or midface fractures. However, given their associated unique evaluation and treatment considerations, they have been given their own sections in this chapter.

Fig. 44.8 Le Fort classification of midface fractures (Image courtesy of Steve Chukwulebe, MD)

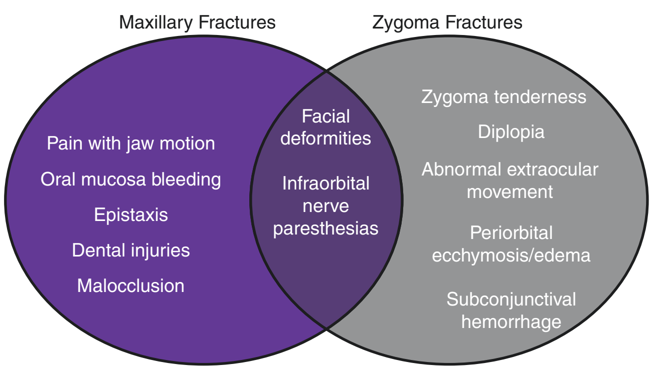

Fig. 44.9 Clinical findings commonly associated with maxillary and zygoma fractures

This secondary evaluation should occur with an Oral Maxillofacial Surgeon within 1–3 days [12].

Return to Sports

There are no clearly delineated return to sports guidelines pertaining to maxillofacial fractures. This fact is secondary to the complex nature of these injuries and the high association with concomitant injuries. Further compounding the issue is the nature of the sport itself (e.g., cross-country versus hockey). A team approach in assessing an optimal timeframe for such a return incorporates the athlete, his/her parents (when appropriate), the athletic training staff, and the treatment team (often consisting of an Oral Maxillofacial Surgeon and/or an Ophthalmologist along with the team physician).

Complications

These fractures carry a significant risk for nonunion or malunion, particularly if there is a delay in treatment. The nature of the injury may lend itself to permanent nerve damage, including involvement of the infraorbital nerve. Lastly, there is also a risk of associated infection with midface fractures beginning at the time of the trauma itself and persisting throughout the definitive treatment.

Pediatric Considerations

Midface fractures are uncommon before the age of 10–12 years old, at which time the facial morphology begins to resemble that of an adult.

Temporomandibular Dislocations

The temporomandibular joint (TMJ) is the only joint in the body capable of both translation and rotation. The nature of these movements contributes to the ability of the TMJ to dislocate, occurring when the mandibular condyle slides out of its articulation and becomes locked (Fig. 44.10). This most commonly transpires in an anterior fashion. However, the condyle can also slide superiorly or posteriorly [26]. Spasms of the powerful external pterygoid, masseter, and internal pterygoid muscles can make this a difficult injury to properly treat.

Mechanism of Injury in Sports

Athletes involved in collision sports are most likely to suffer a dislocation of the TMJ. Sports at a higher risk include football, field hockey, ice hockey, soccer, and lacrosse. However, these injuries can occur with any significant opening of the mouth as well (e.g., yawning during a rain delay, singing “Take Me Out to the Ballgame,” eating between a doubleheader, etc.).

Epidemiology

Overall, this condition is relatively rare. Aside from a predisposition to traumatic collisions, other risk factors include

Fig. 44.10 A left TMJ dislocation (Image courtesy of Kara Toles, MD (with permission provided by the patient for educational purposes); aliem.com)

bruxism (teeth grinding), malocclusion, and joint laxity (e.g., Ehlers-Danlos or Marfan syndrome). Those who have sustained a previous TMJ dislocation do carry a greater risk of a subsequent dislocation [27].

Clinical Presentation

In addition to a preceding trauma, athletes with dislocations of the TMJ most frequently present with the jaw locked in the open position. It is possible for the jaw to be locked in a closed position as well, which is most often the result of displacement of the fibrocartilage articular disc of this joint. Patients may also note referred pain to the ipsilateral ear, head, and/or neck. Drooling and/or garbled speech may be present [28]. The preauricular area can be depressed, suggesting the absence of the mandibular condyle in this space. Additionally, the team physician may note a palpable condyle anterior to the expected TMJ articulation.

Diagnosis

The diagnosis of a dislocated TMJ is usually clinical in nature. In the setting of trauma, imaging should be obtained to rule out a mandible fracture (particularly of the condyles). In such cases, an orthopantomogram is a reasonable first imaging choice. However, as previously discussed, a maxillofacial CT may be necessary in the context of a high index of suspicion and a negative orthopantomogram.

Initial Management

As soon as the concern for an associated mandible fracture has been addressed (which may or may not necessitate further imaging), a TMJ dislocation reduction should be attempted as soon as possible. Prompt action in this regard can help to avoid the onset of trismus [29]. If and/or when trismus occurs, the associated strength of the musculature surrounding the TMJ may prevent an adequate and/or comfortable reduction. Often, procedural sedation (e.g., with ketamine and/or propofol) is necessary to facilitate this procedure, particularly in the setting of associated trismus.

To perform a TMJ reduction, the patient should be placed in a seated position against a firm surface such as the back of a chair or wall. The provider's thumbs should be wrapped with ample gauze, as the jaw can quickly snap back into place upon successful reduction. The thumbs should be placed over the posterior mandibular teeth. The remainder of the provider's fingers are then positioned along the inferior borders of the mandible near the angle. Downward pressure should be applied followed by a posteriorly directed force upon the chin (for most cases, which are due to an anterior TMJ dislocation). If the associated muscles are sufficiently relaxed, this technique should be successful (Fig. 44.11).

Indications for Referral

TMJ dislocations require prompt reduction. However, they should not be reduced on the sideline or in the office if a fracture is suspected. Therefore, if there is concern for an associated fracture, an Emergency Department or Oral Maxillofacial Surgery evaluation is indicated. Superior and posterior dislocations of the TMJ are also indications for a referral to the Emergency Department, as these can be more difficult to reduce. Otherwise, if the team physician is unable to reduce a TMJ dislocation on-site or in the office, a referral to the Emergency Department should be pursued. Lastly, athletes with chronic or recurrent dislo-

Fig. 44.11 Reduction technique for a TMJ dislocation

cations should be referred for further evaluation by an Oral Maxillofacial Surgeon.

Follow-up Care

Once reduced, these athletes are typically advised to consume a soft diet for 1 week to avoid wide opening of the mandible. Analgesic medications and/or muscle relaxers may be necessary as well to minimize the risk of recurrence. Further follow-up with Oral Maxillofacial Surgery may be necessary in the event that the patient has persistent pain at the TMJ following an adequate reduction.

Return to Sports

Assuming there are no concomitant fractures, once the TMJ is reduced and the associated pain is well controlled, athletes can safely return to play.

Complications

Potential complications of TMJ dislocations include associated fractures, particularly of the condyle, which can occur as a result of the reduction as well. Other primary complications include malocclusion and/or persistent pain at the TMJ.

Pediatric Considerations

One of the primary pediatric considerations centers on bruxism. While this can be a predisposing risk factor, it can also lead to a recurrence of a TMJ dislocation. A night guard can prove beneficial in mitigating this risk.

Ocular Trauma

Key Points

- Ocular injuries are frequently associated with facial fractures and vice versa.
- A thorough eye examination and a good index of suspicion are essential in identifying sometimes subtle ocular injuries.
- The initial sideline management of eye trauma is frequently minimal, but the need for a prompt Ophthalmology evaluation is often critical.
- Eye protection has been shown to significantly decrease serious eye injuries in sport.

Introduction

Despite being largely preventable, ocular injuries continue to occur in sports (Fig. 44.12). There are more than 30,000 annual visits to Emergency Departments in the United States secondary to ocular trauma [30]. Rules and regulations have been implemented to protect athletes from these maladies. However, whether the result of noncompliance, inadequate rules, or a lack of enforcement, eye trauma remains a concern for the sideline physician [31]. These injuries are first delineated based on whether the globe is closed (e.g., contusions, corneal abrasions, etc.) or open (e.g., rupture or laceration). Important structures of the eye to consider when assessing for sport-related injuries include:

Fig. 44.12 Ocular trauma due to a hockey puck to the eye

- Sclera: the white, outer layer of the eye. It is the thinnest aspect of the eye and the most likely site of a globe rupture.
- Limbus: the source of ocular stem cells. This structure is responsible for tasks such as replacing the epithelial cells of the eye damaged in a corneal abrasion.
- Aqueous humor: fluid of the anterior chamber that drains through the trabecular meshwork. It can build up in the setting of trauma (e.g., a hyphema), generating increased ocular pressure.

The following section will discuss some of the most serious ocular injuries that a sideline physician might encounter.

Globe Ruptures

Ruptured globe injuries are a common cause of blindness in ocular trauma. However, they may not be readily apparent on physical examination [32]. There are two types of globe rup-

tures: full-thickness injuries that pass through the cornea and sclera (open) and those that do not (closed), with the latter being less common [23]. Delays in getting athletes to definitive treatment in the operating room worsen the patient's prognosis for vision recovery.

Mechanism of Injury in Sports

Common causes of globe ruptures in sports include direct ball impact (e.g., racquetball to the eye), projectile penetration (e.g., an arrow), and a traumatic blow (e.g., an elbow or digit to the eye). Such injuries are also associated with falls, typically onto objects, which can obviously transpire during sporting activities.

Epidemiology

While reported to be relatively uncommon, the true incidence of globe rupture secondary to sport is unclear. Broadly, these injuries are more frequent in men (78.6%), attributed to their increased occupational and recreational risk. A relatively high percentage of these injuries occur in adolescent males [33].

Clinical Presentation

Athletes with a ruptured globe will often report significant pain and decreased visual acuity, which should be verified on physical examination. Patients may only be able to make out shapes in the affected eye. Other pertinent physical examination findings are listed in Table 44.4. It is imperative that the sideline physician not assess the patient's intraocular pressure or even apply significant pressure to the eye if a globe rupture is suspected, as this may further expel the intraocular contents. While open globe ruptures may reveal a collapsed or depressed eye, closed globe ruptures will not and may even result in an elevated intraocular pressure [34].

Diagnosis

The diagnosis of a globe rupture is typically based on clinical presentation. However, if a penetrating mechanism of injury is the cause of the rupture, a maxillofacial CT may be warranted to assess for the presence of an intraocular foreign body. Similar imaging is recommended if a concomitant orbital fracture is suspected. Ultrasound imaging is generally discouraged due to the risk of applying increased pressure to the injured eye.

Table 44.4 Clinical findings associated with ruptured globe injuries

Decreased extraocular movements	Surrounding ecchymosis and swelling
Scleral and/or corneal lacerations	Extrusion of intraocular contents
Significant subconjunctival hemorrhage (often 360°)	Teardrop pupil (Fig. 44.13)
Subluxed lens	Hyphema
Gray-white retina discoloration (commotio retinae)	Abnormal anterior chamber (deep or shallow)

Fig. 44.13 A teardrop pupil, which points to the site of injury in a ruptured globe (Image courtesy of Stack et al. [115])

Initial Management

The sideline physician presented with a possible globe rupture should avoid placing pressure on the affected eye. It should be shielded, preferably with a metal or plastic shield. Counsel the patient to avoid any Valsalva maneuvers, which will increase the intraocular pressure. If possible, providing analgesia and antiemetic therapy may be beneficial in the setting of these painful injuries.

Indications for Referral

All athletes suspected to have sustained a globe rupture should be immediately evaluated in an Emergency Department and/or by an Ophthalmologist. In the setting of an open globe rupture, a surgical repair should be pursued no later than 24 hours after the injury [34]. As such, a sideline physician should have a low threshold to refer for evaluation of this potential condition.

Return to Sports

Patients with a ruptured globe will face a prolonged return to sports (contact or otherwise), if one is possible at all. Decisions in this regard should be made on an individual basis in conjunction with the treating Ophthalmologist.

Complications

Permanent visual loss is the most obvious complication of a ruptured globe. The poorer the visual acuity at the time of the initial evaluation and the longer the time to the operating room (among other factors), the worse the prognosis. Other complications include retinal detachments, endophthalmitis, and/or glaucoma, all of which can occur after the initial injury is repaired [35].

Pediatric Considerations

An important pediatric prognostic consideration is that the younger the patient who suffers a globe rupture, the poorer the outcome [35].

Retrobulbar Hematoma

Retrobulbar hematomas represent true ocular emergencies. Often referred to as orbital compartment syndrome, they can result in irreversible damage to the eye after as little as 60 minutes of ischemia [36]. The underlying pathophysiology involves bleeding and edema within the orbit, which compresses the optic nerve and ophthalmic vasculature, leading to ischemia of the optic nerve and retina.

Mechanism of Injury in Sports

Orbital compartment syndrome can occur with blunt trauma to the eye and orbit. Typically it is associated with other facial fractures, although this is not always the case [37]. Contact sports that can precipitate orbital trauma are often implicated in these injuries, and fractures of the orbit should prompt the savvy sideline physician to consider this concomitant diagnosis.

Epidemiology

The overall incidence (not only in sport) of this condition is rare; it has been cited to be less than 1% in the general population. However, it carries with it a high risk of morbidity [38]. Thus, the prospect of a retrobulbar hematoma warrants consideration in the setting of sports-related facial trauma.

Clinical Presentation

There are several key physical examination findings that are associated with a retrobulbar hematoma. Pain out of portion to examination is often reported. Athletes may also note a sudden and significant decrease in visual acuity and sometimes alterations in color vision. Additionally, a sideline physician may appreciate an afferent pupillary defect, a dilated pupil, proptosis, a diffuse subconjunctival hemorrhage (Fig. 44.14), increased intraocular pressure (by either tonometry, "rock hard" eyelids, or increased resistance when attempting to push on the eye), periorbital ecchymosis, and/or restricted extraocular movements.

Fig. 44.14 A retrobulbar hematoma as indicated by the diffuse subconjunctival hemorrhage, dilated pupil, and proptosis (Image courtesy of lifeinthefastlane.com)

Diagnosis

The diagnosis of a retrobulbar hemorrhage should be entirely clinical. Time should not be spent obtaining further imaging and/or laboratory testing, as these tests can delay the definitive intervention (i.e., a surgical decompression). However, evidence of a retrobulbar hematoma may be present on a maxillofacial CT secured during the evaluation of another injury, which should prompt further evaluation for orbital compartment syndrome.

Initial Management

The initial management of this condition entails transporting the patient to the Emergency Department as soon as possible. En route, care can be taken to keep the athlete upright at 45° of elevation and to avoid Valsalva maneuvers [39]. Antiemetics may be beneficial if they are readily available.

Indications for Referral

All athletes with concern for a retrobulbar hematoma should be referred to the Emergency Department for immediate evaluation and treatment. Optimally, an Ophthalmologist should be awaiting the patient's arrival in the Emergency Department. Symptoms lasting for as little as 60 minutes can lead to irreversible damage. The definitive treatment for this condition is a lateral canthotomy/inferior cantholysis, which should be initiated as soon as the diagnosis is confirmed clinically. Of note, the sideline physician should also consider orbital comaprtment syndrome in the setting of orbital fractures, which too should result in a referral for prompt evaluation.

Return to Sports

Patients with a retrobulbar hematoma will face a prolonged return to sports (contact or otherwise), if one is possible at all. Decisions in this regard should be made on an individual basis in conjunction with the treating Ophthalmologist.

Complications

The primary complication of orbital compartment syndrome is permanent vision loss. The lateral canthotomy/inferior cantholysis can result in drooping of the lower eyelid, although this can be repaired in the future [40]. Otherwise, the patient will need to be observed for post-procedural infections and/or abscesses.

Hyphema

A hyphema is defined as blood in the anterior chamber of the eye between the iris and the cornea. This typically

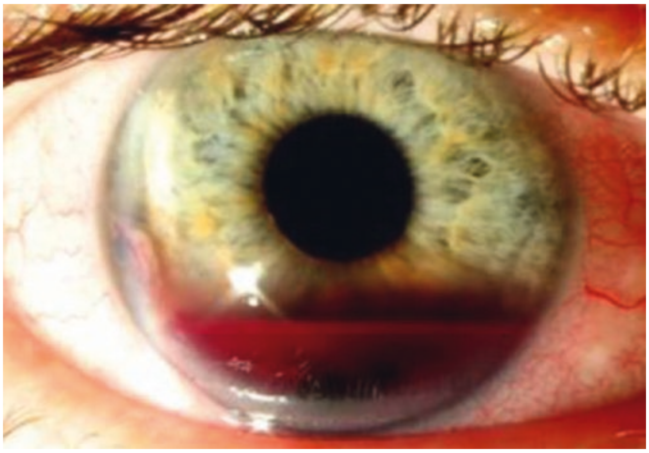

Fig. 44.15 A grade I hyphema with layering blood in the anterior chamber (Image courtesy of EyeRounds.org – University of Iowa)

results in grossly visible layering of blood, although there can be red blood cells in this chamber only detectable via slit-lamp examination as well (microhyphema) (Fig. 44.15).

Mechanism of Injury in Sports

Approximately 70–80% of the time this occurs via blunt trauma to the eye [12]. Common sports-related mechanisms include a ball directly to the eye (e.g., tennis ball or racquetball) or a blow to the eye (e.g., boxing or mixed martial arts). The injury is secondary to tearing of the vessels of the ciliary body, iris, and other anterior structures within the eye.

Penetrating trauma can result in a hyphema as well. Certain risk factors that can contribute to this condition include diabetes mellitus, clotting disorders, uveitis, and the use of anticoagulants or medications that inhibit platelet function (e.g., warfarin, aspirin, other NSAIDs, etc.). Sickle cell disease has been associated with an increased risk for such an injury.

Epidemiology

The peak incidence of hyphemas is between the ages of 10 and 20 years old. However, 77% of cases occur in those over the age of 30. These injuries are three to five times more common in males. Overall, hyphemas occur at a rate of approximately 12 per 100,000 [41].

Classification

The grading of hyphemas provides prognostic value. Grades are assigned based on the amount of blood that fills the anterior chamber (Table 44.5).

The higher the grade of hyphema, the higher the risk of rebleeding, developing glaucoma, permanent visual acuity loss, and/or permanent corneal staining.

Table 44.5 Clinical grading of hyphemas

Grade	Description
I	<1/3 of the anterior chamber
II	1/3 to 1/2 of the anterior chamber
III	>1/2 of the anterior chamber
IV	≈ 100% filled (also called an 8-ball hyphema or blackball)

Clinical Presentation

In addition to a history of recent trauma to the eye (which occurs in the majority of patients), assess for photophobia, blurry vision, decreased visual acuity, ocular pain, nausea, and/or vomiting.

Examination of a suspected hyphema includes evaluating for anisocoria (unequal pupil size). Ocular motility and confrontation are also important components of the assessment. Next, measuring the athlete's visual acuity can be remarkably valuable. A pocket Snellen chart is often sufficient for this evaluation and can help to assess for changes in an athlete's status. At this point (and perhaps sooner), a topical anesthetic (e.g., tetracaine or proparacaine) can help to facilitate the remainder of the evaluation. If possible, try to perform a funduscopic examination. It may be prudent to evert the eyelids to assess for the presence of a foreign body. Direct visualization of the anterior chamber will frequently reveal layering of blood in the anterior chamber.

Given that the most common mechanism of injury involves blunt trauma, it is important to assess for concomitant, potentially more serious, injuries as well. A ruptured globe is chief among these other injuries. However, if there is low suspicion for a globe rupture, the intraocular pressure can be diagnostically valuable. It will be elevated (>21 mmHg) more than 30% of the time in the setting of a hyphema [42].

Diagnosis

The physical examination is generally sufficient for making the diagnosis of a hyphema, and this can often be accomplished on the sideline or in the training room. Laboratory testing is generally not required in the absence of a suspected coagulation disorder. However, if the mechanism of injury does not seem significant enough to result in a hyphema, consideration should be given to pursuing further testing. Underlying (and/or undiagnosed) diabetes mellitus, clotting disorders, and/or tumors should be considered in these instances. Imaging (including orbital CT scans and/or an ultrasound) is typically reserved for the investigation of another associated injury.

Initial Management

Once a hyphema is diagnosed, a rigid shield should be applied to the eye. Ideally, the shield should contain holes to allow for the patient to determine if visual change/loss develops. Athletes should be advised to minimize the movement

of the eye as much as possible. The avoidance of coughing, sneezing, and/or other straining should be encouraged. The patient's head should be kept elevated, optimally to approximately 30° to 45°, which will help to keep the blood in the inferior-most portion of the anterior chamber. Avoid the administration of NSAIDs, and do not allow the patient to eat and/or drink anything (in case a surgical intervention is deemed necessary). Athletes should then be referred for immediate Ophthalmologic evaluation (e.g., in an Emergency Department or Ophthalmology Clinic).

Indications for Referral

The current recommendations for immediate evaluation include anything greater than a grade I hyphema and/or a patient with increased intraocular pressure. The assessment of intraocular pressure may not always be feasible on the sideline or in the training room. Thus, in the presence of a hyphema with an unknown pressure, immediate evaluation is recommended. Any concern for a concomitant injury (e.g., globe rupture, orbital fracture, retinal detachment, retrobulbar hematoma, etc.) should warrant an immediate evaluation as well.

Follow-up Care

Grade I hyphemas are generally managed as an outpatient. However, it should be noted that the intraocular pressure is typically more important than the grading in determining whether or not a patient is admitted to the hospital. Roughly 5% of all hyphemas will require a surgical intervention [8]. Ophthalmology should help to guide the follow-up as a component of the athlete's Emergency Department evaluation. Repeat slit-lamp evaluation for the first 3 days after the diagnosis is the norm with the subsequent duration of treatment contingent on the improvement (or lack thereof) in bleeding and/or intraocular pressure.

Return to Sports

A return to sports should not transpire until the athlete is cleared by an Ophthalmologist, typically after the hyphema resolves. For a grade I hyphema, resolution can occur in 4–5 days. However, larger hyphemas may take longer. Ultimately, the athlete must be comfortable with his/her visual acuity. Additionally, adequate eye protection should be worn moving forward in all sports at risk for eye injuries.

Complications

A significant complication of a hyphema is rebleeding, which typically occurs between 2 and 5 days after the initial injury. This can arise in up to 18% of patients [43]. Generally, the higher the grade of hyphema or the greater the initial intraocular pressure, the higher the risk of rebleeding. This secondary hyphema is often more significant than the initial injury. Visual acuity loss also increases as the severity of the hyphema increases (20/50 vision or worse in 50–75% of grade III or grade IV hyphemas) [42]. Lastly, corneal staining can occur as a result of a hyphema. The corneal stroma becomes opacified, affecting vision in larger hyphemas (Fig. 44.16). While this can be permanent, it will often resolve in months to years. However, it clears peripherally first, leaving the impact on vision remaining until toward the end of the healing process. Risk factors for this complication include persistent hyphemas and those associated with intraocular pressures >25 mmHg.

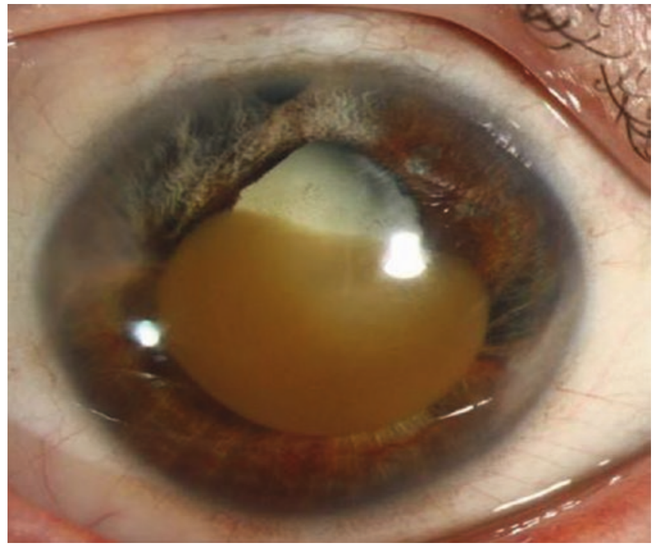

Fig. 44.16 Corneal staining due to a hyphema (Image courtesy of EyeRounds.org – University of Iowa)

Pediatric Considerations

Pediatric patients are at a particularly high risk for rebleeding. Also keep in mind that the accuracy of the physical examination may be decreased in young children, which may alter one's threshold for evaluation in an Emergency Department and/or Ophthalmology Clinic. It is typically recommended that children younger than 7 years old be admitted, as they are at a higher risk of developing amblyopia [12]. Corneal staining in children increases the risk of amblyopia and can result in permanent vision loss.

Retinal Detachment/Tear

The retina is responsible for converting photons into neural impulses that travel to the visual cortex. This structure can be detached or torn, leading to a surgical emergency (Fig. 44.17). While there are three types of retinal detachments, rhegmatogenous retinal detachments (RRD) are the most relevant to the care of athletes as they tend to result from direct trauma to the eye. Tractional retinal detachments (TRD) can occur with penetrating injury. The third type (exudate retinal detachments) are less germane to athletes.

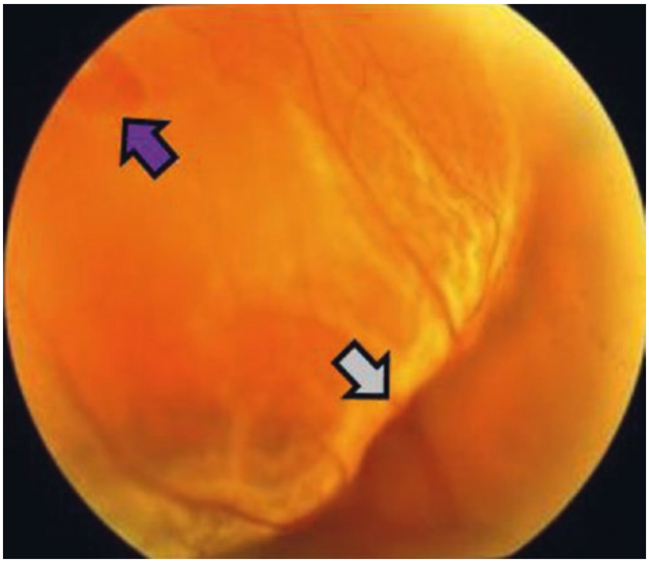

Fig. 44.17 A retinal tear (purple arrow) and a retinal detachment (gray arrow) via funduscopic evaluation

Mechanism of Injury in Sports

Virtually any sport carries with it the risk of experiencing a retinal detachment. Collisions, inherent in the sport or incidental, can result in trauma to the eye and thus a retinal detachment or tear. With that said, it should be noted that an RRD is the most common type of retinal detachment/tear, and the majority of these are spontaneous. Additionally, there has been a reported link to oral fluoroquinolone use [44].

Epidemiology

Overall, the incidence of retinal detachments/tears increases with age. There is also a slight predilection for males over females (approximately 3:2). However, it is unclear whether these epidemiologic characteristics apply to sports-related retinal injuries in the same manner as they do the general population. Athletes with Marfan syndrome are at an increased risk for an RRD, while sickle cell retinopathy can lead to a TRD.

Clinical Presentation

While some athletes may have a traumatic mechanism of injury, retinal detachments and tears can occur spontaneously as well. The most common symptom reported by patients is the presence of flashers (a response to traction) or floaters (condensation in the vitreous gel that produces shadows) [45]. Visual defects may be present, and remember that a superior visual field injury results in an inferior visual field defect. The evaluating team physician can use this information to better guide the funduscopic examination. Athletes may also report the sudden, complete loss of vision. A thorough eye examination is essential, the components of which are noted in Table 44.6.

Table 44.6 Key components of the sideline ocular physical examination

Visual acuity (monocular and binocular)	Visual field confrontation
Anterior chamber evaluation (with a penlight)	Extraocular movement
External exam (including eyelid eversion)	Funduscopic examination (Fig. 44.17)

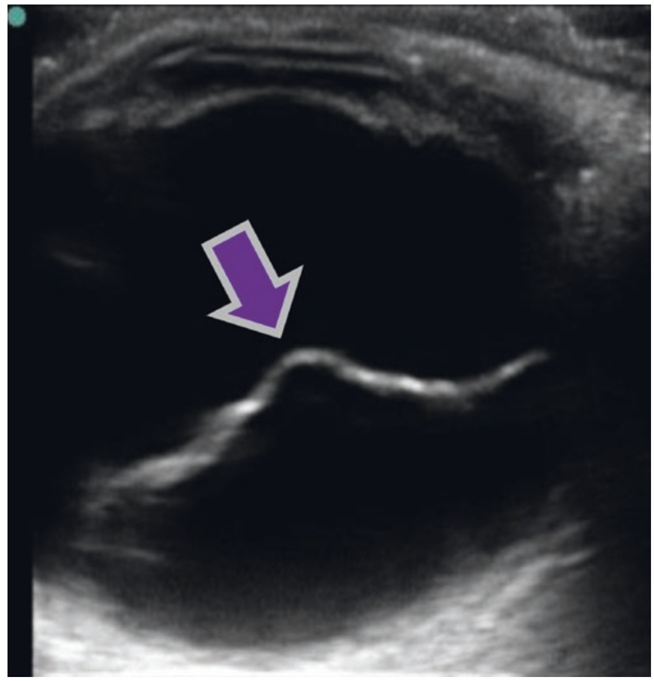

Fig. 44.18 An ocular ultrasound of a retinal detachment (purple arrow); note the lens at the top of the image

Diagnosis

The diagnosis of a retinal detachment and/or tear is most commonly made on physical examination. A dilated exam in the hands of an Ophthalmologist is the best manner by which to confirm the diagnosis. While further advanced imaging (e.g., an orbital CT scan) is not a component of the evaluation (barring a suspected associated injury), ultrasound can be useful in making the diagnosis (Fig. 44.18). Studies suggest that emergency physicians can detect retinal detachments using bedside ultrasound with 97% sensitivity and 92% specificity [46].

Initial Management

No immediate sideline management of retinal detachment or tears is necessary. They require prompt evaluation by an Ophthalmologist.

Indications for Referral

Any time a team physician suspects or diagnoses a retinal detachment or tear, a prompt Ophthalmology evaluation should be pursued. Of note, involvement of macula-sparing

tears or detachments is fixed immediately. However, not all detachments or tears will necessitate such a prompt surgical intervention. The nuances of that decision are likely beyond the scope of the team physician.

Follow-up Care

Rhegmatogenous retinal detachments (RRDs) typically involve the macula and are generally repaired within 3–7 days. The traction retinal detachments (TRDs) are often corrected 1–2 weeks after the injury. Meanwhile, the final category, or exudative retinal detachments (ERDs), tend to resolve on their own. Team physicians need not to be able to detect the specifics of each retinal detachment subcategory on physical examination, but it is important to consider the timeline of the associated injury moving forward.

Return to Sports

As with several other ocular injuries, the return to sport following a retinal detachment is variable. The athlete must be comfortable with his/her visual acuity and receive clearance from the treating Ophthalmologist. Eye protection in the future should be strongly encouraged, if not required.

Complications

The most noteworthy complication of a retinal detachment or tear is long-term, permanent vision loss. There are potential complications following a repair as well, including vitreous hemorrhage and/or cataracts, although they are of lesser immediate concern in the setting of this injury.

Corneal Abrasion/Ulcer

Corneal abrasions are among the most common sports-related ocular injuries, which can progress to corneal ulcers if they do not heal or are improperly treated. For instance, 10% of ocular injuries in the National Basketball Association (NBA) are due to corneal abrasions, while they account for greater than 20% of ball-related ocular injuries in amateur soccer athletes [47]. These injuries are due to the traumatic (or chemical in some instances) removal of the superficial epithelial layer overlying the cornea. Corneal ulcers occur when the injuries are more severe and deeper or when infection sets in, leading to penetration of the thicker Bowman's layer (Fig. 44.19). Since the cornea has one of the highest densities of nerve endings in the body, these injuries are often quite painful [48].

Mechanism of Injury in Sports

Collision sports and those that involve projectiles (e.g., balls) present the greatest risk for corneal abrasions or ulcers. A mechanism as seemingly innocuous as a poke to the eye can result in this painful injury. Commonly offending sports include wrestling, martial arts, boxing, basketball, and soccer.

Epidemiology

There is a relative lack of data on the incidence of corneal abrasions and/or ulcers secondary to sport. However, it has been reported that it is the second most common type of eye injury broadly in this context (after soft tissue injuries) [8]. Athletes who wear contact lenses are at a higher risk, particularly if they wear soft lenses. Additionally, it should be noted that protective eyewear markedly reduces the risk of such injuries.

Clinical Presentation

Prominent features of a corneal abrasion include pain immediately after the insult, a foreign body sensation, photophobia, and/or tearing. Athletes who typically wear contact lenses may report that the associated pain is improved when wearing the lenses, as this may serve as a bandage (although it should not be used as such in treatment). The physical examination of the eye should include the maneuvers highlighted in Table 44.6. Of particular importance, the team physician should be sure to thoroughly inspect both the upper and lower eyelids, including eyelid eversion. Retained foreign bodies can become trapped, particularly in the upper eyelid. Failure to appreciate this finding will result in repeated and likely persistent corneal epithelial defects,

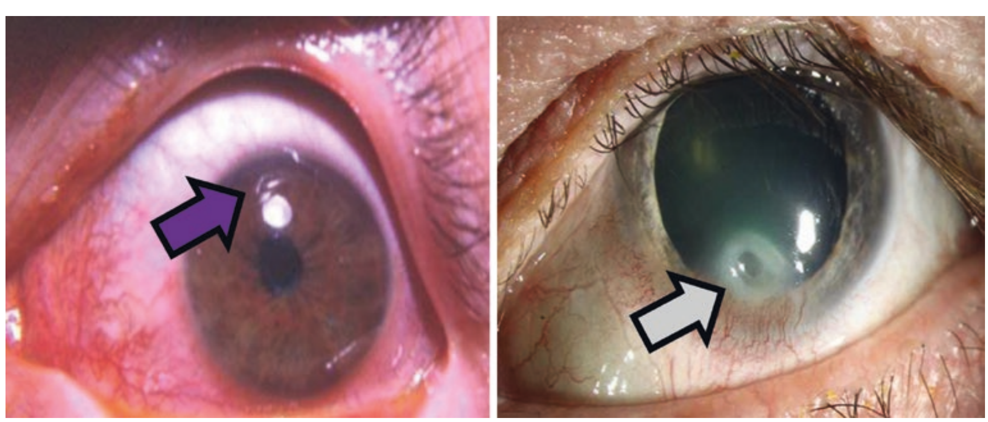

Fig. 44.19 A corneal abrasion (purple arrow) without fluorescein staining is noted on the left; a deeper corneal ulcer (gray arrow) is illustrated on the right, highlighted by its white, scaly appearance (Images courtesy of EyeRounds.org – University of Iowa)

Fig. 44.20 Technique for everting the upper eyelid in search of foreign bodies, in this case revealing a small pustule (purple arrow)

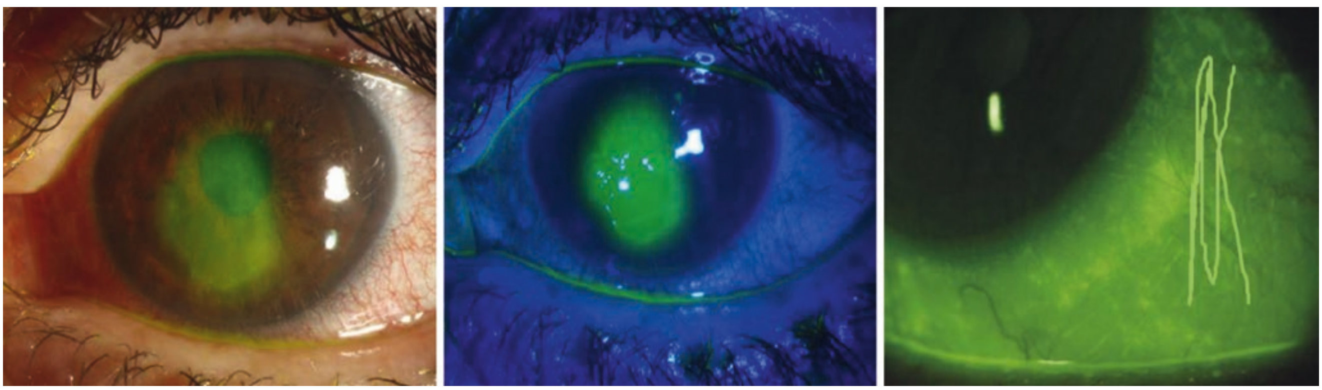

Fig. 44.21 A corneal abrasion visualized with fluorescein staining (left) and with such staining under a cobalt blue light or Woods lamp (middle; both images courtesy of EyeRounds.org – University of Iowa); the "ice rink sign" (right) can occur when repeated, linear abrasions result from a foreign body retained under the eyelid. The fluorescein-uptake lines can mimic the lines on a hockey rink

which may lead to a corneal ulcer. One can assess the undersurface of the upper eyelid by utilizing a cotton-tipped applicator (Fig. 44.20). Ask the athlete to look downward and then grasp the patient's eyelashes and upper lid in one hand, applying gentle traction down and out away from the patient. Next, insert the cotton-tipped applicator just superior to the globe at the tarsal plate. By moving the eyelashes and upper eyelid margin toward the superior orbital rim, the team physician can successfully evert the eyelid and assess for the presence of foreign bodies. Eversion of the lower eyelid can be achieved with simple, gentle downward traction.

In the absence of concern for a ruptured globe, the intraocular pressure should be noted. Lastly, a fluorescein examination should be completed. Strips of fluorescein (or fluorescein-containing anesthetic drops) should be included in a team physician's ophthalmologic toolkit and utilized in conjunction with a blacklight flashlight (at minimal cost) or the cobalt filter on an ophthalmoscope. Fluorescein demarcates the borders of a corneal abrasion and/or identifies other ongoing disease processes (e.g., herpes keratitis). In the setting of a retained foreign body under the eyelid, one might appreciate fluorescein uptake in linear lines, referred to as the "ice rink sign" (Fig. 44.21).

Diagnosis

The diagnosis of a corneal abrasion and/or ulcer is clinical. Further advanced imaging (e.g., an orbital CT scan or an ultrasound) should be entertained if there is concern for an associated orbital fracture, a retained foreign body, and/or other associated injury.

Initial Management

Uncomplicated, small corneal abrasions secondary to trauma can be managed safely by a team physician without immediate Ophthalmologic consultation. Treatment entails prophylactic topical antibiotics, with ointment theoretically superior to drops given its stronger lubricating properties [49]. Erythromycin ointment is a sound choice, typically prescribed four times per day for 1 day longer than the patient's symptoms persist (roughly 3–5 days) [49]. For patients who insist upon drops, sulfacetamide, polymyxin/trimethoprim, ciprofloxacin, and ofloxacin are reasonable. In the case of

contact lens wears, the antibiotic selected should cover for *Pseudomonas* such as ciprofloxacin, ofloxacin, and gentamicin. In addition, athletes should be instructed not to wear contact lenses during the course of treatment. Otherwise, avoid preparations containing steroids given the risk of delayed healing.

Pain control for these athletes is an important component of management. Oral NSAIDs are a reasonable option, and topical ophthalmic NSAIDs (e.g., ketorolac or diclofenac) are acceptable as well. Topical preparations do not appear to adversely impact healing, although they may be painful to apply and/or expensive [50]. There is also evidence that in the setting of small corneal abrasions, topical tetracaine can be utilized for up to 24 hours without adverse outcomes [51]. Patching may help to alleviate pain, but this is primarily reserved for large abrasions and in conjunction with an Ophthalmology consultation [35].

Lastly, eye protective strategies for the athlete with a corneal abrasion are recommended. Participating in eye rest (e.g., avoiding activities that can result straining of the eye such as computer use), wearing sunglasses, and avoiding rubbing or direct contact with the eye will help to ensure healing in a timely fashion.

Indications for Referral

The presence of concomitant injuries (e.g., hyphema, orbital fracture, ruptured globe, etc.) necessitates an immediate referral to an Ophthalmologist (or an Emergency Department with the ability to consult with such a specialist). Additional indications to seek prompt further evaluation and treatment are provided in Table 44.7.

Recurrent corneal abrasions warrant an outpatient referral as well to identify any potential underlying etiologies (e.g., recurrent erosion syndrome).

Follow-up Care

Corneal abrasions generally heal quickly, with smaller injuries resolving in 24–48 hours. These abrasions do not require follow-up unless the associated symptoms persist for more than 3–4 days. It is wise for the team physician to follow-up with the athlete in this window to ensure complete resolution of symptoms. Athletes with large abrasions (>50% of the corneal surface area) or those that wear contact lenses should be reassessed daily until their symptoms have resolved and reepithelialization has occurred, typically within 1 week.

Table 44.7 Indications for immediate ophthalmologic referral in corneal abrasions

Large epithelial defects	Corneal ulcers
Herpes keratitis (dendritic fluorescein stain pattern)	Intraocular foreign body concern
Presence of a rust ring	Concomitant ocular infection

Return to Sports

The athlete can return to sport as soon as his/her visual acuity returns to baseline and the associated pain is controlled. This will commonly take at least 1–2 days for smaller abrasions and up to a week for more substantial injuries. Athletes should not utilize a topical anesthetic in an attempt to expedite a return to sport.

Complications

A concerning complication associated with corneal abrasions is the development of a corneal ulcer. Often the result of a subsequent viral or bacterial infection, corneal ulcers penetrate deeper through the layers of the cornea. Scar formation can subsequently occur, and permanent vision loss may also result. There is a phenomenon known as recurrent erosion as well, which may not be related to another traumatic insult. This entity may require debridement of the corneal tissue.

Pediatric Considerations

When selecting a type of antibiotic for the prophylactic treatment of corneal abrasions, remember that the drops are more irritating than the ointment. The latter is generally better tolerated in the pediatric population. In children, if there is persistent drainage or an unwillingness to keep the eye open 24 hours after the injury, an Ophthalmology consult should be pursued.

Ear Trauma

Key Points

- Delays in identifying or adequately evacuating an auricular hematoma can result in permanent disfigurement (Fig. 44.22).
- There are several methods for properly and sufficiently draining an auricular hematoma; a sideline physician should be comfortable with at least one such method.
- Tympanic membrane injuries may result in significant pain and temporary hearing loss.
- A tympanic membrane rupture should raise one's suspicion for a temporal bone fracture as well.

Introduction

The ear is an extremely complex structure that can be divided into the external, middle, and inner ear. The pinna and external auditory canal comprise the external ear, whereas the middle ear serves as the home of the tympanic membrane, malleus, incus, and stapes. This middle ear machinery subse-

Fig. 44.22 Wrestling, particularly without the use of headgear, can result in significant and potentially permanent ear trauma

quently propagates vibrations to the inner ear to produce sound. The inner ear itself is composed of the cochlea and labyrinth. A disruption of any of these components can detrimentally impact one's hearing. This section will focus on noteworthy sports injuries involving the external and middle ear, as a timely diagnosis of these conditions will help to reduce the risk of potential permanent cosmetic deformities and/or hearing loss.

Auricular Hematomas

An auricular hematoma is a collection of blood in the subperichondrial space of the external ear (Fig. 44.23). These almost exclusively occur on the anterior surface of the pinna [52]. If untreated, this can result in pressure necrosis within the cartilage of the ear as the accumulating blood prevents nutrient delivery to the cartilage. When combined with neocartilage formation and fibrosis, permanent disfigurement can occur. It is critical that these injuries be identified promptly and treated adequately via drainage and occasionally incision.

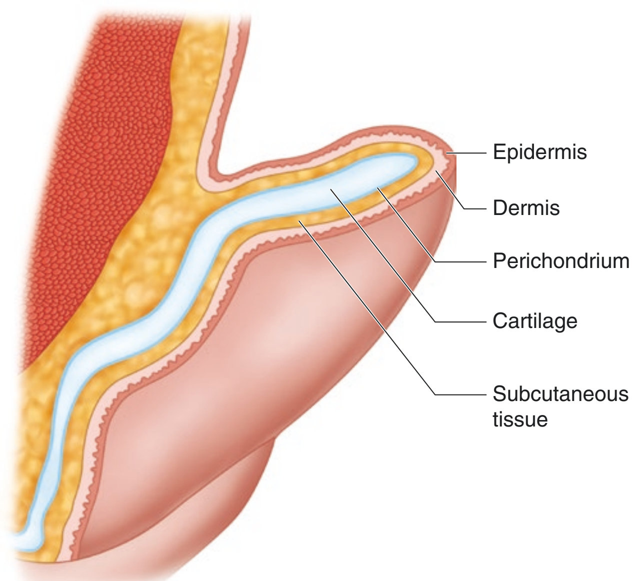

Fig. 44.23 The layers of the external ear presented in cross section

Mechanism of Injury in Sports

Auricular hematomas are typically the result of direct, blunt trauma and/or friction/shearing to the ear. This causes bleeding between the perichondrium and elastic cartilage of the ear. Contact sports are typically implicated in this injury, including wrestling, mixed martial arts, judo, boxing, and rugby.

Epidemiology

Overall, the exact prevalence is unknown given a lack of historical recording. However, these injuries are much more prev-

alent in young males due to their increased participation in the aforementioned sports, particularly wrestling. Regardless, they do affect both genders. In one study, collegiate wrestlers who wore headgear (26%) were less likely to suffer an auricular hematoma compared to those that did not (52%). Overall, wearing properly fitting headgear consistently can decrease the incidence of this condition by 50% [53]. Improperly fitted headgear, however, may not prevent this injury.

Clinical Presentation

The diagnosis of an auricular hematoma is made based on clinical history and physical examination. Often, a history of direct trauma to the auricle is noted, but this is not always the case. Athletes will frequently note a painful ear, and hearing loss may also be reported. The lack of headgear use should raise one's suspicion for such a condition. Additionally, athletes with chronic cartilage deformities, often referred to as "cauliflower ear", can still develop an acute auricular hematoma. The sideline provider should be able to discern the difference and be prepared to intervene on the fluctuant, tender, ecchymotic acute injury while avoiding aspirating or incising the chronic malady (Fig. 44.24). If there is any question about the acuteness of the injury, assessing the contralateral ear for contour and/or tactile differences may be beneficial. The athlete's external auditory canal and tympanic membrane should be thoroughly evaluated as well.

Diagnosis

A careful physical examination is generally all that is needed to facilitate the prompt diagnosis of an auricular hematoma. Ultrasound can be helpful in differentiating an acute on chronic hematoma from a chronic cartilage deformity. No further laboratory testing and/or imaging will prove beneficial in the diagnosis of this condition for the majority of athletes.

Initial Management

Management of an auricular hematoma should be within the skillset of a team physician. Care should be taken to perform this procedure in as sterile a fashion as possible to avoid complications such as chondritis, perichondritis, and/or an abscess formation. Betadine and/or chlorhexidine should be utilized to help prevent these conditions. The administration of anesthesia constitutes the next step, and it can be achieved through either a local injection or a ring block, either option employing lidocaine or bupivacaine. The ring block provides anesthesia to the entire ear and may facilitate a procedure that is better tolerated for the patient. As shown in Fig. 44.25, anesthetic is injected both superior to the attachment of the helix to the scalp both anteriorly and posteriorly (addressing the auriculotemporal nerve) and inferior to the ear anteriorly and posteriorly (anesthetizing the greater auricular nerve). Remember that the facial nerve courses

Fig. 44.24 The more fluctuant, tender, acute auricular hematoma (left; image courtesy of Otolaryngology Houston; www.ghorayeb.com) versus the firm, non-tender "cauliflower ear" (right; image courtesy of Skidmore K, Hatcher JD. Cauliflower ear. *StatPearls*. 2018 Dec 9)

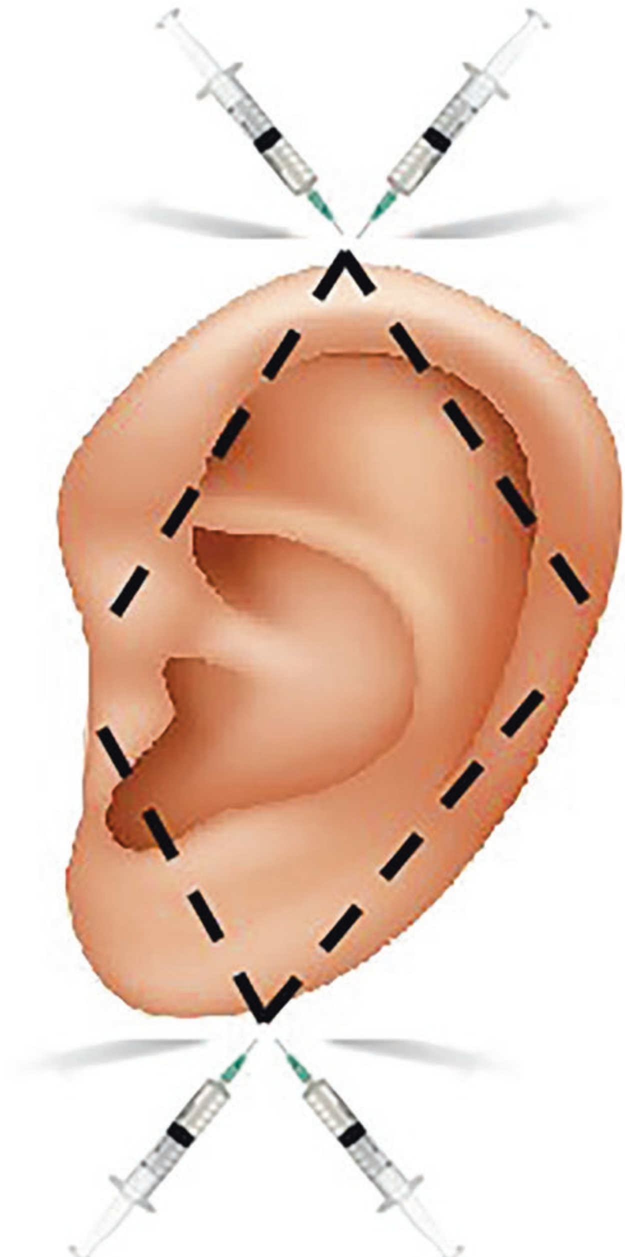

Fig. 44.25 The approach for a ring block of the ear; note that the injection should be administered behind the auricle

through this territory (anterior and inferior to the tragus); accordingly, paresis of this nerve may occur. The inclusion of epinephrine with the anesthetic for certain anatomic sites (including the ear) continues to be a somewhat contentious topic. It has been argued in the literature that no cases of vasoconstrictor-induced tissue necrosis have been reported since commercial lidocaine with epinephrine was introduced in 1948 [54]. As such, it is most likely safe to use lidocaine with epinephrine in this context, particularly if implementing a ring block.

Fig. 44.26 The incisional landmarks for an auricular hematoma drainage; note the superior incision along the curve of the anterior pinna with a potential incision site along the conchal bowl inferiorly (if necessary)

The technical manner in which the remainder of the procedure is approached depends on both the size of the auricular hematoma and the timing of presentation. If the hematoma is <2 cm in diameter and presents within 48 hours, needle aspiration is appropriate. There may be a risk of reaccumulation of the hematoma with this approach, but studies have not found this to be statistically significant [55]. With this approach, the most fluctuant area of the hematoma should be identified. Using an 18- or 22-gauge needle, aspirate the contents of the hematoma while also milking the area to ensure complete drainage. Direct pressure should then be applied to the region for 5–10 minutes, followed by the application of a pressure dressing (discussed below). Of note, this technique has been described using an intravenous catheter as well. Specifically, an 18-gauge catheter is utilized to aspirate the hematoma, and the catheter is left in place. Trimmed down to a length of approximately 1 cm, the catheter tip remains to facilitate further drainage [56].

For larger auricular hematomas (>2 cm) or those that present after more than 2 days, an incisional approach is recommended. With an 11-blade scalpel, a superficial, curvilinear incision should be made along the natural curve of the anterior pinna. This may require two incisions, with the other in the conchal bowl [52] (Fig. 44.26). The hematoma can then be evacuated with gentle pressure, and thorough irrigation should then follow. Finally, pressure dressings should be applied.

The second component of the successful treatment of an auricular hematoma involves an adequate pressure dressing. For hematomas treated via needle aspiration, one should place gauze around the ear, which can be achieved by cutting out the center of the gauze and draping it around the ear.

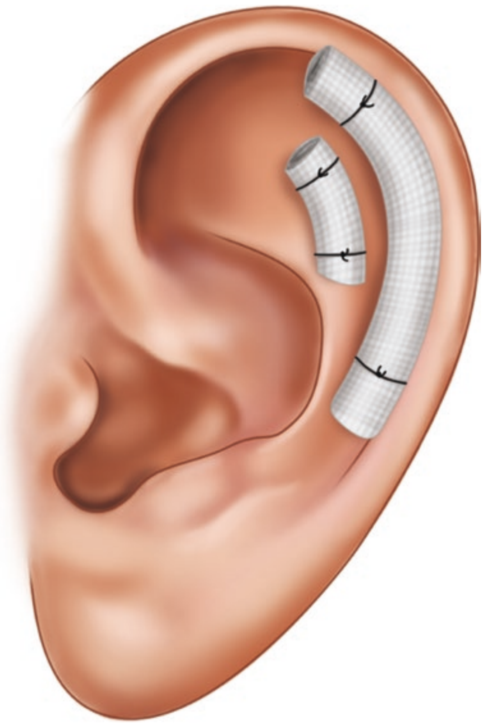

Fig. 44.27 A pressure dressing applied after an auricular hematoma needle aspiration (Image courtesy of Michelle Lin, MD; aliem.com)

Petroleum gauze, saline-soaked cotton balls, or plaster surrounded in padding should then be molded to the inner contours of the auricle (Fig. 44.27). Sterile gauze is then placed over the entire ear, and a head dressing (often with Kerlix gauze) is applied over the top of the ear and around the head to keep the entire dressing in place.

The post-procedural dressing for auricular hematomas requiring an incision is a bit more complex. It is appropriate to use absorbable or non-absorbable sutures to reapproximate the incision edges, leaving a small, dependent area open to drain. However, it is often more advantageous to apply bolsters. Petroleum gauze or dental rolls on either side of the ear are sutured into place via through-and-through non-absorbable suture (e.g., 3-0 or 4-0 nylon). Care should be taken to ensure that the sutures are tight enough to prevent reaccumulation but not too tight to cause tissue necrosis. The sutures are typically left in place for 5–7 days. If a conchal bowl incision is also made, the area can be packed with petroleum gauze.

Antibiotic prophylaxis is advised following this procedure, although the supporting evidence for this practice is not robust. Historically, coverage of *Pseudomonas* has been recommended for 7–10 days. The first-line treatment in those older than 18 has been a fluoroquinolone. However, given the increased risk of tendon damage with this class of antibiotics, fluoroquinolones may be suboptimal in athletes. Amoxicillin-clavulanate is a reasonable choice to cover against skin flora infection despite its lack of efficacy against *Pseudomonas*. This is also the preferred prophylactic antibiotic in this setting for athletes under the age of 18.

Indications for Referral

This is a condition that is within the scope of a sideline physician. However, auricular hematomas greater than 1 week old may contain granulation tissue and warrant a Plastic Surgery or Otolaryngology referral [57]. Also, if concern for a post-decompression cosmetic deformity or other complication exists, a referral is indicated to improve the cosmetic outcome.

Follow-up Care

These patients should be re-evaluated daily for the first 3–5 days to assess for evidence of reaccumulation of the hematoma, pressure necrosis, and/or infection. If sutures were placed as a component of the compressive dressing, they should be removed within approximately 7 days.

Return to Sports

There is some debate as to the proper return to sport for athletes following the treatment of an auricular hematoma. Some have argued for no activity that places the ear at risk for additional trauma until it is healed, which can occur as soon as 7 days [56, 58]. Others contend that athletes should be allowed to participate in full activity with a compressive dressing and proper ear protection [59]. The ultimate decision should be made in consultation with the athlete (and parents when appropriate) after discussing the potential complications of returning to sport before complete healing has transpired. Regardless of the timing of the return to sport, athletes should be strongly encouraged to wear protective headgear moving forward.

Complications

As previously referenced, permanent cosmetic deformity can occur with these injuries. This condition, often referred to as “cauliflower ear”, can develop as new cartilage is formed that is associated with scarring and tissue necrosis. Risks for this complication include failure to promptly recognize and treat an auricular hematoma, insufficient drainage, repeated need for aspiration, and a premature or early return to sport [60]. Additional potential complications include an infection of the auricular cartilage, referred to as chondritis or perichondritis. Subsequent abscesses can also develop in the aftermath of an auricular hematoma.

Tympanic Membrane Ruptures

The sudden onset of ear pain in conjunction with decreased hearing in an athlete can be disconcerting to both the ath-

Fig. 44.28 A direct blow to the ear can result in a tympanic membrane rupture

lete and the sideline physician. A rupture of the tympanic membrane can account for this precise scenario and can be skillfully treated in most cases by a sideline physician. The tympanic membrane is responsible for conductive hearing, amplifying sound by a factor of 17; thus, damage to it may be readily apparent clinically [61]. While the majority of these injuries heal well without complication, a sideline physician should always consider other significant concomitant injuries, particularly a temporal bone fracture.

Mechanism of Injury in Sports

Tympanic membrane ruptures can occur secondary to a variety of mechanisms. Barotrauma is one such mechanism of injury, which can result from scuba diving or mountain climbing. Trauma to the side of the head, such as in boxing (Fig. 44.28), can rupture the tympanic membrane as well. While a less frequent occurrence in sports, direct trauma to the membrane could lead to its compromise. An underlying otitis media should be considered as a contributing component in the setting of an athlete's tympanic membrane rupture as well.

Epidemiology

There is no epidemiological data regarding the incidence or prevalence of tympanic membrane ruptures in athletes specifically. In fact, it has not been well studied in the general population either, although estimates of traumatic tympanic membrane perforations suggest a rate of 6.8 cases per 1000 individuals [62].

Clinical Presentation

There are a constellation of signs and symptoms that should raise a team physician's suspicion for a tympanic membrane perforation (Table 44.8). Recall that infection remains a leading cause of tympanic membrane rupture, and thus assessing for infectious symptoms is advised. If the patient notes significant pain and/or complete hearing loss, the pursuit of additional, more significant injuries should be initiated.

Table 44.8 The signs/symptoms of tympanic membrane rupture

Mild pain	Partial hearing loss
Purulent/bloody drainage	Tinnitus
Vertigo	Otorrhea
Nausea	Vomiting

Fig. 44.29 A central tympanic membrane perforation (Image courtesy of Sean Flanagan, MBBS, FRACS)

A complete otic physical examination is essential to make this diagnosis. Direct visualization of the tympanic membrane with an otoscope will often reveal the perforation (Fig. 44.29). One can also use insufflation if there is doubt. The tympanic membrane should not move if ruptured or perforated. Also, if one holds pressure on the tragus, nystagmus or vertigo may ensue [8]. Called the fistula sign, this occurs due to the transmission of pressure through the middle ear into the labyrinthine fistula. The team physician should also

be sure to check the hearing in both ears. On a related note, Weber testing may also be of some utility. By placing a tuning fork on the frontal bone, hearing should be louder in the injured ear (suggesting a conductive hearing impairment). Conversely, if the hearing is louder in the unaffected ear, there may be an otic nerve injury. Lastly, one should palpate the bones surrounding the ear, particularly the temporal bone, to assess for tenderness suggestive of a concomitant injury.

Diagnosis
Properly diagnosing a ruptured tympanic membrane generally only requires a thorough physical examination. If there is concern for a temporal bone fracture, a temporal bone CT scan should be secured.

Initial Management
Isolated, uncomplicated tympanic membrane ruptures can be treated effectively in the training room and/or office. The vast majority of these injuries heal without complication or further intervention, with 68% resolved within 1 month and 94% healed at the 3-month mark [8]. Athletes should be counseled to keep the auditory canal dry to minimize the risk of infection. Otic antibiotic drops are recommended, with ofloxacin being a reasonable choice. Otic preparations of fluoroquinolones carry a substantially lower risk to tendons than systemic therapies [63]. An appropriate treatment regimen consists of four to five drops in the affected ear twice daily for 1–2 weeks. However, the evidence supporting the use of prophylactic antibiotics for tympanic membrane perforations is limited. One study did suggest that ofloxacin drops shortened the closure time and improved the closure rate of such injuries but did not affect hearing improvement or the rate of middle ear infections [64].

Indications for Referral
While most tympanic membrane ruptures can be handled without referral, it is important to recognize which signs and/or symptoms warrant an immediate evaluation by a specialist. Patients with marked or complete hearing loss and/or facial/otic nerve involvement should be seen by an Otolaryngologist promptly. Concern for a basilar skull fracture (e.g., ecchymosis over the mastoid (i.e., Battle's sign), raccoon eyes, cerebrospinal fluid rhinorrhea) warrants an evaluation by both an Otolaryngologist and a Neurosurgeon. Additionally, patients with significant vertigo should be evaluated by an Otolaryngologist.

Follow-up Care
Athletes should follow up with an Otolaryngologist approximately 2 weeks after the initial injury to ensure that the tympanic membrane is healing and no other sequelae have developed.

Return to Sports
Barring a concomitant injury (e.g., a temporal bone fracture or otic nerve injury), athletes can return to sport once their pain is adequately controlled. In water-based sports, care should be taken to protect the injury as it heals (e.g. ear plugs). If there is question regarding a return to sport, one should seek an Otolaryngology evaluation to help guide this decision.

Complications
Tympanic membrane ruptures can result in persistent defects, which may need to be patched (e.g., paper patch, fat plug, or surgical tympanoplasty). Otherwise, as the membrane heals patients may develop a cholesteatoma, which is why the involvement of an Otolaryngologist in follow-up is advisable.

Pediatric Considerations
Since otitis media is more common in younger age groups, a team physician should give additional consideration to this condition as a contributing factor in the pediatric athlete. If suspicion for otitis media is present, oral antibiotics may also be appropriate.

Nasal Trauma

Key Points
- Nasal trauma can result in significant injuries or compromise to the airway of an athlete; a sideline physician must be prepared to maintain a patient's airway first and foremost (Fig. 44.30).
- Proper treatment of a nasal fracture may also necessitate the management of epistaxis and/or a septal hematoma.
- The majority of epistaxis originates anteriorly from Kiesselbach's plexus; targeting interventions at this region often yields the highest rate of success.

Fig. 44.30 Nasal trauma is a common occurrence in sport

Introduction

Anatomically, the nose is comprised of the nasal process of the frontal bone along with the frontal process of the maxilla, ethmoid, vomer, palatine, and nasal bones (Fig. 44.31). The blood supply within the nose is worth noting based on the location of the vessels. The anterior and posterior ethmoidal arteries, a terminal branch of the sphenopalatine artery, a portion of the greater palatine artery, and the septal branch of the superior labial artery converge to form Kiesselbach's plexus along the anteroinferior septum. Meanwhile, the posterior aspect of the nasal septum is primarily supplied by the sphenopalatine artery. Finally, the nose also consists of several cartilaginous components, including the nasal septum and the lateral cartilage (the lower end of which supports the tip of the nose).

Nasal Fractures

As the most prominent facial structure, the nose is the most commonly fractured structure in the adult face and the third most fractured bone overall [65]. The majority of these fractures occur in the lower half of the nasal region since these bones are thinner and broader. Given the frequency of these fractures, athletes may have sustained previous fractures, making it more difficult to discern if a new fracture is present and/or the extent of a new fracture.

Mechanism of Injury in Sports

Participation in virtually any sport carries with it a risk of a nasal fracture. Studies have suggested that the incidence of such injuries is actually higher in limited contact team sports (e.g., baseball and softball). One report revealed that only 13.2% of nasal fractures occurred in football. There is also a higher rate of such fractures in organized competition as opposed to recreational play [66].

Epidemiology

Young males are the most likely to suffer nasal fractures, with the peak incidence occurring in the 20s to 30s. While these fractures are rare in children younger than 5 years old, they do increase with age. This has been attributed to increasing participation in competitive and high-speed sports [67]. One study noted that while patients 17 years of age or younger accounted for 42% of all nasal fractures, this same age demographic accounted for 61% of sports-related nasal fractures [68].

Fracture Classification

Strictly speaking, there is no specific nasal fracture classification system. However, it is worth noting the type of fracture patterns associated with the velocity and direction of injury. Low-velocity injuries (e.g., an elbow to the nose) generally result in a simple fracture pattern. However, injuries at high velocities, such as from a hockey puck, are more likely to result in complex or comminuted fracture patterns and other concomitant injuries (e.g., intracranial and/or cervical spine pathology). Blows to the lateral aspect of the nose tend to be the most common, and they can result in displaced fractures. Trauma to the anterior nose may lead to nasal obstruction. Lastly, an injury originating along the inferior nose may cause septal cartilage and/or nasal tip damage.

Clinical Presentation

The best time to evaluate a nasal fracture is within the first few hours of the injury prior to the onset of edema. Regardless, after such an injury athletes may report some degree of nasal airway obstruction. Visual deformities are almost always apparent (Fig. 44.32). This may include periorbital swelling and/or ecchymosis. Patients may have associated epistaxis and/or a septal hematoma. These conditions should be addressed first and are discussed later in this chapter.

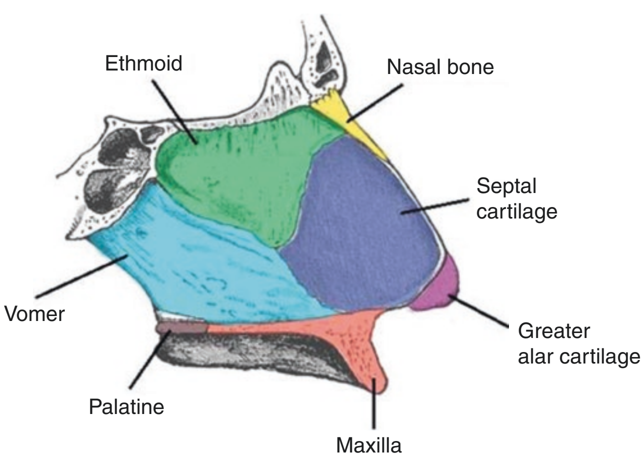

Fig. 44.31 The bony anatomy of the nose (Image courtesy of Oliver Jones; The TeachMeSeries)

Fig. 44.32 A nasal bone fracture in a rugby player

There are several noteworthy physical examination pearls for the sideline physician to consider. Be sure to evaluate both nasal passages, paying particularly close attention for bulging septal hematomas and/or septal dislocations. A lighted nasal speculum can be very useful for this evaluation. Palpation of all of the nasal bones for deformity and/or crepitance is a must. The team physician should also be certain to palpate the remainder of the face, including the dentition, to assess for concomitant injuries. Finally, in the presence of clear drainage from the nose, one should ensure that this is not cerebrospinal fluid. The athlete may report a postnasal drip or the taste of something sweet in the mouth. A ring or halo test can prove useful in this regard by taking a sample of the fluid and placing it on filter paper or even a pillow case. If the fluid separates leaving blood at the center and clear fluid surrounding it in a halo pattern, one should be concerned for a cerebrospinal fluid leak (Fig. 44.33).

Diagnosis

Routine imaging, including plain radiography, is usually not necessary to make the diagnosis of a nasal bone fracture (Fig. 44.34). This has been investigated in the Emergency Department, where such imaging did not significantly impact either the diagnosis or management of nasal trauma [69]. If imaging is pursued, ultrasound is a reasonable option as well, having been shown to have an accuracy rate of 100% (compared to 92% for CT) [70]. CT scans should still be considered if there is concern for complex nasal fractures and/or other associated facial fractures.

Initial Management

In the setting of an acute nasal fracture, the sideline physician must first address a septal hematoma and/or epistaxis, if present. The management of those entities is discussed in other sections of this chapter. Attention can then be turned to the fracture(s). The goal of the intervention is to establish an acceptable appearance to the premorbid location, to achieve bilateral nasal airway patency, and to prevent intranasal stenosis or septal perforation [71]. This can be done immediately on the sideline or in the training room if significant edema has not developed. However, it is acceptable to attempt a reduction 3–5 days after the injury once the athlete's edema has improved or resolved.

Prior to embarking upon a closed reduction, it is wise to advise the athlete that the procedure can be aborted at any time in lieu of a re-examination in several days [67]. Patients should also be apprised of the potential outcomes of the reduction. One study reports that even after an immediate closed reduction, the incidence of nasal deformities requiring subsequent surgical intervention ranges from 14% to 50% [72]. This study is juxtaposed by a report that 91% of patients were satisfied with their closed nasal fracture reduction at 3 months, 87% at 3 years, and only 3% proceeded to

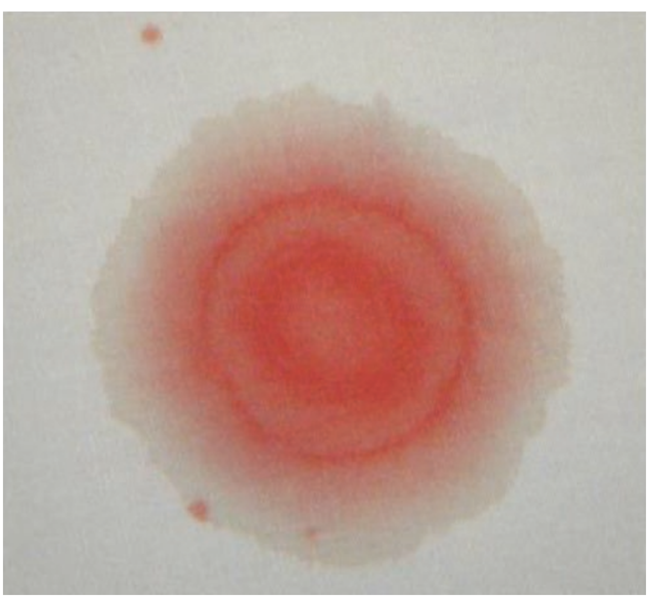

Fig. 44.33 Halo sign concerning for a cerebrospinal fluid leak due to a cribriform plate fracture (Image courtesy of Vishwas Kulkarni)

Fig. 44.34 Plain radiography illustrating a nasal fracture in a collegiate wrestler (Image courtesy of Morteza Khodaee, MD, MPH)

a surgical intervention. Overall, long-term patient satisfaction with this procedure is thought to be between 70% and 90% [67]. Thus, while there is still a risk of a future surgical intervention, a closed reduction is an appropriate procedure to perform.

If pursued, closed reduction of a nasal fracture should be preceded with the application of anesthetic. There are various methods for providing this pain relief, including topical cocaine (unlikely to be available on the sideline for most providers), 1% lidocaine with epinephrine, a hematoma block with 1% lidocaine with epinephrine, and/or bilateral infraorbital nerve blocks. Once adequate anesthesia has been achieved, the provider should place the palmar surface of his or her thumb against the lateralized segment of the nasal bone with the remaining fingers extending past the zygomatic process. Broad and gentle pressure is then applied to the bony step-off until movement of the nasal bone is achieved into the desired position (Fig. 44.35). While an audible or palpable click may be appreciated, this is not always the case. It should be noted that this approach may fail if the fragment segments overlap or the necessary reduction requires lateralization of a fracture fragment [67].

Regardless of whether a reduction was attempted, successful, or unsuccessful, patients should be advised to keep the head and nose elevated as much as possible to reduce edema in the nasal region. Regular icing may also help in this regard. Nasal decongestants should also be recommended. Prophylactic antibiotics are also advised, with amoxicillin-clavulanate appropriate for a duration of 7 days. However, this recommendation is based more on expert opinion rather than quality studies assessing their efficacy or benefit in this context.

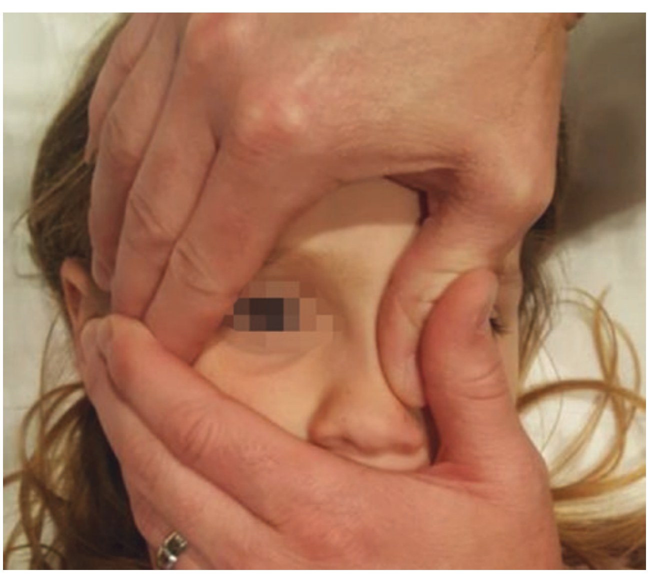

Fig. 44.35 Technique for a closed reduction of a nasal bone fracture

Indications for Referral

There are several indications for immediate referral in athletes who have sustained nasal fractures. First, if there is a delay in presentation greater than 2 weeks, fibrous connective tissue may have developed, making a closed reduction very difficult [73]. Any evidence of concomitant facial fractures warrants a referral as well, particularly those involving the orbit. If the team physician encounters a septal hematoma that cannot be treated in the training room, an inability to achieve hemostasis, and/or an athlete with difficulty breathing, prompt evaluation in an Emergency Department should be sought. Additionally, if there is concern for a cerebrospinal fluid leak, an immediate referral to an Otolaryngologist and/or an Emergency Department is indicated.

Follow-up Care

Athletes with nasal fractures should be evaluated by an Otolaryngologist between 3 and 7 days of the initial injury. Sufficient time should have elapsed to allow for a significant amount of edema resolution. The Otolaryngologist will be able to assess the efficacy of the initial closed reduction attempt (if performed), entertain a closed reduction attempt at that time, or discuss the possibility of an open reduction if deemed necessary.

Return to Sports

Athletes with uncomplicated fractures may be able to return to sport approximately 2 weeks following the injury if they are engaged in noncontact sports. Typically, these fractures heal within 3 weeks in adults, at which time a full return to sport can be considered [74]. If a surgical intervention is required, a decision on a timeline for a return should be made in conjunction with the treating Otolaryngologist. Regardless, it should be strongly encouraged that the athlete utilizes adequate facial protection for several weeks upon resuming participation (Fig. 44.4).

Complications

Persistent cosmetic deformities can result from nasal fractures, which can be mitigated with proper follow-up. One such deformity is referred to as saddle nose, which is typically due to a loss of nasal height when the nasal bridge collapses. Other potential complications include septal deviation, compromised nasal passage patency, and the subsequent development of a septal hematoma. Another particularly important potential complication is meningitis. If an athlete has an associated fracture of the cribriform plate, he or she is at risk for such an infection. Thus, it is imperative that the sideline physician carefully evaluate for evidence of a cerebrospinal fluid leak.

Pediatric Considerations

In general, the pediatric nose is more malleable and less prone to comminution given its high cartilaginous content. With that

said, edema can develop very quickly in children. This confluence of factors can make the diagnosis of nasal fractures more difficult in this demographic. Regardless, the indications for a closed reduction of a nasal fracture in children are essentially no different than those in the adult population [75].

Septal Hematoma

A septal hematoma is a collection of blood in the space between the septal cartilage and the perichondrium (Fig. 44.36). The net result is a separation of the cartilage from its source of nutrients (i.e., the perichondrium). These injuries typically occur with a blow to the cartilaginous inferior portion of the nose. They can occur both unilaterally and bilaterally. If the condition is not identified and treated, avascular necrosis can occur in as little as 3 days [76].

Mechanism of Injury in Sports

Septal hematomas typically result from a direct blow to the nose (e.g., facial contact with the mat in wrestling, being struck in the face by a ground ball in softball, a field hockey stick to the face).

Epidemiology

The overall incidence of septal hematomas is rare, although the exact incidence is unknown. Given the potential devastating consequences of missing this condition, it should be considered in all athletes who sustain nasal trauma.

Clinical Presentation

Aside from noting recent direct trauma to the nose, athletes with a septal hematoma may report significant pain (potentially also due to a concomitant fracture), difficulty breathing through the nose, and swelling. Epistaxis may be experienced by some athletes as well.

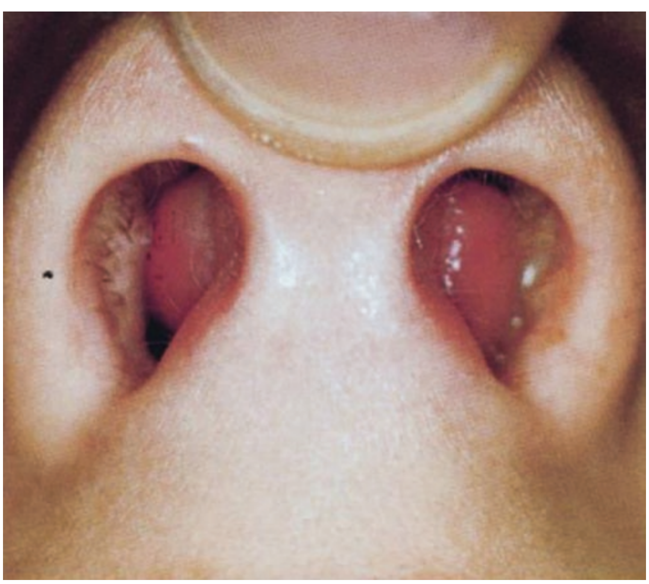

Fig. 44.36 Bilateral nasal septal hematomas (Image courtesy of Handler and Myer [116])

On physical examination, an external nasal deformity may or may not be present. Once it is clear that the patient's airway is protected, be certain to palpate the nasal and facial bones to assess for fractures. Assuming none are identified, the athlete should then evacuate the nasal passages (i.e., blow his/her nose). This helps to clear the field of blood, including clotted blood that may masquerade as a septal hematoma. Visual inspection (with a lighted speculum, if available) will reveal asymmetric bulging of the nasal mucosal fold. Widening of the septum can be an early indication of a developing septal hematoma. The classic appearance of a septal hematoma is a bluish-red mass, although fresh injuries may not possess such discoloration. Tenderness and fluctuance of this mass are often present. In the event that the injury is more proximal, a cotton-tipped applicator can be used to assess for tenderness and/or fluctuance.

Diagnosis

No laboratory testing and/or imaging is necessary to arrive at this diagnosis; physical examination is generally sufficient.

Initial Management

A team physician should be prepared to treat a septal hematoma, whether it be in the training room or the office. Once identified, a nasal decongestant (e.g., 0.05% oxymetazoline) should be instilled into the nasal passages. Local anesthetic can then be administered; 1% lidocaine with 1:100,000 epinephrine is an appropriate choice for both anesthesia and vasoconstriction[2] [67]. Aspiration with an 18- or 22-gauge needle is recommended first to confirm the presence of a blood and thus a septal hematoma. This should be attempted in the most fluctuant aspect. Once verified, a 15-blade scalpel can be utilized to make an incision in the most fluctuant portion of the hematoma, taking care not to penetrating too deep and damage the underlying cartilage (Fig. 44.37). A hemostat can then be utilized to open the incision slightly, if needed. Copious irrigation with saline is advised; an 18-gauge angiocatheter can prove to be valuable for this aspect of the procedure. Some advocate for the placement of a wick or drain, but this is generally unnecessary unless there is suspicion for a septal abscess [77]. In the context of bilateral septal hematomas, the incisions should be offset to avoid the potential of a through-and-through perforation. Subsequent anterior packing of the bilateral passages is advised for 2–4 days [78].

[2] As previously stated, there have been no reports of vasoconstrictor-induced tissue necrosis attributed to lidocaine with epinephrine since 1948. We contend that this is a safe medication in the nose (among other anatomic sites), which is also supported by Marston et al. [67].

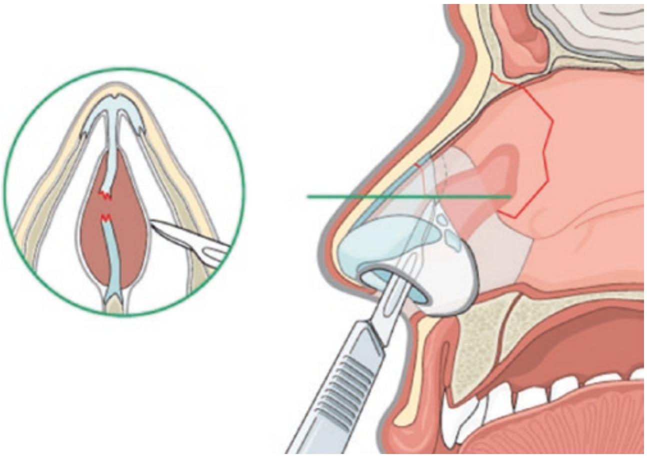

Fig. 44.37 The approach for incising a septal hematoma (Image courtesy of AO Foundation)

Following the successful drainage of a septal hematoma, antibiotic prophylaxis is recommended, particularly with the placement of anterior nasal packing [67]. A 7-day course of amoxicillin-clavulanate or clindamycin is acceptable. It is important to counsel the athlete that reaccumulation of the hematoma can occur. Thus, increasing pain, swelling, and/or difficulty breathing should prompt an immediate re-evaluation.

Indications for Referral

The initial indications for referral include an inability to control any associated bleeding, significant coexisting facial fractures, and/or difficulty breathing. The recurrence of a septal hematoma also warrants a prompt referral to an Otolaryngologist.

Follow-up Care

All patients with a septal hematoma should be evaluated by an Otolaryngologist in approximately 3 days. This appointment is beneficial to assess for a potential failed drainage attempt, reaccumulation of the hematoma, development of an abscess, presence of another infection, and/or evidence of another complication.

Return to Sports

In the absence of other associated injuries (or complications), the athlete can return to play once the nasal packing is removed and there are no difficulties with nasal breathing. Upon resuming participation, it should be strongly encouraged that the athlete utilizes adequate facial protection for several weeks (Fig. 44.4).

Complications

Untreated or suboptimally treated septal hematomas can lead to a myriad of potentially devastating complications. These conditions and their associated clinical findings are presented in Table 44.9.

Table 44.9 Potential complications of septal hematomas

Complication	Clinical Presentation
Septal abscess	Tenderness at the nasal tip; pain out of proportion to exam
Septal necrosis	Tenderness at the nasal tip; pain out of proportion to exam
Cavernous sinus thrombosis	Lethargy; focal neurologic abnormalities (e.g., a lateral gaze palsy)
Sinusitis	Facial pain; nasal obstruction; purulent drainage
Saddle deformity	Visual loss of nasal bridge height (Fig. 44.38)
Hematoma reaccumulation	Increased pain and pressure within the nose

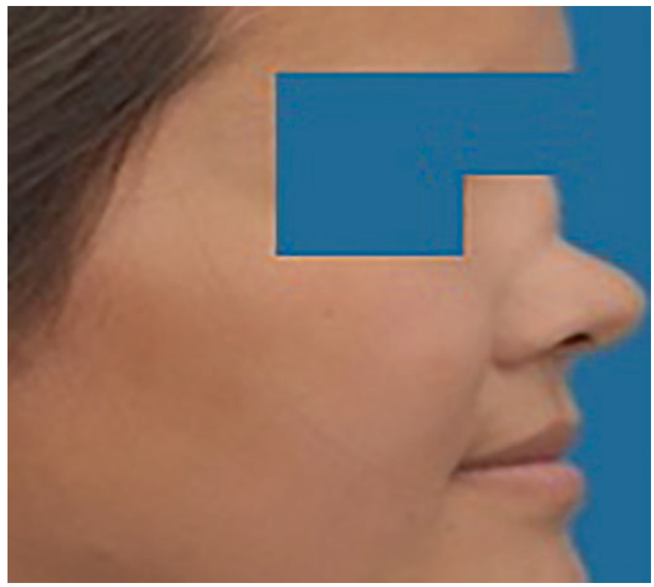

Fig. 44.38 A saddle nose deformity featuring decreased height of the nasal bridge

Pediatric Considerations

The nasal septum in pediatric patients serves as a growth center for the nose. Failure to recognize and treat septal hematomas properly can restrict further nasal development. Similar to nasal fracture treatment, the indications for the treatment of pediatric septal hematomas mirror the indications for adults. Children should be followed for 12–18 months following septal hematoma treatment to ensure that there are no significant cartilaginous changes and/or cosmetic defects [78].

Epistaxis

With a rich mucosal blood supply, the nose is primed to bleed. Common causes include digital exploration, low humidity, foreign bodies, genetic conditions (e.g., hereditary hemorrhagic telangiectasia), and of course trauma.

Epistaxis can be severe, prolonged, and difficult to control. Nasal bleeding is typically described as either anterior or posterior. Kiesselbach's plexus, a confluence of vessels in the anterior septum, is the origin of 90% of all epistaxis cases [79]. Posterior epistaxis stems from the posterolateral branches of the sphenopalatine artery primarily, but the carotid artery can be the source as well. Such bleeds can become serious enough to warrant an inpatient admission.

Mechanism of Injury in Sports

Epistaxis can occur in virtually any sport. As suggested previously, the environmental conditions may be sufficient to result in bleeding from the nares. Additionally, the prospect of a fall onto the nose, contact with an opponent or teammate, or direct trauma with a ball, stick, bat, or other projectile can result in nasal bleeding that necessitates a medical intervention.

Epidemiology

Epistaxis is a very common problem, with estimates suggesting that it affects up to 60% of the population [80]. However, only 10% of the population will present for a medical evaluation. The true incidence of this entity is unknown since many individuals do not seek medical attention. Similarly, there is minimal data on the incidence of epistaxis in athletes. In the population at large, epistaxis is most common in those under the age of 10 and over the age of 50.

Clinical Presentation

Athletes with epistaxis will report blood from one or both nares. Blood down the posterior oropharynx may be reported as well, particularly in the setting of posterior epistaxis. This may result in nausea. In traumatic scenarios patients may note significant swelling and/or ecchymosis. The epistaxis itself is typically not painful; however, pain may result from a concomitant septal hematoma, nasal fracture, and/or other facial fracture. Additionally, a sideline physician should inquire about recent medication use such as aspirin or other NSAIDs.

Before direct nasal inspection, we recommend an initial attempt at tamponade. The athlete should grasp the nasal septum just below the nasal bridge with the thumb and index finger (Fig. 44.39). Enough pressure should be applied to blanch the finger tips, and the pressure should be maintained consecutively for 10–15 minutes. It may be worthwhile to have the patient lean forward slightly as well to minimize the swallowing of blood. Other tips of the trade include the application of a cold compress to the nasal bridge to aid in achieving hemostasis.

If the epistaxis persists after this intervention, ensure that all clots are evacuated from the nose. This can be accomplished with suction or gentle blowing of the nose. Direct visual inspection of the nasal passages should then commence and is crucial in identifying and addressing the source of bleeding. Properly positioning the patient in an upright, sniffing position will help to facilitate an optimal view. The use of a lighted nasal speculum (if available) and application of a vasoconstrictor such as oxymetazoline can be beneficial as well. In fact, in one study showed that the use of oxymetazoline resolved epistaxis in 65% of patients in the Emergency Department [81]. The team physician should evaluate Kiesselbach's plexus first, looking for bleeding, ulceration, and/or erosion. If no source of bleeding is appreciated, one should be concerned for a posterior source.

Fig. 44.39 Positioning for anterior epistaxis tamponade

Diagnosis

Epistaxis is a clinical diagnosis; imaging in the setting of epistaxis is not typically needed. Even in the setting of a traumatic event, only suspected complex nasal fractures warrant further advanced imaging (e.g., a maxillofacial CT). Depending upon the severity of the bleeding, patients may warrant laboratory testing to assess for anemia and/or coagulation abnormalities. However, these tests are generally to assess for the severity of the epistaxis or a contributing etiology rather than making the diagnosis itself.

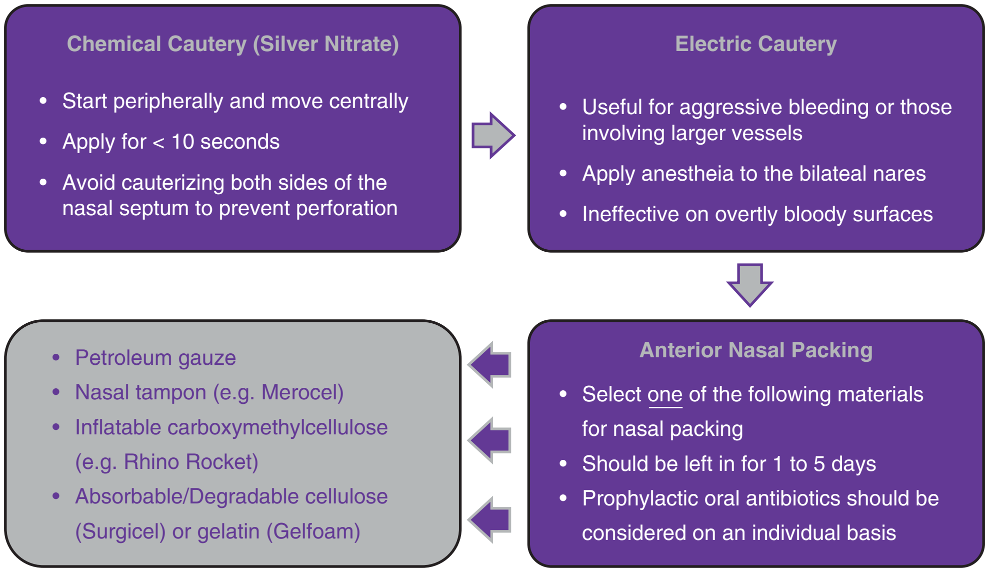

Fig. 44.40 An algorithm for managing anterior epistaxis

Initial Management

The first steps in managing epistaxis as described above serve as a precursor to the visual inspection of the nares. If the source of bleeding is directly visualized and the aforementioned interventions are not successful, one should proceed in a stepwise fashion to control the bleeding. Figure 44.40 provides an outline for treatment moving forward.

There are a few nuances to nasal packing that are worth noting. The application of petroleum gauze can be technically difficult. However, when similar gauze (in one study bismuth subnitrate and iodoform paste-impregnated gauze, which is akin to Xeroform) was compared to nasal tampons, there appeared to be no difference in the efficacy of controlling epistaxis [82]. If a nasal tampon is used, it should be coated in a topical antibiotic, such as bacitracin. When using an inflatable balloon-style catheter (e.g., a Rhino Rocket), soak the catheter in sterile water; do not use saline, apply lubricants, or utilize topical antibiotics. These substances may impair the cellulose coating on the balloon. While studies on this type of balloon catheter have not shown them to be superior to nasal packing, they do appear to insert easier and are less uncomfortable for patients [83]. The use of systemic antibiotics following anterior nasal packing is a topic of debate. While it may be common practice for Otolaryngologists to do so, there is not sufficient evidence to suggest that it is necessary, particularly to prevent toxic shock syndrome. This decision should be made on an individual basis and depends upon the patient's perceived risk of infection (e.g., diabetes mellitus). If prescribed, amoxicillin-clavulanate or a second-generation cephalosporin are appropriate choices for as long as the packing remains in place.

If the epistaxis cannot be controlled with the aforementioned measures, the patient may require a more aggressive intervention (e.g., embolization or surgical ligation). Conversely, the bleeding may be posterior, in which case other interventions should be entertained. There are commercial available products that can aid in this endeavor (e.g., Epistat and Brighton balloon). However, these may not be readily available to the team physician given the relative rarity of their utilization. More commonly available is a Foley catheter, which can be utilized to tamponade a posterior nasal bleed. Once the nasal passage is anesthetized (topical lidocaine is adequate), a 10–14-French Foley catheter should be coated with a petroleum-free lubricant (to avoid balloon degradation). The catheter can then be advanced along the floor of the nose until it is seen in the posterior oropharynx. The balloon should then be filled with 5–7 mL of sterile water and retracted gently until it lodges in the posterior oropharynx. At that juncture the balloon should be completely filled with an additional 5 mL of sterile water. The tube is then clamped in place (using something such as an umbilical clamp, for instance) with sufficient surrounding padding at the anterior tip of the nose to prevent pressure necrosis (Fig. 44.41). Many clinicians will also pack the anterior nasal passage at this point as well to avoid blood pooling anteriorly. Other options for posterior packing exist (e.g., cotton packing), although they are more complex and carry additional significant risks. Unlike its anterior counterpart, antibiotics are typically prescribed for posterior nasal packing. Given that these patients should be evaluated by an Otolaryngologist and/or Emergency Department provider, initiation of the antibiotic regimen can be reserved until that time.

Indications for Referral

The indications for an emergent referral to an Otolaryngologist or an Emergency Department include anterior epistaxis that

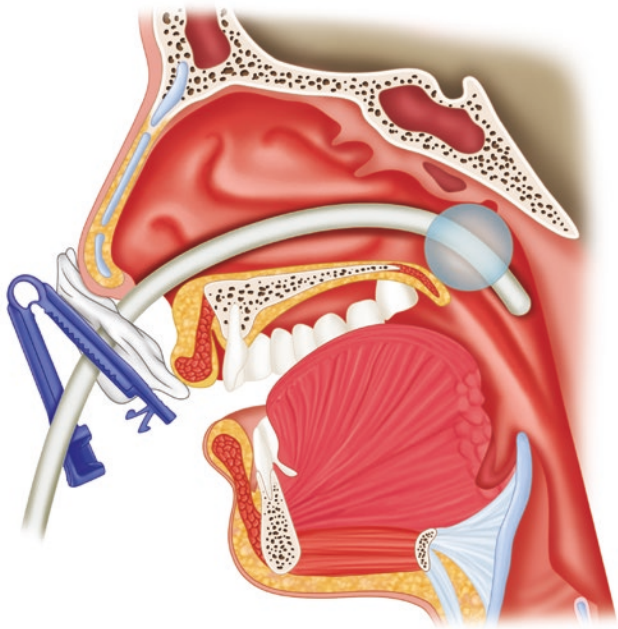

Fig. 44.41 Proper Foley catheter placement for posterior epistaxis

cannot be controlled and posterior epistaxis (controlled or otherwise, as even if controlled it may only be a temporizing measure prior to a surgical intervention). Athletes with atraumatic recurrent epistaxis also warrant a nonemergent referral to assess for a possible underlying etiology (e.g., tumor).

Follow-up Care

If the epistaxis is controlled with digital tamponade or cautery, follow-up with the team physician in 1–2 days is advisable to ensure that there is no persistent bleeding, infection, and/or other associated complications. If anterior packing is utilized, follow-up with an Otolaryngologist is reasonable within 24–48 hours. Athletes who are treated with posterior nasal packing will require emergent evaluation by an Otolaryngologist, who should assist in guiding the athlete's follow-up timeline depending upon the extent of intervention and/or the presence of complications.

Return to Sports

In the case of isolated anterior epistaxis, once the athlete's bleeding is controlled, he or she may be cleared for return to sport. If packing is left in place, the athlete must be able to comfortably breathe and perform without restrictions and/or limitations. Patients requiring packing for posterior epistaxis should not be cleared for participation until the packing is removed and the cessation of bleeding is verified.

Complications

Recurrent epistaxis is a complication that can be somewhat mitigated by avoiding blowing one's nose, refraining from digitally exploring the nares, and minimizing heavy lifting. Nasal saline washes and water-soluble ointments can assist in promoting healing of the nasal mucosa. When nasal packing is required, the overall complication rate is said to be approximately 3% [84]. Toxic shock syndrome is a reported complication, and its risk is greatest with retention of the nasal packing for more than 72 hours. Other potential complications include septal hematomas, abscesses, pressure necrosis, sinusitis, and/or acute airway obstruction due to dislodging of the nasal packing.

Pediatric Considerations

Chemical and electric cautery are still appropriate means for controlling epistaxis in younger patients if pressure tamponade is unsuccessful. Both work well if the patient remains calm. In this demographic, if nasal packing is needed (anterior or posterior), the threshold for consulting an Otolaryngologist is lower. Additionally, if needed, nasal balloon catheters are suitable for use in pediatrics athletes, although they may not be as well tolerated in this age group.

Dental Trauma

Key Points

- There is a high rate of associated injuries in the context of dental trauma; team physicians should be cognizant of other entities such as concussions, mandible fractures, temporomandibular joint injuries, and/or lacerations (particularly of the lip) (Fig. 44.42).
- The Ellis classification can aid a sideline physician in assessing an athlete's need for immediate dental intervention and appropriateness for return to play.
- Tooth fracture fragments or avulsed teeth that cannot be successfully replanted should be placed in a preservation liquid such as a balanced salt solution, milk, contact solution, or sterile saline.
- Dental fractures that do not involve the pulp can be evaluated by a Dentist or Oral Surgeon in 24–48 hours. More significant fractures necessitate an immediate referral for definitive treatment.
- Mouthguards are an extremely effective means for reducing the incidence of dental trauma and should be encouraged for athletes in high-risk sports where it is not mandatory (e.g., basketball and soccer).

Introduction

Sport-related activities find themselves among the most common causes of dental trauma. It has been estimated that an athlete has a 10% chance of suffering such an injury each

Fig. 44.42 Direct trauma to the face, especially by high-velocity projectiles, can result in significant dental injuries

season and between a 33% and 56% chance of encountering dental trauma during his or her career [85].

From an anatomical perspective, the tooth can be divided into the root and the crown, with the former below the gingival line and the latter visible above it (Fig. 44.43). The layers of the crown (from external to internal) contain the enamel (white, or at least it is supposed to be) and dentin (yellow). These layers cannot regenerate themselves [86]. The nerves of the tooth reside in the next layer, which is the pulp. As for the root, it rests in alveolar bone and is attached by the periodontal ligament. Preserving this ligament is critical to successfully replanting an avulsed tooth (a topic that is discussed later) [87].

Mechanism of Injury in Sports

Direct blows to the teeth are the primary cause of dental trauma in sport. This can occur in virtually any sport, ranging from mechanisms such as falls onto the face to contact with projectiles (e.g., sticks, bats, balls, or pucks) to encounters with the anatomy of another participant. It has been reported that such injuries occur more frequently in organized sports, but this could be a function of underreporting in nonorga-

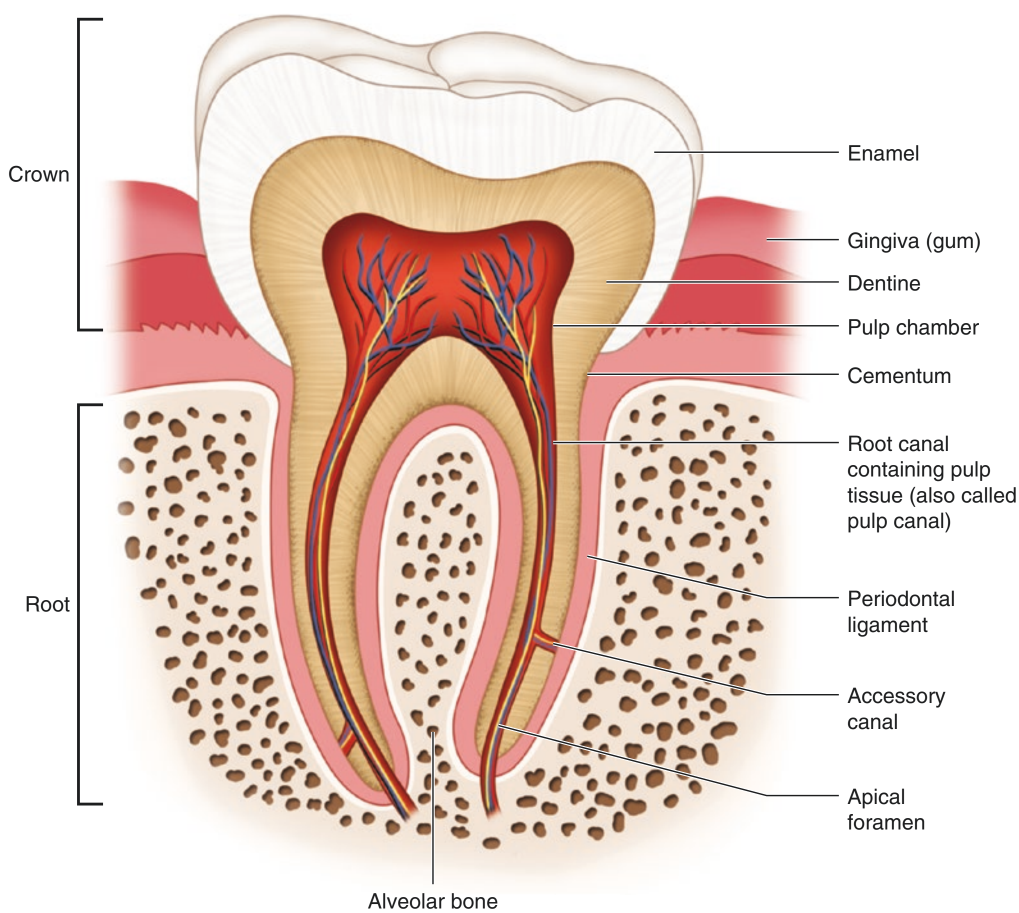

Fig. 44.43 The anatomy of a tooth

nized sporting endeavors [8]. Outdoor sports that occur in warmer times of the year show a higher incidence of such injuries as well [88]. With regard to specific sports, the use (and mandatory implementation in some sports) of mouthguards has significantly affected the context in which dental trauma transpires. For instance, at one point the prevalence of dental injuries in football and rugby was 54% [89]. However, it is now reported that basketball and soccer players are at significantly higher risk for such injuries compared to football players [90]. Mouthguards (ranging from stock mouthguards to "boil and bite" mouthguards all the way to custom-fit mouthguards from a dental professional) have been extremely beneficial in this regard, preventing more than 200,000 dental injuries a year [29]. The National Federation of State High School Associations (NFHS) mandates that mouthguards be used in football, field hockey, ice hockey, lacrosse, and wrestling (for wrestlers with braces) [91]. The American Dental Association (ADA) has recommended the use of mouthguards in numerous (or even most) sports, both in practice and competition, as outlined in Table 44.10 [92].

Epidemiology

Males are typically more impacted by dental trauma than females by a rate of approximately three to one, although this could be skewed based on relative rates of exposure. Overall, 25% of the population between 6 years old and 50 years old have reported dental trauma, with the rate even slightly higher in children [8]. Studies have cited that 31% of such trauma is secondary to sports injuries [93]. Anatomically based epidemiological facts worth noting include that most dental injuries involve a single tooth (with 80% occurring in one of the four maxillary incisor teeth); the left side is more commonly affected than the right side; and there is a high incidence of recurrence [94].

Dental Fractures

Dental fractures are a relatively common occurrence in sport. However, not all such injuries require emergent intervention, and the athlete can return to participation immediately in certain cases. An astute sideline physician will always consider the location of the fractured tooth fragment, as it can be embedded in the surrounding soft tissue (e.g., the lip), aspirated, or even lodged within another participant.

Table 44.10 ADA recommendations for sports in which mouthguards should be used [92]

Acrobatics	Basketball	Boxing
Field hockey	Football	Gymnastics
Handball	Ice hockey	Lacrosse
Martial arts	Racquetball	Roller hockey
Rugby	Shot putting	Skateboarding
Skiing	Skydiving	Soccer
Squash	Surfing	Volleyball
Water polo	Weightlifting	Wrestling

Fracture Classification

The Ellis classification system is utilized to describe the severity of dental fractures and helps guide their management (Fig. 44.44 and Table 44.11, with the relative frequency (%) of dental fractures noted in parentheses)[3] [8].

Clinical Presentation

The majority of athletes will report a traumatic event involving the head, neck, and/or face. It can be useful to inquire about the direction of force associated with the injury, as it may aid the sideline physician in locating missing tooth fragments and/or identifying other associated injuries. Aside from the historical pearl of trauma to the face, athletes will also typically note pain (although Ellis I fractures may go largely unnoticed initially). Sometimes the associated pain may be appreciated after the consumption of a hot or cold liquid or with mastication. Bleeding may be present, raising the suspicion for a concomitant oral laceration (e.g., gingiva, lip, tongue, etc.). Similarly, the athlete may have pain elsewhere in the face, including the ear, temporomandibular joint, and/or neck. The savvy team physician should also assess for the presence of concussion-related symptoms.

Physical Examination

As with all facial trauma, the sideline physician should first ensure that the athlete is protecting his or her airway and breathing appropriately. Once these potential issues have been evaluated, verify that the athlete's circulation is intact. Next, the skull, cervical spine, and other facial structures (i.e., eyes, ears, and nose) should be inspected. Prior to proceeding to the dental examination, one should perform an extraoral assessment. As detailed previously in this chapter, be sure to palpate the mandible, maxilla, zygoma, temporomandibular joint, and mastoid bones. Any identified lacerations should be thoroughly inspected for foreign bodies (e.g., dirt, sand, tooth fragments) and removed, if found.

At that juncture one can proceed to the intraoral evaluation. Visual inspection is paramount, with care taken to first account for any missing teeth (with dental avulsion, or Ellis V injury, management discussed later in this chapter).

[3]Some dental texts have extended the Ellis classification through Ellis IX injuries. This further delineation of dental trauma past an Ellis V is beyond the scope of this chapter, and such injuries should warrant immediate evaluation by a Dentist or Oral Surgeon.

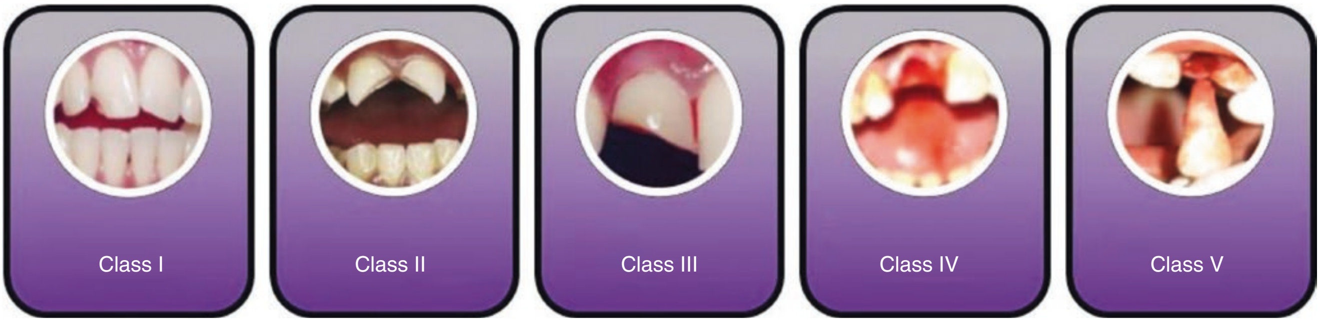

Fig. 44.44 The Ellis classification system for dental fractures

Table 44.11 The Ellis classification system for dental fractures

Ellis Grade	Description of the Injury
I	Enamel only; rough or chipped tooth (15.8%)
II	Enamel and dentin; painful with exposure to the air, cold liquids, and/or palpation (39.9%)
III	Enamel, dentin, and pulp; red-pink pulp will be exposed (25.7%) (Fig. 44.45)
IV	The entire tooth and root; the extent of root involvement impacts outcomes (18.5%)
V	An avulsion of the tooth (Fig. 44.45; discussed later in this chapter)

Fig. 44.45 A dental fracture involving tooth #9 (Ellis III) and a dental avulsion of tooth #8 (Ellis V)

Careful inspection of the affected tooth or teeth provides significant insight into the grading of the dental fracture. Gently wiping the affected tooth may reveal a spot of blood, indicative of an Ellis III injury. Be sure to assess the buccal mucosa, gingival tissue, tongue, and the frenulum for damage. Any lacerations should be probed for foreign bodies as well. Then, palpate the alveolar bone for potential fractures. Having the athlete bite down may result in malocclusion. Lastly, evaluate the mobility of the teeth. Patients with malalignment and/or excessive mobility are more likely to have underlying fractures (alveolar and/or root), and further imaging is warranted. The tongue blade bite test, as discussed previously in this chapter, can aid in the identification of associated fractures as well.

Diagnosis

Some have argued that not all dental fractures are visible on physical examination, and thus any tooth trauma warrants imaging [29]. While a low index of suspicion is certainly prudent, routine referral (emergent or otherwise) for imaging of Ellis I and Ellis II fractures is not necessary. However, there is a role for further imaging when attempting to distinguish between an Ellis III and an Ellis IV injury. Clinically this is relevant because while both are dental emergencies, the nature of the intervention performed by a dental professional may vary. Occlusal plain radiography (intraoral radiographs similar to those secured in a Dentist's office) is the imaging of choice, which will likely necessitate an evaluation by a Dentist or an Oral Surgeon. Such imaging may not be accessible for all team physicians, but some may possess the ability to secure this radiography. Of note, two periapical views at different angles are often required to identify a root fracture [8]. Lateral, anterior views can also prove to be beneficial in this setting. Keep in mind that similar to bony fractures, dental fractures may not be evident initially on imaging. If concern for a root fracture persists, repeat imaging may be sought 1–2 days after the traumatic event.

Otherwise, additional imaging decisions are predicated on the concern for concomitant injuries. For instance, an athlete with a missing tooth segment and dyspnea may warrant a chest radiograph to search for a potentially aspirated fragment. Even when an Ellis III or Ellis IV dental fracture is not suspected, an orthopantomogram may be secured to determine if a tooth fragment is lodged in the surrounding soft tissue. Lastly, in the setting of significant facial trauma with associated dental injuries, an orthopantomogram or maxillofacial CT may be indicated to evaluate for a mandibular fracture, for example.

Initial Management

The initial management of dental fractures depends upon the extent of injury, as detailed below. Regardless of the extent of injury, significant attention should be placed on

Fig. 44.46 Some tooth preservation options for dental fracture fragments and avulsions

locating the fracture fragment(s). Remember that the section of tooth could be expelled, lodged in the athlete's soft tissues or that of an opponent, or even aspirated. If located, the fragment should be placed in a balanced salt solution (e.g., Hank's Balanced Salt Solution), milk, contact solution, or a commercially available tooth preservation system (Fig. 44.46).

- Ellis I: If only the enamel is involved, the athlete can continue to participate in sports if desired and his or her pain is controlled. In the event that the tooth fragment is found, it can be reattached up to 48 hours later.
- Ellis II: This injury does not necessarily require a removal from activity either. If the patient can tolerate the discomfort of the exposed dentin and the edges of the tooth do not present a risk to the athlete or others, continued participation in sport is reasonable [29]. Treatment should be pursued within 1–2 days. Of note, there may be an increased risk of bacterial infection the longer the dentin is exposed.
- Ellis III: Athletes should be removed from sport and promptly evaluated by a Dentist or Oral Surgeon, preferably within 3 hours. No immediate treatment on the sideline or in the office is necessary. Minimal exposure of the pulp may allow for direct capping, although a root canal may be necessary for more extensive injuries.
- Ellis IV: The tooth component should be handled as little as possible and promptly placed in a preservation solution. Gentle pressure to the fracture site can be applied (e.g., with sterile gauze held in place by the athlete biting down lightly) to prevent further movement of the remaining tooth. No further immediate on-site management is advised, and the athlete should be referred for definitive management.
- Ellis V: These injuries amount to dental avulsions; the overall management of this condition is discussed later in the chapter.

Indications for Referral

Ellis I and Ellis II fractures should be evaluated by a Dentist within 48 hours. Ellis III or greater fractures warrant an immediate referral for further dental evaluation and treatment. As indicated above, some dental injuries may require a root canal. In certain more severe cases, dental extraction may be required with subsequent replacement using a dental implant.

Follow-up Care

Virtually all athletes with dental fractures should be advised to follow up with a Dentist or Oral Surgeon. This may amount to nothing more than an initial follow-up visit 24–48 hours after the incident. In other contexts, patients may need a root canal 1–2 weeks after the trauma. On the far end of the spectrum, an Ellis IV fracture involving the root may require up to 2–3 months of immobilization and subsequent follow-up for up to 2 years [8]. Additional management of these injuries includes making sure that the athlete's tetanus status is current. Flossing should be avoided until the injury is fully healed, but general oral hygiene is otherwise essential. A soft diet may be necessary to avoid exacerbating the patient's pain. And after such an injury it is important for the team physician to ensure that the athlete has an adequate mouthguard, which may require a new mold/fitting.

Return to Sports

Athletes can safely be allowed to resume sporting participation as soon as their pain is controlled in the setting of Ellis I and Ellis II fractures. The return to sport after the management of more significant dental fractures is variable and should be made in conjunction with the consulting Dentist or Oral Surgeon. The use of a mouthguard is highly encouraged moving forward to decrease the risk of reinjury.

Complications

Dental fracture complications can occur despite optimal management. These complications range from permanent cosmetic deformities to complete loss of the affected tooth. Bacterial infections can develop when exposure of dentin and/or the root occurs. As such, prophylactic antibiotics (e.g., penicillin VK or clindamycin) are recommended when pulp is exposed [95]. In general, the longer the delay to definitive treatment, the poorer the outcome, whether cosmetic or functional.

Pediatric Considerations

The Ellis classification system can still be utilized in the setting of pediatric dental fractures even though the injury may involve primary teeth rather than permanent teeth. The incidence of certain classes of injuries does differ. For instance, the more severe root fractures are less common in primary teeth [96]. Management of these injuries may differ with care taken to avoid damaging the underlying permanent tooth. For this reason, primary teeth should generally not be replanted. Otherwise, the overall management of these injuries is roughly the same as in adults; however, one should consider consultation with a pediatric dental specialist if a referral is necessary.

Dental Avulsions

Defined as a complete traumatic displacement of a tooth from the alveolar socket, dental avulsions are also referred to as Ellis V fractures. They require prompt identification and treatment by the sideline physician.

Clinical Presentation

Direct trauma to the head and/or face generally precedes this injury. Given the force often required to create these injuries, athletes may have altered consciousness (raising a team physician's concern for an intracranial hemorrhage or concussion), marked facial pain, and/or brisk bleeding from the oropharynx. Otherwise, dental avulsions should be suspected when an athlete presents a seemingly intact tooth to the medical staff or there is an absent tooth on intraoral examination. When the athlete reports such an injury but cannot find the tooth, it is important to locate the potentially avulsed tooth, evaluating the surrounding playing surface, the athlete's soft tissues, and even considering aspiration.

Physical Examination

The physical examination for dental avulsions should commence as was previously described in the "Dental Fractures" section of this chapter. After the integrity of the patient's airway, breathing, and circulation are confirmed, the team physician should assess for damage to the other facial structures and/or facial bones. The intraoral examination is also as noted above. The primary difference will be the finding of a dental socket devoid of tooth contents.

Diagnosis

Identifying a dental avulsion is generally accomplished on a clinical basis alone. However, further advanced imaging (e.g., an orthopantomogram or a maxillofacial CT) may be warranted if a retained tooth fragment, alveolar fracture, and/or other facial fracture is suspected. Also keep in mind that a missing tooth could be found in other anatomic areas, including the nasal cavity (by penetration equating to an intrusion dental luxation, which is discussed later in this chapter) or the chest (via aspiration). If there is concern for such an occurrence, the requisite imaging should be pursued.

Initial Management

If the athlete is noted to have altered mental status or cannot protect his or her airway, one should not attempt to replant the avulsed tooth. In those instances, the tooth should be placed in a preservation solution until it can be safely replaced (Fig. 44.46). Barring such a contraindication, the sideline physician can and should make an effort to return the tooth to its proper location. Studies suggest that the time out of the socket can impact the success of replantation, with 66% of teeth replanted within 1 hour functionally normal and radiographically healed at 5 years [97]. Research has shown that replantation within 5 minutes produces the best results [98]. The tooth should be rinsed in water or saline but not wiped or scrubbed. Try to handle the tooth primarily by the crown to prevent disturbing the root structures. Reinsertion of the tooth is straightforward, but care should be taken to minimize disruption of the socket. Often there is a "click" when the tooth is successfully replaced [99]. Hold the tooth directly in place for 5 minutes. This freshly replaced tooth will require further stabilization (e.g., splinting) by a Dentist or Oral Surgeon. In the event that the tooth cannot be successfully replanted, it should be protected in one of the aforementioned solutions or even between the patient's gingiva and buccal mucosa until securing definitive treatment.

Indications for Referral

All patients with dental avulsions should be immediately referred to either a Dentist's office or an Emergency Department with the requisite capabilities to successfully replant and/or splint such injuries.

Follow-up Care

The vast majority of dental avulsions necessitate splinting, which will require follow-up care (Fig. 44.47). The duration of splinting can be variable with 1–2 weeks being relatively common. Similar to dental fractures, a soft diet may help to decrease the athlete's pain with mastication. Prophylactic antibiotics are often prescribed (e.g. penicillin VK or clindamycin). General hygiene is otherwise highly encouraged to decrease the risk of subsequent infection following a dental avulsion.

Return to Sports

The athlete's return to sport will be contingent upon the recommendations of the Dentist or Oral Surgeon assisting in the provision of care. Participation should not be considered until the patient's splinting is removed and the tooth is deemed stable (typically 2 weeks or longer depending upon the severity of injury). As expected, a well-fit molded mouthguard should be utilized upon the patient's return. One should avoid mouthguards that can be displaced upon impact, which includes the over-the-counter variety [29].

Complications

Delays in replanting dental avulsions can make subsequent attempts to relocate the affected tooth more difficult if coagulation or blood clotting occurs in the socket. The nature of the injury itself can lead to a root canal, loss of the tooth, and/or a surrounding infection.

Pediatric Considerations

When a primary tooth is avulsed, it generally should not be replanted. Doing so may damage the underlying development of the permanent teeth.

Dental Luxations

Blunt trauma to the teeth may not result in fractures or avulsions but rather varying degrees of loosening of the tooth itself. Sometimes referred to as subluxations or luxations, these dental injuries occur in sports, and a well-trained team physician should be able to deftly treat them and recognize when further evaluation with a dental specialist is indicated.

Injury Classification

Typically, these injuries involve the maxillary incisors, although they can involve any tooth. With that said, there are several terms that are utilized to further delineate the traumatic events that fall within the realm of subluxations or luxations [29].

- Concussion: This trauma results in pain to the touch of the affected tooth, but there is no loosening or visual displacement. Root fractures can still occur, however, and should be considered by the astute sideline physician.
- Subluxation: Limited mobility of the tooth is the hallmark of this condition. Visible displacement may not be appreciated, although bleeding from the gingiva can be seen.
- Lateral luxation: This description can be a bit misleading because it encompasses lateral or anterior/posterior displacement of the affected tooth (Fig. 44.48). It is often accompanied by a fracture of the alveolar plate, and the tooth is usually firmly situated in the socket.
- Extrusive luxation: These injuries are apparent by a partial displacement of the injured tooth as it protrudes outward from the tooth socket.
- Intrusive luxation: A tooth that is relocated through the floor of the tooth socket falls into this category. For the most part, an associated fracture of the alveolar socket should be considered. These are the most severe injuries within luxations.

Fig. 44.47 Splinting of a dental avulsion involing tooth #21 with a fracture involving tooth #11 (Image courtesy of Rls, CC BY-SA 3.0. Savas et al. [117])

Fig. 44.48 A lateral luxation of tooth #9 (Image courtesy of Arikan and Sari [118])

Clinical Presentation

Similar to its dental injury cousins (e.g., dental fractures and dental avulsions), luxations are the result of direct trauma to the head and/or face. This force may be directly to the anterior aspect of the teeth or the result of contact with the distal most ends of the teeth. These traumatic events are almost always associated with pain at or near the affected tooth. However, as noted above, the athlete may not report laxity of the tooth. Patients may describe an abnormal bite or an unusual sensation with mastication.

Physical Examination

The physical examination for dental luxations mirrors that for its dental predecessors. After the athlete is stabilized with regard to his or her airway, breathing, and circulation, care should be taken to assess for associated facial fractures. Once those etiologies have been ruled out and/or addressed, a thorough intraoral evaluation should be completed. The presence of tooth laxity is an important feature to note. Athletes may have concomitant dental fractures (e.g., an Ellis III fracture) with a luxation.

Diagnosis

A thorough physical examination is generally all that is required to identify a dental luxation. As with other dental trauma, additional advanced imaging is indicated if there is concern for a facial fracture or concomitant injury. An immediate orthopantomogram or maxillofacial CT is not necessary for all isolated dental luxations (e.g., concussions or subluxations). However, subsequent imaging may be obtained in follow-up to evaluate for a possible root fracture and/or to ensure that the tooth is seated properly in the socket [29]. Athletes with more significant luxations (i.e., lateral, extrusions, or intrusions) may benefit from such advanced imaging, which will likely be at the discretion of the consulting dental specialist. Also remember that intrusion injuries can violate the nasal cavity, which may necessitate imaging.

Initial Management

Once the athlete's airway, breathing, and circulation are confirmed to be intact and other associated potential injuries have been evaluated for and/or treated, the team physician should turn toward managing and stabilizing the dental luxation. It is likely beyond the scope of the sideline physician to splint injured teeth in the training room or office. With that said, not all of these injuries require immediate intervention. For instance, a tooth concussion, likely resulting in pain but no tooth laxity, will not need any emergent dental management. In the setting of a tooth subluxation, there again may be no need for immediate intervention. The sideline physician should assess the patient's degree of tooth laxity. If the tooth is deemed to be at an increased risk for falling out or the sport itself presents a significant risk for such an occurrence or additional injury (e.g., boxing, mixed martial arts, and wrestling), the athlete should be withheld from participation until the tooth is stabilized [29].

Lateral, extrusion, and intrusion luxations should be immediately managed by relocating the tooth to its natural position without exerting excessive force. For extrusions and intrusions, this may require anterior or posterior manipulation in addition to a force aimed at positioning the tooth within its socket. Such interventions can help to preserve the tooth and decrease the risk of subsequent complications, which are discussed below. It should be noted that with lateral luxations, the tooth can be wedged in a fractured alveolar plate. In these cases the tooth may be extremely difficult to manipulate. Local anesthesia (e.g., a local dental block or an inferior alveolar nerve block) may be necessary to facilitate such a reduction. Regardless, the next management step for these athletes is to seek an immediate dental consultation.

Indications for Referral

It is advisable that all dental luxation injuries be evaluated by a Dentist or Oral Surgeon, but the timing of this referral varies. As previously suggested, dental concussions and minor subluxations can be seen within the next 24–48 hours. If the team physician deems a dental subluxation to be at high risk to fall out, a more prompt (i.e., same day) evaluation is appropriate. All lateral, extrusion, and intrusion luxation injuries should be immediately referred to a Dentist or Oral Surgeon. The affected tooth may require prompt splinting (or potentially a surgical intervention) in order to optimize the chances of saving the injured tooth and limit the risk of sequelae.

Follow-up Care

As indicated above, dental luxations should be evaluated by a Dentist or an Oral Surgeon, with continued follow-up at the discretion of the treating subspecialist. The most significant of these injuries may require follow-up for up to 1 year. With that said, there are a couple of other considerations to keep in mind. A soft diet should be recommended for these athletes until they are able to eat without pain. Antibiotics are generally recommended for all lateral, extrusion, and intrusion luxations. Similarly, if there is bleeding from the base of a subluxed tooth, antibiotics are advised. The argument here is that damage to the periodontal ligament can allow for the spread of bacteria and subsequent infection [29]. Penicillin and clindamycin are appropriate selections in this context, and a typical course lasts for approximately 10 days. Additionally, an antibiotic

mouthwash may be beneficial to further prevent the spread of infection.

Return to Sports

Athletes with dental concussions and minor subluxations can return to sport as soon as the associated pain is controlled, if so desired by the athlete. They should return to sport with a mouthguard to further protect the affected tooth (or teeth). More substantial subluxations and lateral, extrusion, and intrusion luxations will require an immediate dental evaluation. Splinting of the injury tooth is likely to ensue, and the return to sport in most cases should not occur until the splinting is removed. In the event of a splinted subluxed tooth, one can consider a return while the splint is in place if a mouthguard is worn. This mouthguard should be molded to fit around the existing splint [29]. Regarding the duration of splinting of subluxations, it may be kept in place for 2–3 weeks. However, if multiple teeth are involved in an extrusion injury, the splinting may persist for up to 6–8 weeks. Regardless, upon clearance for a return to sport (made in conjunction with the dental specialist, team physician, athlete, and parents when appropriate), a new, freshly molded protective mouthguard for the athlete should be utilized.

Complications

There are several noteworthy complications that may arise following luxations, even if they are treated promptly and effectively. These complications include darkened teeth (due to pulp death), dental abscesses, loss of surrounding bone, and/or root resorption [29]. If the dental luxation is missed or not relocated promptly, the tooth can actually fuse with the surrounding bone, making it extremely difficult to relocate. Ensuring prompt follow-up with a dental specialist can help to correct and/or limit these issues.

Pediatric Considerations

The primary consideration for pediatric athletes with dental luxations is to assess whether the permanent teeth are affected. Radiographs will prove to be valuable in this regard. For extrusion injuries (generally with >3 mm of extrusion), the tooth may be extracted altogether [96]. It is imperative that a prompt dental evaluation be pursued to avoid sequelae to the underlying permanent teeth. Minor intrusion injuries and lateral luxations may either re-erupt or reposition themselves on their own, but this should not spare the athlete from the aforementioned dental evaluation [100]. And as an additional aside, pulp death (and thus darkening of the injured tooth) is less common in pediatric patients compared to adults. This is due to the open apex of the tooth, which allows the pulp to revascularize [29].

Facial Lacerations

Key Points

- The primary tenants of facial laceration care include evaluating for concomitant injuries (and treating them first when necessary), irrigating the wound, inspecting for foreign bodies, and performing a skilled primary closure (when indicated).
- The optimal repair of facial lacerations, both in terms of cosmetic outcome and patient comfort, involves the use of adequate anesthesia, including regional nerve blocks when appropriate.
- The most important aspect of a lip laceration repair involves the meticulous reapproximation of the vermillion border.
- It is imperative to note the types of eyelid lacerations that warrant immediate subspecialty referral, including those that are full thickness, involve the tear drainage system, or those with orbital fat prolapse.
- Auricular and nasal lacerations necessitate that each anatomical layer be closed or reapproximated individually.
- Most tongue and intraoral lacerations will heal well and rapidly without any further intervention.

Introduction

Facial lacerations in sports come in numerous shapes, sizes, and complexities, ranging from an apparently simple laceration after being struck by the rogue cleat of an opponent all the way to a gaping, deep injury to the forehead during pugilistic activity (Fig. 44.49). With that said, even seemingly minor injuries should be taken seriously and thoroughly evaluated given the complexity of the underlying facial structures. Additionally, facial injuries carry the heightened emphasis on an optimal cosmetic outcome along with an ideal functional result. In short, a well-prepared team physician should be capable of addressing facial lacerations in their various forms, keenly recognizing when subspecialty assistance is necessary.

Mechanism of Injury in Sports

Lacerations in sport are common and can occur in virtually any sport. Blunt trauma from another participant or direct contact with a projectile may result in such an injury. Also, some rather unique mechanisms of injury can precipitate a facial laceration, such as from the blades of an ice skate, fishhooks, the edges of skis, and even environmental features (e.g., rocks).

Fig. 44.49 A significant forehead laceration in boxer

Epidemiology

The true incidence of facial lacerations in sport is a very difficult figure to accurately identify. Some injuries may be initially deemed "minor" thus avoiding further evaluation and treatment. Consequently, they are unlikely to be reported and captured in an epidemiologic study. Conversely, formal assessments on the topic have produced widely variable results. For instance, in the 2004 Olympics, 14 of the 19 injuries (occurring in team sports only for the purposes of this study) involving the head and neck were lacerations [101]. Meanwhile, another study assessing the injuries of a Swedish hockey team over 3 seasons found a total of 95 injuries, with 29 of the cases (31%) diagnosed as facial lacerations [102]. Overall, facial lacerations appear to be a relatively common occurrence in sport.

Clinical Presentation

The clinical presentation of facial lacerations is typically straightforward. Direct trauma to the face is the norm, and the mechanism of injury is most often penetrating (e.g., from an opponent's fingernail or the blade of an ice hockey skate). With that said, blunt trauma can also result in these lacerations, such as from a mixed martial arts gloved–hand blow to the eyebrow region. Bleeding is frequently present, and the rich vascularity of the facial structures means that the bleeding may be brisk. Pain may or may not be associated with these injuries.

Diagnosis

Lacerations to the face and its structures (e.g., ears, nose, lips, and intraoral surfaces) rarely require further imaging.

Fig. 44.50 Proper irrigation of a facial laceration in a mountain biker (Image courtesy of Morteza Khodaee, MD)

Considerations for imaging include the need to evaluate for retained foreign bodies and/or to assess for concomitant fractures (e.g., mandible fractures). Depending upon the concern, plain radiography, maxillofacial CT scans, or an ultrasound can be utilized, if needed.

Initial Management

One of the first and most important steps in caring for all lacerations entails thoroughly irrigating the injured tissue (Fig. 44.50). Tap water is safe and effective for this purpose if sterile saline is not available. Exploration for foreign bodies should then occur. Additionally, nonviable tissue should be removed in order to ensure proper reapproximation of the wound edges. Consideration should then be given as to whether a repair is necessary at all as opposed to allowing the wound to heal by secondary intention. Of note, it is considered safe and appropriate for facial lacerations to be closed within 24–48 hours with proper cleansing [103]. Other considerations that might steer a provider toward allowing a wound to heal by secondary intention include associated open fractures, heavily contaminated wounds, and those that involve human bites [104]. If the wound is deemed appropriate for a repair by the team physician, he or she should contemplate the optimal type of material to utilize for the repair, whether it be skin adhesive, Steri-Strips, superficial sutures, or deep, absorbable sutures in conjunction with superficial sutures.

There are a couple of other associated issues that also should be addressed in the context of facial lacerations. The team physician should ensure that every athlete's tetanus status is current. The routine use of prophylactic antibiotics is not necessary (aside from bite wounds) and is made on an individual basis. A prolonged time to presentation (>24 hours) is one such indication for the administration of antibiotics.

General post-procedure wound care instructions should highlight daily gentle cleaning of the wound with soap and water. Topical antibiotic ointment (e.g., bacitracin/polymyxin) should be applied one to two times daily. Athletes

should be advised to avoid submerging the affected area in water until the wound edges are healed, typically once the sutures are removed.

Indications for Referral

Overall, if the team physician is not comfortable with the complexity of the facial laceration a referral should be sought immediately. Similarly, if there is concern for a suboptimal cosmetic outcome, an evaluation by a Plastic Surgeon is appropriate. Otherwise, lacerations that cause nerve injury or those that result in bleeding that cannot be controlled warrant prompt evaluation in an Emergency Department with a possible Plastic Surgery consultation to follow.

Follow-up Care

Typically, sutures placed in the facial structures should be removed within 5–7 days. This can be done in the training room or the office barring any complications. It is important to note that scar formation can occur for up to 1 year after the repair (and up to several years in children) [105]. Thus, athletes should be encouraged to keep the lacerations covered during that interval, whether that be with a hat, protective garment, or sunscreen.

Return to Sports

Once the laceration is repaired with any associated bleeding and/or pain controlled, athletes are safe to resume full activity without restrictions.

Pediatric Considerations

Broadly speaking, the repair of facial lacerations in younger patients may require sedation in order to achieve optimal closure and wound reapproximation. If the patient is not able to remain calm and still for the repair with local anesthesia or a regional block of the affected area, a referral to a specialist and/or an Emergency Department may be necessary to facilitate wound closure.

Further discussion of the unique considerations for specific lacerations of facial structures is discussed below. The general tenants of facial laceration care as described above apply unless otherwise noted.

Eyelid Lacerations

Eyelid lacerations can appear visually complex and perhaps even intimidating (Fig. 44.51). The relatively small amount of territory contains several layers of tissue and important associated structures. Technically, the eyelid itself is the skin that overlies the orbicularis muscle. Just beneath this muscular layer is a fibrous sheet of tissue that comprises the orbital septum. Toward the posterior aspect sits orbital fat separating the orbital septum from the levator muscle, which functions to retract the eyelid. Lastly, the conjunctiva coats the inner aspect of the eyelid and makes direct contact with the surface of the eye.

Fig. 44.51 A complex, full-thickness upper eyelid laceration that should not be repaired by a team physician (Image courtesy of EyeRounds.org – University of Iowa)

There are several other important structures around the eyelids that the astute team physician should recall. The lacrimal system is key among them. Along the medial aspect of the eyelid both superiorly and inferiorly rest puncta. Tears pass through these structures in canaliculi, which converge to form a canaliculus that in turn drains into the lacrimal sac. This entity sits just lateral to the nose and empties into the nasolacrimal duct. Recognizing these structures is critical to creating a plan for repairing eyelid lacerations, as damage to any of them warrants a referral to a subspecialist.

Initial Management

Before a team physician entertains an eyelid repair in the training room or office, he or she should thoroughly assess the athlete for other ocular injuries. An open globe injury is chief among these concerns, and if sufficient suspicion is present a referral for immediate evaluation should be pursued. Other entities associated with eyelid lacerations include hyphemas, corneal abrasions, and foreign bodies. Most of these conditions are discussed elsewhere in this chapter. Some of these conditions may also warrant a CT scan of the orbits (e.g., to confirm a ruptured globe or to assess for the presence of a foreign body).

Having addressed these concerns, one can then focus on repairing simple eyelid lacerations. Size matters, and these injuries are no different. Horizontal lacerations along the skin lines do not require primary closure if they involve <25% of the lid [106]. These wounds can be closed with Steri-Strips linearly followed by the placement of triple antibiotic ointment. The use of tissue adhesive is also reasonable, but extreme care must be taken to avoid ocular exposure or inadvertently gluing the eyelids together [107].

Simple lacerations that surpass the 25% threshold should be closed via primary repair. Local anesthesia with lidocaine or bupivacaine is appropriate. The wound should be thoroughly irrigated as discussed earlier in this section. The wound should be closed in layers, if necessary. Deep layers can be reapproximated with 5-0 or 6-0 Vicryl. Non-absorbable sutures are acceptable for the superficial layer, specifically 6-0 or 7-0 nylon or Prolene. Absorbable sutures are also appropriate for this aspect of the repair, and the smaller the suture size the better (e.g., 6-0 fast-absorbing gut). When placing the sutures, apply just enough tension to slightly evert the wound edges.

Indications for Referral

There are several clear indications for a referral to an Ophthalmologist or a Plastic Surgeon, which are provided in Table 44.12.

Follow-up Care

The repaired eyelid laceration should be reassessed in 24–48 hours to evaluate the healing and for the possibility of a developing complication. Otherwise, any non-absorbable sutures that were placed should be removed in 5–7 days.

Complications

Eyelid lacerations present a variety of potential pitfalls. For instance, if the tear drainage system is involved and not properly repaired, the athlete may have permanent tearing. This complication can be avoided (or at least minimized) by probing the canalicular system as performed by an Ophthalmologist, and if damaged stenting may be required. Eyelid laceration repairs with excessive tension can result in scarring and traction on the eyelid, potentially resulting in permanent dry eye.

Pediatric Considerations

It stands to reason that the repair of an eyelid laceration in a pediatric athlete may be difficult. Simply evaluating the extent of the injury (and other potential injuries) may be challenging by itself. Other reasons that these lacerations may present difficulties include the smaller anatomy, the athlete's concern over the impending procedure itself, and the discomfort of the repair. Referral to an Emergency Department may be necessary to perform procedural sedation for both a thorough evaluation and a subsequent repair.

Table 44.12 Indications for specialty referral for eyelid lacerations

Full-thickness lid lacerations	Orbital fat prolapse
Lacerations involving the lid margins	Tear drainage system involvement
Avulsed tissue	Orbital injury (including foreign body)

Auricular Lacerations

Auricular lacerations to the ear may involve any (or all) of the layers of the ear, including the cartilaginous components of the pinna in addition to the skin and subcutaneous adipose tissue of the lobule. Be sure to consider an other potential cause of these lacerations in sport stemming from earrings. Regardless of the cause, it is essential that each anatomic layer be reapproximated. Repairs are best performed within the first 24 hours of the injury.

Initial Management

Adequate anesthesia of the ear can be achieved via local injection with an anesthetic or via a ring block (as discussed in the section "Auricular Hematomas" in this chapter and shown in Fig. 44.25). When repairing auricular lacerations, 5-0 sutures are typically appropriate. Absorbable, monofilament suture should be utilized first to close the perichondral layer and cartilage layers, respectively. This can be performed in a simple, interrupted fashion. Remember that ear cartilage can fracture easily, so avoid the use of forceps with teeth. The overlying subcutaneous and skin layers can then be closed with simple, interrupted 5-0 or 6-0 absorbable suture. Non-absorbable suture can be utilized in these layers as well, with the benefit being an aesthetically pleasing wound that heals faster due to less associated inflammation compared to absorbable suture. However, it obviously necessitates a subsequent suture removal. Interrupted sutures are preferred over running sutures since they may enable better wound approximation and allow for drainage from the wound, thereby decreasing the risk for a post-traumatic auricular hematoma or seroma [52]. The skin wound edges should be everted slightly and not be excessively tight.

Indications for Referral

There are several indications for a referral to an Otolaryngologist in order to repair an auricular laceration, as noted in Table 44.13.

Follow-up Care

The repair should be re-evaluated in 24–48 hours to assess for evidence of local infection, an auricular hematoma, and/or a seroma. Otherwise, if non-absorbable sutures are placed, they should be removed in 7–10 days.

Table 44.13 Indications for Otolaryngology referral for auricular lacerations

Auricular avulsions	Lacerations involving the external auditory canal
Cartilage fractures	Associated middle or inner ear injuries
Chronically split earlobe	Evidence of a basilar skull fracture

Complications

Auricular notching may follow even after a pristine repair. The result of auricular cartilage loss, this can occur secondary to poor skin or cartilage alignment, cartilage damage, vascular insufficiency, and/or a local infection. Other potential complications include the development of an auricular hematoma or a seroma and a local infection (chondritis or perichondritis). *Pseudomonas* accounts for 95% of chondritis and perichondritis cases [108]. As such, adults should be treated with oral fluoroquinolones (despite the risk of potential tendon damage) or be committed to parenteral antibiotics.

Pediatric Considerations

If a pediatric patient develops chondritis or perichondritis, parenteral antibiotics (e.g., ceftazidime) may be necessary to treat the infection as fluoroquinolones are generally avoided in this age group [109].

Nasal Lacerations

Lacerations to the nose can be quite complex as well, involving the external skin, cartilage, and/or intranasal mucosa. Similar to auricular lacerations, it is imperative that the skilled team physician close all of these anatomic layers, respectively. Care should be taken to maximize the cosmetic outcome in nasal lacerations.

Initial Management

As with most lacerations, adequate anesthesia is critical to a successful repair. For nasal lacerations, this can be achieved via local injection or via a regional block. Specifically, an infraorbital nerve block should provide excellent coverage to facilitate the necessary reapproximation (Fig. 44.52).

The key to repairing nasal lacerations lies in the alar margins at the entrance of the nares. 5-0 or 6-0 non-absorbable suture should be utilized to repair this layer in an interrupted fashion while following the contours of the nose. It is acceptable to place these sutures in a vertical mattress fashion. If the athlete prefers to avoid future suture removal (in the context of an isolated, superficial nasal laceration), 5-0 fast-absorbing gut suture can be considered. Regarding the cartilage layer of the nose, there is some disagreement in the literature as to if this layer should be reapproximated. Some contend that the cartilage will be closely approximated by repairing the overlying tissue, and thus the direct placement of suture through the cartilage is unnecessary [110]. Others argue that the cartilage should be reapproximated directly via the placement of interrupted, 5-0 absorbable monofilament suture [67]. The most important aspect of the cartilage layer is to ensure that it is realigned. Therefore, if necessary, a suture repair of this layer appears safe. Lastly, if the mucosal layer is involved, utilize 5-0 or 6-0 non-absorbable suture to repair this layer in a simple interrupted fashion.

Indications for Referral

Significant injuries to the nasal cartilage, ala, and/or columella (the cartilage bridge between the nares) warrant an urgent referral to an Otolaryngologist or a Plastic Surgeon (Fig. 44.53). Other indications for such a referral include noteworthy tissue avulsion and/or associated fractures that will require a subspecialist evaluation (e.g., nasal bone fractures).

Fig. 44.52 External (left) and intraoral (right) approaches to the infraorbital nerve block

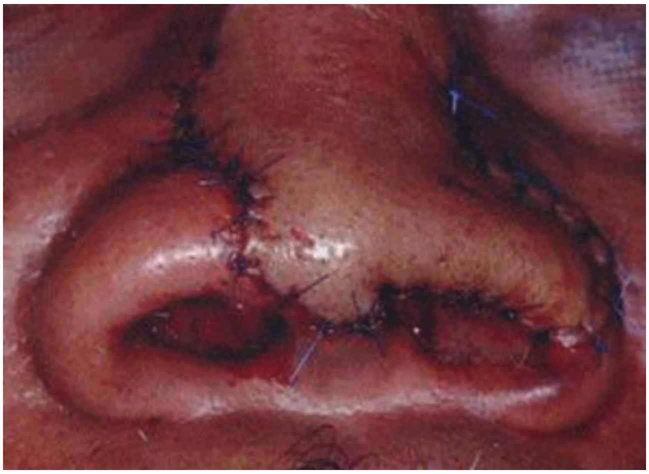

Fig. 44.53 A complex nasal ala and columella laceration that warrants a subspecialist referral (Image courtesy of Herford [119])

Fig. 44.54 The vermillion border (black line) is the most critical landmark in repairing lip lacerations

Follow-up Care

All nasal laceration repairs should be re-evaluated in 24–48 hours to assess for adequate healing and the absences of complications. Otherwise, non-absorbable sutures should be removed roughly 5 days after placement.

Complications

Deformities to the nasal cartilage can result from a suboptimal repair. Other potential complications include local infection, abscess formation, and nasal passage obstruction due to injury-associated edema.

Pediatric Considerations

Finer, non-absorbable suture (6-0) should be considered in pediatric nasal lacerations. Otherwise, there are no additional significant special pediatric considerations.

Lip Lacerations

The lips play several critical roles in our daily lives, several of which are not readily apparent. Social interaction is promptly directed at the lips (as well as the eyes), making the lips of significant aesthetic importance. Tactile sensation, food consumption, and speech articulation are additional functions of the lips [111]. Therefore, proper repair of lip lacerations carries appreciable weight for the injured athlete.

Initial Management

Primary closure of lip lacerations is the preferred route unless the wound is >24 hours old and there is a risk of infection. Upon embarking on the procedure, selecting the anesthesia plan may be slightly different than for other facial lacerations. Direct infiltration of the lip should be avoided as it will distort the tissue and affect the cosmesis of the repair. Thus, the adept team physician will select between the infraorbital nerve block (Fig. 44.52) for upper lip lacerations and the mental nerve block for lower lip injuries (Fig. 44.56, which is discussed in detail later in this chapter). After thorough irrigation, debridement of the lip should be kept to a minimum. Removing such tissue can distort the original architecture of the lip and affect the cosmetic outcome. The presence of a large amount of devitalized tissue warrants consultation with a Plastic Surgeon, Otolaryngologist, or Oral Maxillofacial Surgeon.

The vermillion border (the demarcation between the lip and the surrounding facial skin) is the landmark of critical importance for lip lacerations (Fig. 44.54). The vermillion tissue is either wet (inner portion of the lip when the mouth is closed) or dry (the outer, visible portion when the lips are closed). Superficial dry vermillion lip lacerations are straightforward. They should be closed with 5-0 or 6-0 chromic or fast-absorbing chromic gut suture. Simple interrupted sutures will suffice for this repair.

As for superficial wet vermillion lacerations, a majority of them do not require closure. However, if such a laceration is >2 cm, has a gaping defect, or persistently bleeds it should be closed with absorbable 5-0 or 6-0 chromic gut. It is best to bury these sutures to avoid irritating the gums, mucosa, and/or tongue.

The most technically difficult lip laceration repair is the one that traverses the vermillion border. The first stitch in this repair is the most important. 6-0 non-absorbable suture should precisely reapproximate the vermillion border. Once this suture is in place, the remainder of the laceration can be closed with simple interrupted sutures. Non-absorbable 5-0 or 6-0 suture is preferred for this endeavor. If there is a con-

cern about access to follow-up, 6-0 fast-absorbing chromic gut suture could be utilized in stead.

Through-and-through lacerations of the lip warrant special consideration as well. Before the repair commences, traumatized minor salivary glands should be excised to prevent the formation of a mucocele. The vermillion border should then be approximated with a single 6-0 non-absorbable suture as described above. Then, the deep fibrofatty junction should be closed with 4-0 or 5-0 absorbable suture. Next, re-irrigate the wound from the external aspect. If the orbicularis oris muscle is involved it should be closed with 5-0 absorbable suture. At that point the wet and dry vermillion borders should be reapproximated as detailed previously. The remaining skin beyond the vermillion border can then be closed with 5-0 or 6-0 non-absorbable suture. Again, 6-0 fast-absorbing gut could be used for this step, particularly if follow-up is a concern.

Antibiotic prophylaxis is the next issue to consider in managing these wounds. The risk of wound infection following a laceration in or around the lip has been cited to be up to 27% [112]. The literature, however, is mixed on whether or not empiric antibiotics are beneficial. The totality of the evidence suggests that when significant mucosal involvement is present (i.e., the wet vermillion border), prophylactic antibiotics are appropriate (e.g., amoxicillin or clindamycin) [111].

Following the laceration repair, patients should apply topical antibiotic (e.g., bacitracin) to the laceration twice per day. A soft diet is advised for 2–3 days with water rinsing performed after food consumption. It is advisable to avoid spicy and/or salty foods until the wound is healed. Also, these patients should refrain from using straws to avoid compromising the repair.

Indications for Referral

Plastic Surgery, Otolaryngology, or Oral Maxillofacial consultation should be considered in the context of crush wounds or a significant loss of tissue (especially those involving the upper lip), avulsion or luxation dental injuries, and/or associated facial fractures (e.g., Le Fort or mandible fractures).

Follow-up Care

Reassessment of the laceration repair should be performed by the team physician in 48–72 hours to ensure adequate healing. When non-absorbable sutures are placed, they can be removed in 3–5 days. Any absorbable sutures that persist beyond 5 days can also be extracted as well.

Complications

As previously suggested, suboptimal lip laceration repairs may result in cosmetically unacceptable outcomes and/or affect one's speech. Significant scar development may occur in addition to localized infection and/or abscess formation.

Pediatric Considerations

As a general rule, 6-0 suture should be utilized for the repair of pediatric lip lacerations whenever possible. Due to the significant cosmetic and functional repercussions of this procedure, one's threshold for referring the anxious or uncooperative pediatric athlete for procedural sedation should be lowered.

Tongue Lacerations

The only muscle in the body attached at one end (barring injury), the tongue is extremely vascularized. As such, significant hemorrhage may result, which can impair one's ability to repair the laceration (when needed) and also lead to hypovolemia or even shock. Hemostasis is the name of the game with tongue lacerations, as the muscle generally heals well and cosmesis is a secondary concern (if at all).

Initial Management

Many tongue lacerations will not require any intervention. Particularly in adolescents, adequate healing will often transpire without a primary repair; treatment consists of a soft diet and dilute peroxide mouth rinses over several days. Indications for a suture repair are noted in Table 44.14.

When indicated (Fig. 44.55), adequate repair of the laceration requires sufficient anesthesia, which can be particu-

Table 44.14 Indications for primary closure of tongue lacerations

Lacerations >1–2 cm in length
Through-and-through lacerations
Large flaps or gaps in the tongue (Fig. 44.55)
Lacerations with significant hemorrhage
Bisection of the tongue

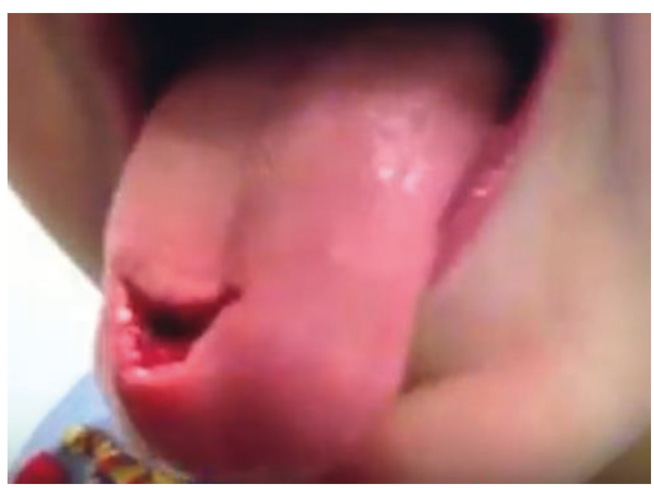

Fig. 44.55 A tongue laceration with a gap that warrants primary closure

larly difficult in very young patients. Consider the use of lidocaine-soaked gauze applied to the tongue for 5 minutes. One may also utilize 1% lidocaine with epinephrine to both anesthetize the tongue and help control the bleeding. Alternatively, an inferior alveolar nerve block (potentially bilaterally) can be effective in anesthetizing the tongue.

Once sufficient analgesia is achieved, sutures can then be placed in an interrupted fashion. If possible, it is recommended to wrap the distal aspect of the tongue in gauze and maintain traction to facilitate an optimal repair. It is generally recommended that 4-0 absorbable sutures be utilized. Such sutures can become untied, and consequently burying the knots is advisable. When feasible, closure of all three layers of the tongue should be attempted (i.e., the inferior mucosa, muscle, and superior mucosa). However, closing the muscle layer of the tongue is generally sufficient to control the bleeding, and the mucosal layers will heal rapidly even if not completely reapproximated. For through-and-through lacerations, one should consider closing the top half of the tongue separately from the bottom half. Cosmesis is not generally a concern in the setting of tongue lacerations.

While most lacerations will be due to human bites (by technicality), the risk of infection is low. Prophylactic antibiotics are not typically necessary, but in cases where they are deemed appropriate (e.g., contaminated wounds and associated fractures), amoxicillin-clavulanate is an appropriate choice. Dual therapy with trimethoprim-sulfamethoxazole and clindamycin in penicillin-allergic athletes is a reasonable option. Three to five days of treatment is considered sufficient.

Post-repair instructions should highlight the maintenance of good oral hygiene to reduce the risk of infection, a soft diet for a couple of days to facilitate rapid healing, and the use of ice chips or popsicles (any flavor) to reduce the associated tongue edema.

Indications for Referral

In the event that the laceration complexity is beyond the skillset of the team physician or the patient's comfort level will not tolerate the repair (i.e., procedural sedation is needed), an immediate referral to an Emergency Department should be sought.

Follow-up Care

Re-evaluation of the laceration repair is recommended within the first 48 hours to assess the wound healing and/or for complications. If dental injuries are noted, follow-up with a Dentist is advised within 24 hours of the injury. In the event that non-absorbable sutures are utilized, these should be removed approximately 5–7 days after placement.

Complications

Complications of tongue lacerations are rare but include localized infections. Suture knots can pose one potential additional complication. If they are not buried the knots can loosen and potentially compromise the repair.

Intraoral Lacerations

Perhaps surprisingly, the majority of intraoral lacerations are the result of blunt trauma. However, traumatic causes exist (e.g., tooth bites), as do external mechanisms (e.g., an opponent's finger). Due to the robust vascular supply, these lacerations heal quickly, and a majority of them will not require any intervention. Many of those that do necessitate an intervention can be addressed by the capable sideline physician. With that said, there are a couple of nuances of these injuries that warrant subspecialist evaluation and treatment.

Initial Management

Care should be taken to assess for concomitant injuries. Be certain to ensure that the airway is intact and protected. Dental injuries (e.g., avulsion, luxation) should be treated in advance of addressing any lacerations that are appreciated.

Recognizing which intraoral lacerations require primary care closure is obviously critical in managing these injuries. Typically, if the wound is wide enough to collect food particles, is greater than 2 cm in length, or there is a flap of tissue that can interfere with mastication, a primary closure is recommended. In those cases, local or regional anesthesia should then be considered. Lidocaine or bupivacaine, either with or without epinephrine, is appropriate for such anesthesia. If one elects to proceed with a regional nerve block, several options are available to a provider depending upon the location of the intraoral laceration. An infraorbital nerve block (Fig. 44.52), mental nerve block (Fig. 44.56), and an inferior alveolar nerve block are all viable options. Once sufficiently numb, one may proceed with the necessary repair.

Fig. 44.56 Illustration of the proper placement of a mental nerve block

Absorbable suture is the preferred choice for these lacerations, for instance 4-0 or 5-0 chromic gut.

For gaping buccal mucosa and gingival lacerations in need of primary closure, one should place deep, buried, interrupted sutures in the submucosal tissue. These can then be followed by additional interrupted sutures in the mucosal surface. It is prudent to use as few sutures as possible to avoid irritation with chewing or tongue movement. For lacerations that feature gingival avulsions, further dissection of the tissue may be needed to facilitate the repair. If there is insufficient mucosal space to allow for passage of the suture, one can guide the needle between two nearby teeth, creating a flossing-type suture. The suture is then passed around a tooth and back through the avulsed tissue, with the knot overlying the lacerated tissue (Fig. 44.57) [104].

Post-procedural recommendations are similar to those provided for lip lacerations. These include a soft diet for 2–3 days and mouth rinsing with water following eating. Athletes should try to avoid spicy and/or salty foods until the wound is healed. Lastly, it is advisable to refrain from using straws; negative pressure can lead to ecchymosis and/or bleeding from the wound. In the setting of gaping wounds, antibiotic prophylaxis (e.g., amoxicillin or clindamycin) is recommended [113].

Indications for Referral

Crush wounds and those with large defects should be evaluated by an Oral Surgeon or a Plastic Surgeon. Injuries that involve the salivary glands, parotid duct, and/or submandibular duct should be sent for immediate evaluation by such a subspecialist. As with other facial lacerations, when concomitant facial fractures are present that require a subspecialist evaluation, the athlete should be promptly referred. Such additional injuries include dental avulsions or luxations.

Follow-up Care

Most patients will not require any further follow-up (so long as absorbable suture is used), as these wounds heal rapidly. Any patients with risk factors for delayed wound healing (e.g., diabetes mellitus) should have the repair re-evaluated in 48–72 hours.

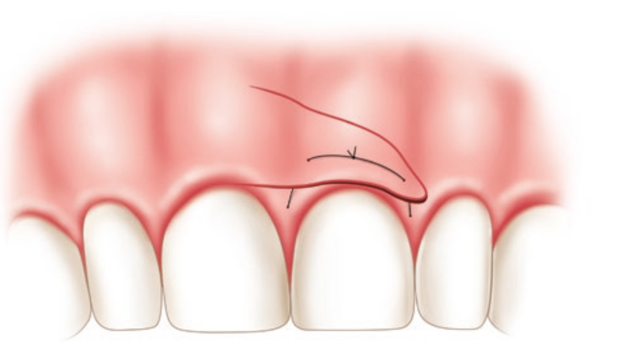

Fig. 44.57 A gingival avulsion laceration status post-primary closure

Complications

Intraoral lacerations in isolation are not fraught with significant complications. Local infection and abscess formation are the primary complications for which a team physician should be vigilant.

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Soft Tissue Neck Injury

45

Allison D. Lane

Key Points

- Cervical strain is the most common soft tissue neck injury.
- Soft tissue neck injuries in sports are most common in contact sports and with high-velocity trauma.
- Be aware of the potential for injury to vessels, digestive tract, and airway that require emergent evaluation and treatment.
- In cases of penetrating injury with retained foreign body, stabilize the object, and *DO NOT* remove it.
- Follow your ABCDs for initial management.

Introduction

In sports, blunt neck trauma is much more common than penetrating trauma.

The most common soft tissue neck injury in sports is a cervical strain. In most cases, this is usually a benign and self-limiting condition. However, there are other much less common injuries, which, if not recognized and treated appropriately, may cause significant morbidity or mortality. All soft tissue neck injuries occur more frequently in contact sports and with high-velocity trauma.

Anatomy

Neck Borders

- Posterior – spine
- Anterior – larynx/trachea
- Superior – head
- Inferior – chest

Neck Zones (Table 45.1 and Fig. 45.1)

- Zone I – thoracic inlet to cricoid cartilage
- Zone II – cricoid cartilage to angle of mandible
- Zone III – angle of mandible to skull base

Neck Contents

- Musculoskeletal – vertebral bodies; cervical muscles, tendons, ligaments; clavicles; first and second ribs; hyoid bone
- Neural – spinal cord, cervical roots of phrenic nerve and brachial plexus, recurrent laryngeal nerve, cranial nerves IX–XII, stellate ganglion
- Vascular – carotids, vertebral arteries, vertebral vein, brachiocephalic vein, jugular veins
- Visceral – thoracic duct, esophagus and pharynx, larynx and trachea
- Glandular – thyroid, parathyroid, submandibular
- Fascia – superficial and deep cervical fascia [1, 2].

Table 45.1 Zones of the neck for penetrating trauma

	Location	Contents
Zone 1	Thoracic inlet to cricoid cartilage	Mediastinal structures, thoracic duct, proximal carotid artery, vertebral/subclavian artery, trachea, lung, esophagus
Zone 2	Cricoid cartilage to angle of mandible	Carotid/vertebral artery, larynx, trachea, esophagus, jugular vein, vagus, and recurrent laryngeal nerves
Zone 3	Angle of mandible to skull base	Distal carotid artery, vertebral artery, distal jugular vein, salivary/parotid glands, CN 9–12

A. D. Lane (✉)
Department of Emergency Medicine, University of Arizona/Banner University Medical Center, Tucson, AZ, USA
e-mail: alane@aemrc.arizona.edu

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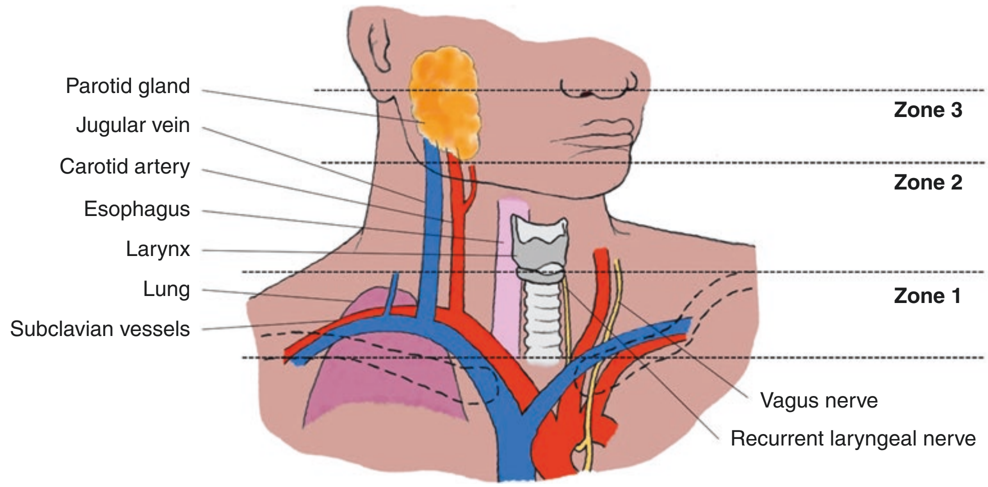

Fig. 45.1 Neck zones (Illustrated by Dr. Yvonne Chow)

Blunt Neck Trauma

Cervical Strain

Mechanism of Injury

- This is most commonly a stretch injury to the muscle fibers (strain) and/or ligament (sprain) from a traction-type injury or forceful contraction against resistance and most commonly occurs at the musculotendinous junction.
- Cervical sprain and strain often occur in combination, in addition to a contusion from a direct blow to the soft tissues [3, 4].

Classification

- Injury may be either acute or chronic.
  – Chronic may include repetitive stress injury or abnormal strained posture.
- Injury may also be high velocity or low velocity.
  – Low velocity may vary from acute to chronic in presentation [4].

Clinical Presentation: Signs and Symptoms

- Presentation may vary from acute to chronic.
- Pain can range from mild to severe.
- Radicular pain can occur in all types of cervical spine injuries including strain.
- Those with voluntary movement of the neck are less likely to have more significant injury.
- Edema of cervical tissues without pitting is common. They may be boggy to tight. There may be mild warmth over involved tissues.
- Limited range of motion due to muscle spasm is likely and does not necessarily reflect severity of injury.
- In those with radicular symptoms, consider performing the Spurling maneuver to assist in differentiating nerve impingement from arthritic or disc herniation rather than muscular or ligamentous strain. Reproduction of radicular symptoms with this maneuver suggests more than just strain [4].

Diagnosis

- The most important issue is to differentiate cervical strain from a more serious injury.
- Evaluation of the mechanism of injury may provide clues to the relative risk of strain versus other injury [4].
- Imaging to determine more serious injury may include x-ray (AP/lateral/odontoid views), CT, and/or MRI.

Initial Management

- Consider placement of a cervical collar if more than cervical strain is considered, if high-risk patient or mechanism, and according to protocols.
- History and physical have been shown effective in guiding the need for imaging using NEXUS criteria or the Canadian c-spine rule [4].
- NEXUS criteria
- The NEXUS criteria for c-spine is a well-validated clinical decision rule used to assist in safely ruling out cervical spine injuries without the need to obtain radiographic studies.
- Initial validation studies showed a sensitivity of 99.6% for clinically significant cervical spine injuries (majority of subsequent studies showing 90-100% sensitivity) and a sensitivity of 99.0% for all cervical spine injuries.
- There is no age cutoff, though use caution when applying the criteria to patients over 65 years of age, as sensitivity may be as low as 66-84% [5].
- The National Emergency X-Radiography Utilization Study (NEXUS) Low-Risk Criteria (NLC) state cervical spine radiography is indicated for patients with trauma unless they meet all the following criteria:
  – No posterior midline cervical spine tenderness
  – No evidence of intoxication
  – A normal level of alertness

  - No focal neurologic deficit
  - No painful distracting injuries
- Canadian c-spine rule.
- The Canadian c-spine rule is also well validated. It performs as well or better than NEXUS in terms of sensitivity, but is more complex. It is highly sensitive for cervical spine injury, with a majority of studies showing 99-100%. It is easiest to use with a smartphone app or digital reference.
- Exclusion criteria:
  - Non-trauma patients
  - GCS < 15
  - Unstable vital signs
  - Age <16
  - Acute paralysis
  - Known vertebral disease
  - Previous c-spine surgery
- Step 1: Presence of high-risk factors.
- Imaging is recommended if any of the following high-risk factors are present:
  - Age 65 or greater
  - Extremity paresthesias
  - Dangerous mechanism (fall from at least 3ft/5 stairs, axial load injury, high-speed motor vehicle collision, rollover, ejection, bicycle collision, motorized recreational vehicle)
- If none of the previous high-risk factors are present move on to the next step.
- Step 2: Presence of low-risk factors.
- Imaging is recommended if none of the low-risk factors listed below are present:
  - Sitting position in the emergency department
  - Ambulatory at any time
  - Delayed (not immediate) neck pain
  - No midline tenderness
  - Simple rear-end motor vehicle collision (not pushed into traffic, hit by bus/large truck, roll over, hit by high-speed vehicle)
- If a low-risk factor is present move on to the next step.
- Step 3: Active rotation of the neck 45 degrees left and right
- Imaging is recommended if the patient is unable to actively rotate their neck.
- The cervical spine can be clinically cleared if the patient can rotate their neck.
- Type of imaging
- Consider CT for high-risk patients, severe head injury, high-energy mechanisms, or neurologic deficits as CT has higher sensitivity than plain films for detection of fractures.
- Consider performing an MRI with negative CT or lateral plain films in the setting of multiple injuries, obtunded or difficult to examine patients, persistent focal neurologic deficits or radicular pain, or new neurologic symptoms to eliminate instability and cord injury. MRI is better for evaluating spinal cord and ligamentous injury.
- High-risk patients should be reassessed even with normal static radiologic evaluation to make sure no ligamentous instability is present. Muscle spasm and limited range of motion can hide subluxation and ligamentous injury [6].

Treatment

- Pain control with NSAIDs is the mainstay of treatment, but consider the addition of acetaminophen and muscle relaxants if needed.
- Use ice for inflammation and pain control. Consider treatment with heat for muscle spasm.
- Stress early mobilization with range of motion exercises. Soft collars have not proven effective, and their use for longer than 10 days is associated with deconditioning and weakness of the cervical muscles.
- Discuss avoidance of acute unusual postures for prolonged periods of time and chronic straining or repetitive motion of the neck [3, 4].

Follow-Up Care

- Follow up with athletic staff or provider familiar with rehabilitation therapies.
- Early range of motion and therapeutic exercises may be of benefit.
- Consider outpatient dynamic flexion/extension imaging in 1 week if continued pain or tenderness, as muscle spasm and limited range of motion can hide subluxation and ligamentous injury [4].

Return to Sports

- Those who are compliant with the recommended treatment generally quickly return to full function. Athletes may gradually return to sport as able based on symptom resolution, full cervical range of motion, and normal strength [3, 4].

Complications

- There are usually few long-term sequelae, but injury may lead to decreased function secondary to disuse and pain [4].

Esophageal Injury

Mechanism of Injury

- There may be direct injury from compression between the sternum and thoracic spine, like with a posterior sternoclavicular dislocation or spinal fractures.
- Another mechanism for injury may be an increase in intraluminal pressure against a closed glottis, which can result in a tear of the esophageal wall.
- This rarely occurs in isolation without other associated injuries [7, 8].

Clinical Presentation: Signs and Symptoms

- Presentation may include subcutaneous emphysema, dysphagia, odynophagia, hematemesis, hemoptysis, bloody saliva, tachycardia, and fever.
- This also may be clinically silent [2, 8, 9].

Diagnosis

- Contrast-enhanced esophagography has sensitivity of about 89%.
- If esophagography is negative, consider barium swallow (barium is more sensitive but has an increased risk of pulmonary complications).
- Endoscopy should also be considered. Flexible endoscopy has a lower diagnostic yield than rigid, but also a lower complication rate.
- The sensitivity approaches 100% with both contrast-enhanced imaging and endoscopy [2, 8, 9].

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Emergent evaluation in the emergency department is required.
- If unstable, they should go directly to the operating room [2].

Return to Sports

- Return will depend on the severity of injury and recovery.

Complications

- These are often missed and a delay in diagnosis is associated with increased morbidity and mortality.
  – When surgery is performed within 24 hours post-injury, the survival rate is over 90% (compared to 65% when over 24 hours).
- The leading cause of delayed death in neck trauma is esophageal injury.
- Most of these injuries have associated laryngotracheal injury.
- There is the potential for developing mediastinitis and abscess formation [2, 7].

Vascular Injuries

Mechanism of Injury

- Injuries to the carotid artery and vertebral artery (occlusion, dissection, thromboembolism, intimal tears, pseudoaneurysm, rupture, AV fistula, and transection) may occur.
- Hyperextension and rotation can stretch the carotid artery and compromise the vessel, and acute flexion may crush the carotid artery between the mandible and spine, which could lead to thrombosis or intimal damage.
- Hyperflexion, hyperextension, distraction, facet dislocation, and cervical spine fractures are also associated with vertebral artery injury [1, 8, 9].

Most Common Sports

- Any sport involving neck range of motion has the potential for blunt vascular injury. Vertebral artery injury, specifically, may be associated with sports involving repetitive neck movements, especially swimming [1].

Clinical Presentation: Signs and Symptoms (Table 45.2)

- These injuries may be asymptomatic initially or present with only vague symptoms.
- "Hard signs" of vascular injury include bruit/thrill, expanding or pulsatile hematoma, pulsatile or severe hemorrhage, and pulse deficit.
- "Soft signs" of vascular injury include hypotension, shock, stable hematoma, and central or peripheral nerve ischemia.
- Other signs/symptoms may include altered level of consciousness, vomiting and dizziness, hemiparesis, and Horner's syndrome.
- Carotid artery injury, specifically, may present in a variety of ways. There may be ipsilateral cerebral findings with contralateral hemiplegia, sensory loss, and aphasia. A bruit may be present and could indicate partial disruption of the artery. Horner's syndrome (ipsilateral miosis, ptosis, anhidrosis) would suggest disruption of the thoracic sympathetic chain encircling the artery.
- Vertebral artery injury, in particular, is difficult to diagnose since many are asymptomatic due to collateral circulation. Others will present with vague symptoms such as nausea and vomiting, dizziness, vertigo, or visual disturbances. There may also be a variety of neurologic deficits without an apparent source because the vertebral artery connects to the basilar artery and an embolic event may affect the ipsilateral or contralateral posterior circulation [1, 2, 8, 9].

Diagnosis

- The "gold standard" for diagnosis has been four-vessel angiography.

Table 45.2 Signs indicating vascular injury

"Hard signs"	Emergent management	Bruit/thrill, expanding or pulsatile hematoma, pulsatile or severe hemorrhage, pulse deficit
"Soft signs"	Close observation in the hospital and reevaluation	Hypotension, shock, stable hematoma, central or peripheral nerve ischemia

- The sensitivity is 99%.
- However, this is now mostly used only with inconclusive testing or positive CTA or duplex sonography for surgical planning.
- CTA has overtaken angiography as the initial test.
  - CTA is faster, less expensive, and noninvasive and does not require interventional radiology.
  - Sensitivity is now 90–100% and even better with 64-slice.
- Duplex ultrasound may also be used, typically only in those who are asymptomatic or sustained a low-risk event. Some studies show near comparable CTA sensitivity, though this is operator dependent. In addition, the majority of carotid artery injuries occur near the bifurcation, where ultrasound visualization is less reliable.
- MRA has reported 75% specificity and 67–95% sensitivity, though this is difficult to obtain in the acute trauma setting [1, 2, 8, 9].

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Emergent evaluation in the emergency department is required.
- If unstable, they should go directly to the operating room [2].

Return to Sports

- Return depends on the severity of the initial injury, type/quality of repair, and individual recovery.
- It may also need to take into consideration return to sports if athlete is on anticoagulation therapy [1].

Complications

- Luminal narrowing and thrombosis occur in the majority of vascular injuries and many are placed on anticoagulation therapy.
- Embolic events are potential complications.
- Life-threatening hemorrhage is also a potential complication [1, 8].

Laryngotracheal Injuries

Mechanism of Injury

- Injuries may include vocal cord damage or disruption, arytenoid cartilage dislocation, tracheal transection, cartilage fractures of the thyroid or cricoid, hyoid bone fracture, mucosal tears, or submucosal hematomas.
- There is anterior protection of the larynx from trauma by the mandible and posterior by the spine. However, compressive force on the larynx (usually mid- to lower neck) may crush the larynx against the vertebral column.
- Airway compromise may occur from thyroid cartilage fractures by shortening and destabilizing the thyroarytenoid tendon, fractures of the cricoid cartilage ring may have associated loss of subglottic airway, and soft tissue injury may cause edema/hematoma that affects patency [8, 10].

Most Common Sports

- This is more likely in clothesline injuries, high contact sports, or when an athlete strikes a stationary object.
- This may occur in any sport where there is a powerful direct blow to the neck, such as with clothesline, crush, or strangulation injuries.
- Most occur in high-velocity sports (like cycling, motorcycle racing, ice hockey) or in martial arts.
- Cheerleading accounts for the most laryngotracheal injuries in female athletes.
- Fatal laryngeal injuries have been reported in baseball, and clothesline injuries during snowmobiling, skiing, and motorcycling.
- There is a case series of three lacrosse players who sustained laryngeal fractures from being hit with a lacrosse ball to the neck [9, 11, 12].

Epidemiology

- Although not prevalent, laryngotracheal injury is second only to intracranial injury as the most common cause of death among patients with head and neck trauma.
- Sixty percent of all laryngotracheal traumas are due to blunt neck trauma [9, 11].

Clinical Presentation: Signs and Symptoms

- The most common presenting symptom is hoarseness.
- Also, patients may have dysphagia, dyspnea, hemoptysis, stridor, tachypnea, drooling, cervical subcutaneous emphysema or crepitation, laryngeal tenderness, and distortion of anterior neck anatomy.
- Typically there is no clear correlation between presenting signs and symptoms of an athlete with laryngeal injury and the extent of the injury [9, 11].

Diagnosis

- Perform high-resolution CT once the airway is secure [8, 9].

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Emergent evaluation in the emergency department is required.
- If unstable, they should go directly to the operating room [2, 8].

Follow-Up Care

- Regular endoscopic exams are used to assess recovery.
- Phonic and speech therapy evaluation are used for follow-up as well [8, 9].

Return to Sports

- Return depends on severity of initial injury, quality of repair, and individual recovery. Prior to return, symptoms and clinical findings will need to have resolved.
- Considerations will include quality of airway, voice, and swallowing. Many athletes are able to regain normal speaking and swallowing and are able to return to athletics [8, 10, 11].

Complications

- Cricotracheal separation is a potential complication.
  - This is associated with high chance of asphyxiation and mortality.
  - This is associated with recurrent laryngeal nerve injury.
- The most common complication is dysphonia with hoarseness, voice fatigue, and poor vocal control.
- Other complications include granulation tissue formation, laryngeal and tracheal stenosis, subglottic stenosis, vocal cord paralysis from recurrent laryngeal nerve injury, and laryngoesophageal fistula [8–10].

Pediatric Considerations

- There is a high association of laryngotracheal injury with cervical spine injury (up to 50%).
- There are anatomy differences between children and adults.
  - The larynx is higher in the neck and protected by the mandible in children.
  - There are less laryngeal fractures due to elasticity of cartilage in children.
  - There is an increased likelihood of subsequent airway obstruction due to loosely attached submucosal tissues leading to increased likelihood of soft tissue damage with edema or hematoma in children.
  - The cricothyroid membrane is narrower and less likely to have laryngotracheal separation in children [9].

Penetrating Trauma

Most Common Sports

- While extremely uncommon, penetrating injuries may occur in sport. Here are a few examples:
  - In 1989 Buffalo Sabres goalie Clint Malarchuk was slashed to the side of his neck by another player's skate. There was pulsatile hemorrhage, as he had cut a jugular vein, but the athletic trainer was able to place pressure to the wound within 10 seconds and Malarchuk was taken to the hospital. He ultimately received 300 stitches and missed five regular season games [13].
  - A high school track and field manager was speared through the neck with a javelin in 1993 while setting up for a meet. The javelin entered from behind the ear and remained in the neck, with about a foot exiting from the anterior neck. The patient was immobilized on a backboard and EMS cut off 6 feet of the javelin just so he could fit in the helicopter to transport to the trauma facility. He went to the operating room and luckily had only sustained injury to the muscle mass [14].
  - In 2012 an official at a high school track and field event died from being impaled by a javelin. The javelin hit him in the cheek and traveled down to his neck. He grabbed the javelin and pulled it out. He was taken to the hospital but died from rupture to the carotid artery [15].
  - In 2015 a mountain biker fell and was impaled by a tree branch. He drove himself to the hospital, keeping the branch in place. After the airway was secured and imaging obtained, surgeons removed the branch. The man was discharged and has had no further complications [16].
  - In 2017 a 7-year-old girl fell on the tail end of her arrow during an archery competition, which pierced her neck. The arrow ends were cut and stabilized prior to transport to the hospital. She had no life-threatening injuries [17].
  - A 13-year-old football player presented 3 hours after penetrating injury to the suprasternal region from the hook of a goal post. He had an expanding hematoma over the neck but airway was patent. There was a deep laceration to the suprasternal region, and saliva was noted to be coming from the wound. There was also subcutaneous emphysema in the neck. X-ray showed pneumomediastinum. Contrast-enhanced CT neck and chest along with esophagogram showed esophageal injury in the neck, subcutaneous emphysema of the neck, and pneumomediastinum. The injury was repaired in the operating room; patient discharged on day 8 and was noted to be doing well 3 months later on follow-up [18].

Zone 1 Injury

Epidemiology

- This is the highest risk zone.

Classification

- Located from thoracic inlet to the cricoid cartilage.
- Structures include the mediastinal structures, thoracic duct, proximal carotid artery, vertebral/subclavian artery, trachea, lung, and esophagus.

Clinical Presentation: Signs and Symptoms

- Presentation varies depending on injured structure(s).
- Given the presence of vascular structures, evaluate for “hard” and “soft” signs.
- Given the presence of digestive tract structures, evaluate for subcutaneous emphysema, dysphagia, odynophagia, hematemesis, hemoptysis, bloody saliva, tachycardia, and fever.
- Given the presence of airway structures, evaluate for hoarseness, dysphagia, dyspnea, hemoptysis, stridor, tachypnea, drooling, cervical subcutaneous emphysema or crepitation, laryngeal tenderness, and distortion of anterior neck anatomy.

Diagnosis

- Imaging is based on suspected injuries as noted in the blunt trauma section.

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Apply direct pressure for active bleeding.
- Do not remove any penetrating object.
- Consider emergent evaluation in the emergency department.
- If unstable, they may need to go directly to the operating room.
- In areas that are difficult to access, consider operative management regardless.

Return to Sports

- Return depends on the severity of injury and recovery.

Zone 2 Injury

Epidemiology

- This is the most exposed zone and most likely to be injured.
- Injuries in this zone have the best prognosis (larger areas of exposure, easier proximal and distal control).

Classification

- Located from the cricoid cartilage to the angle of the mandible.
- Structures include the carotid/vertebral artery, larynx, trachea, esophagus, jugular vein, vagus, and recurrent laryngeal nerves.

Clinical Presentation: Signs and Symptoms

- Presentation varies depending on injured structure(s).
- Given the presence of vascular structures, evaluate for “hard” and “soft” signs.
- Given the presence of digestive tract structures, evaluate for subcutaneous emphysema, dysphagia, odynophagia, hematemesis, hemoptysis, bloody saliva, tachycardia, and fever.
- Given the presence of airway structures, evaluate for hoarseness, dysphagia, dyspnea, hemoptysis, stridor, tachypnea, drooling, cervical subcutaneous emphysema or crepitation, laryngeal tenderness, and distortion of anterior neck anatomy.

Diagnosis

- Imaging should be performed based on suspected injuries as noted in the blunt trauma section.

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Apply direct pressure for active bleeding.
- Do not remove any penetrating object.
- Consider emergent evaluation in the emergency department.
- If unstable, they may need to go directly to the operating room.

Return to Sports

- Return depends on the severity of injury and recovery.

Zone 3 Injury

Classification

- Located from the angle of the mandible to the skull base.
- Structures include distal carotid artery, vertebral artery, distal jugular vein, salivary/parotid glands, and cranial nerves 9–12.

Clinical Presentation: Signs and Symptoms

- Presentation varies depending on injured structure(s).
- Given the presence of vascular structures, evaluate for “hard” and “soft” signs.
- Given the presence of cranial nerves, evaluate for signs of neurologic abnormalities.

Diagnosis

- Imaging performed will be based on suspected injuries as noted in the blunt trauma section.

Initial Management

- Consider the need to establish an emergent airway.
- Consider the need for cervical spine immobilization.
- Apply direct pressure for active bleeding.
- Do not remove any penetrating object.
- Consider emergent evaluation in the emergency department.

- If unstable, they may need to go directly to the operating room.
- In areas that are difficult to access, consider operative management regardless.

Return to Sports

- Return depends on the severity of injury and recovery [2, 8].

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Part VIII

Sports-Specific Injuries

Morteza Khodaee

Aquatic Sports

46

Jack Spittler

> **Key Points**
> - As aquatic sports take place in the water, the nature and evaluation/treatment of injuries is unique.
> - Someone certified in lifeguarding should be available whenever possible during aquatic sport events.
> - Injuries depend on the type of aquatic sport.

Introduction

Aquatic sports are those that are based in water as opposed to on land. This chapter will specifically focus on acute trauma related to swimming, diving, water polo, waterskiing, wakeboarding, scuba diving, and surfing. Rowing and sailing are discussed separately as their own chapters. There are some common themes; however, each aquatic sport has its own unique type of acute injuries that are commonly seen (Table 46.1). Aquatic sports trauma management poses some significant challenges due to the nature of the aquatic environment. Evaluation and treatment of injured athletes is often difficult in the aquatic environment, and therefore these athletes should be examined on dry land whenever possible. Extrication should be done as quickly and carefully as possible due to the risk of drowning or submersion injury. The process of extrication from the aquatic environment can often be challenging and could result in further injury if not done correctly. Therefore, someone certified in lifeguarding should be available whenever possible during aquatic sports events [1].

Table 46.1 Acute injuries by aquatic sport

Aquatic sport	Traumatic injury	Body part(s) commonly affected
Swimming	Fracture Subluxation Concussion/ head injury	Metacarpal, foot, ankle Shoulder, patella Head, neck
Diving	Laceration Fracture Tendon/ ligament injury Concussion/ head injury	Upper extremity, head, face Carpal, distal radius UCL, EPL, wrist (TFCC injury) Head, neck
Water polo	Laceration/ tissue injury Fracture Dislocation Tendon/ ligament injury Concussion/ head injury	Face, eye (corneal abrasion, hyphema, globe rupture), trunk/ extremity (esp. from finger/ toenails) Tooth, nose, facial bone, orbit, phalanges, metacarpal bones Shoulder, IP joint, MCP joint UCL, knee, hip, shoulder Head, neck
Waterskiing	Tendon/ ligament injury Fracture Contusion/ abrasion Laceration	Lower extremity, hip Lower extremity, hip Lower extremity, hip Lower extremity, hip
Wakeboarding	Laceration Concussion/ head injury	Head, face, neck Head, neck
Scuba diving	Barotrauma Fracture Laceration Marine envenomation Fin foot	Ear, lung Teeth, extremities Extremities Multiple Feet
Surfing	Laceration Fracture Dislocation Tendon/ ligament injury Concussion/ head injury	Head, face, neck, lower extremity Upper extremity, finger, lower extremity, head, face, neck Shoulder Lower extremity Head, neck

J. Spittler (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: john.spittler@cuanschutz.edu

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Swimming

Although most musculoskeletal (MSK) problems in swimming are chronic, as it is a noncontact sport, there are occasionally acute traumatic injuries. Metacarpal fractures can occur when striking the hand on the wall at the finish of a sprint. Other hand fractures can occur by hitting the hand on another swimmer or on the lane line. Fractures or lacerations of the foot and ankle can occur from striking the foot on the wall during a flip turn [2]. Acute subluxation or dislocation of certain joints may occur in swimming, especially since many swimmers have underlying ligamentous laxity [3]. Shoulder subluxation (anterior) may occur during hand entry in backstroke, and patellar subluxation may occur with breaststroke kick [4]. Head and neck injuries can occur when a swimmer strikes the wall during backstroke, collides with another swimmer, slips and falls on the pool deck, or strikes their head on the pool bottom when diving in [5]. These injuries in swimming are especially dangerous when they occur in the pool, as safe extrication can be challenging and, if not done correctly, drowning or other serious injury may occur.

Diving

Traumatic injuries in diving are usually related to contact with the diving board or upon water entry. Given that divers enter the water hands first, they are at particular risk for upper extremity injuries. Hand and wrist injuries in divers most commonly occur in the dominant hand (due to hand positions) and usually involve the carpal bones and distal radius [6]. Injury types may include lacerations, fractures, tendon ruptures (e.g., extensor pollicis longus), ligament tears (e.g., ulnar collateral ligament), and triangular fibrocartilage complex (TFCC) injuries [6]. There are no reports in the literature of finger fractures or dislocations in divers. Concussions may occur in diving, due to contact with either the water or the diving board [7]. Other head, neck, and face injuries may occur and are most frequently lacerations or soft tissue injuries [8]. Especially in children, the odds of injury caused by contact with the diving board significantly increases when performing a flip, handstand, or backward dive [8].

Water Polo

Water polo players are susceptible to traumatic injuries given the intense physical contact and high velocities (60–70 km/h) reached by the water polo ball [9]. The most commonly injured body parts are the head and face [10, 11]. This is mainly due to the fact that the only required protective equipment by the main governing body, *Federation Internationale de Natation* (FINA), is a swimming cap with malleable ear protectors [12] (Fig. 46.1). This leaves water polo athletes particularly vulnerable to traumatic injuries of the head and face that include facial laceration, ear drum perforation, nasal/facial bone fracture, dental trauma, corneal abrasion, hyphema, globe rupture, and orbital fracture [9, 10, 13, 14]. According to the World Dental Federation (FDI), water polo is a medium-risk sport for dental injury due to frequent body contact [15]. Despite this risk, FINA

Fig. 46.1 Water polo cap with malleable ear protectors

does not require the use of a mouthguard. In one survey of water polo players, only 7.7% reported using a mouthguard even though nearly 50% had witnessed a dental injury and over 20% had experienced a dental injury themselves [16]. The next most commonly injured body part in water polo is the upper extremity. Anterior shoulder subluxation or dislocation may occur given inherent vulnerability of the shoulder while in the throwing position (abducted and maximally externally rotated), which may result in rotator cuff tear, ligament/capsule tear, labral injury, and shoulder instability [14, 17]. Traumatic injuries to the elbow, wrist, and hand are less common, but can still occur in water polo. These injuries may include thumb UCL tears, interphalangeal/metacarpophalangeal joint dislocations, fractures of the phalanges and metacarpal bones, and traumatic OCD lesions of the capitellum due to hyperextension of the elbow [14, 18]. Other less common injuries may include abdominal (e.g., spleen, kidney) or groin injuries (e.g., male genitals) due to underwater grappling and kicking, cervical spine and head injuries (including concussions) from extreme blows or hitting the head while diving into the pool, and acute knee/hip injuries due to the explosive nature of the eggbeater kick in water polo [9]. Lacerations of the skin may occur due to trauma from fingernails and toenails; however, most water polo matches begin with a "nail check" to ensure proper trimming of nails. If a player is found to have long or sharp nails, especially if it causes injury to another player, he or she may be ejected from the match at the discretion of the referee.

Waterskiing and Wakeboarding

Waterskiing and wakeboarding involve hydroplaning behind an engine-powered marine vessel while being tethered to a tow cable [19]. Both waterskiers and wakeboarders are voluntarily able to pull themselves in and out of the wake of the boat, sometimes to complete acrobatic maneuvers [20]. Due to high speeds achieved, traumatic injuries are not uncommon. The most common body area injured in waterskiing is the hip and lower extremity with most common injury types being sprains/strains (e.g., ACL), fractures, contusions/abrasions, and lacerations (in descending order) [19]. In wakeboarding, the hip and lower extremity is less commonly injured, likely due to having both lower extremities strapped in together simultaneously, while each leg is separated in waterskiing. The most common body site for injury in wakeboarding is the head and neck, with the predominant injuries being lacerations and concussions [19]. Other injuries to the trunk, shoulder, and upper extremity can occur, but are less common. There are case reports of significant injuries in both sports, including spinal cord injuries, significant fractures, cardiac rupture, and intracranial hemorrhage [21–23].

Scuba Diving

Scuba diving is a sport where individuals are exposed to elevated pressures and underwater immersion, which can result in various injuries. Prevalence of overall injury in scuba diving ranges from 7 to 35 injuries per 10,000 divers and from 5 to 152 injuries per 100,000 dives [24]. The most common traumatic injury in scuba diving is barotrauma (i.e., ear or pulmonary), due to changes in pressure. In addition, air trapped under dental fillings or temporary crowns can reexpand during the diver's ascent, causing severe tooth pain [25]. Risk factors for barotrauma injury include loss of buoyancy control, rapid ascent, and repetitive deep diving [24]. There have also been case reports of non-barotraumatic dental injuries (e.g., tooth fractures) due to ill-fitting mouth regulators, and there seems to be a higher risk of dental injury with commercial versus customized mouthguards [26, 27]. Therefore, it is important to have a thorough dental evaluation before scuba diving. Other traumatic injuries in scuba include marine envenomation, "fin foot," and trauma (fractures, lacerations, concussions) related to striking various objects. Fin foot is pain that results after the foot has been constricted for a period of time by the swim fins and then the pressure is released. The resultant hyperemia causes transient swelling, erythema, and pain in the foot [28].

Surfing

Surfing is a popular recreational sport with a high rate of acute injury. Almost one-third of surfers sustain an injury that causes them to seek medical attention, miss work, or take time off of their sport each year [29]. A study of involving surfing-related injuries presenting to US Emergency Departments from 2002 to 2013 shows that acute, traumatic injuries are more common presentations than chronic, overuse injuries [30]. Of these acute traumatic injuries, a high proportion are lacerations (about 40%), with most common locations being the head/face/neck and lower extremities [30, 31]. There is some protection against lacerations from wetsuits, but these are generally only worn in colder water conditions, so use will vary based on location and time of year. Sprains and strains account for the second most common type of traumatic injury in surfing, and these are predominantly in the lower extremities [30]. Fractures are also fairly prevalent (10% of acute injuries) in surfing and usually affect the upper extremity, but also the lower extremity and the head/face/neck [30]. Fractures are usually caused by contact with the surfboard or the seafloor. Surfers should consider using protective surfboards and avoid shallow waters to reduce the risk of fracture. Finger fractures are relatively common and are often related to use of the surf leash. This is caused by grasping the leash while it snaps into tension,

causing hyperflexion of the distal phalanx, which results in trans-phalangeal fracture [32]. Avulsion injuries, open fractures, and partial finger amputations may occur when the leash gets wrapped around a finger [32]. Other less common traumatic surfing injuries include concussions, other closed head injuries, and dislocations (usually shoulder) [30]. Even though there is risk of significant head injury, head gear is rarely used in surfing, likely due to the culture of the sport [33]. Finally, it is important to note that all outdoor aquatic athletes may be at an increased risk for sunburn and subsequently skin cancers, such as melanoma. Therefore, it is important to counsel these athletes on adequate use of water-resistant or waterproof sunscreen.

What Do the Physicians Need to Know When Covering an Aquatic Sport Event?

- Understand and prepare for the specific injury types that your aquatic sport presents.
- Have and practice an emergency action plan for extrication of serious injuries from the aquatic environment, preferably with at least one certified lifeguard on-site.
- Designate a specific area on land (or in an aquatic vessel) for safe evaluation and treatment of injuries.
- Ensure that athletes are wearing the correct protective equipment (e.g., water polo headgear) and have received proper certification/training (e.g., scuba diving) to help prevent injury.

General Rule About Return to Play

- In addition to general return-to-play guidelines for specific injuries, the aquatic environment presents some challenges.
- An athlete wearing a cast will have a difficult time returning to play until it is removed due to risk of water damage to the cast and/or underlying skin.
- Extra precaution should be taken with return to play clearance for injuries that could place the athlete at risk of drowning such as head injuries or cardiopulmonary injuries.

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47

Baseball and Softball

Andrew T. Gomez and Ashwin L. Rao

Key Points

- Collision with other athletes, contact with the ground or surroundings, and contact with balls or bats commonly result in the sport of baseball and softball.
- Blunt chest trauma, often from a ball, may cause commotio cordis and early defibrillation is critical for survival.
- Upper extremity injuries are common in baseball and softball athletes, including ulnar collateral ligament (UCL) injury, superior labral anterior-posterior (SLAP) tear, proximal humeral epiphysiolysis, and biceps tendinopathy.
- Hand, wrist, and finger injuries also commonly occur in baseball and softball athletes.
- Other injuries sustained in baseball and softball include concussion, testicular injury, and various neurologic and vascular injuries.
- Pediatric populations are at risk for head, face, and dental injuries.

Introduction

Baseball and softball are similar sports but subtle differences abound. Both sports involve a batter that attempts to strike, or bat, a pitched ball with ample force in order to sequentially traverse four bases; defense is played by nine fielders distributed across the field in assigned positions. Softball uses a ball with a larger diameter relative to baseball. The two sports also differ in pitching methods (underhand in softball versus overhead in baseball), field and bat dimensions, and length of the game. Both sports are played at a variety of levels, from youth leagues, high school, recreational leagues, NCAA, and professional leagues (Fig. 47.1).

A. T. Gomez
Swedish Providence Medical Group - Mill Creek Sports Medicine, Everett, WA, USA

A. L. Rao (✉)
Department of Family Medicine, University of Washington, Husky Stadium Sports Medicine Center, Seattle, WA, USA
e-mail: ashwin@uw.edu

Injury Epidemiology

Major League Baseball Epidemiology

Major league baseball athletes most often suffer major injuries (athlete placed on disabled list) to the upper extremity (Table 47.1) [1]. Pitchers sustain the most injuries relative to other positions [2]. In general, position players more frequently injure the lower extremity, while pitchers more often injure the upper extremity (Table 47.2) [4]. Overall, shoulder and elbow injuries are the leading two causes of time away from play, followed by chest/back/spine injuries, wrist/hand/finger injuries, lower leg/knee injuries, and upper leg/thigh injuries [2].

NCAA Men's Baseball Epidemiology

The majority of injuries that affect NCAA men's baseball players involve the upper extremity. A substantial fraction of injuries relate to throwing, contact with the ground (Table 47.3), and frequently affect the shoulder. The arm and hand are also commonly injured sites. The lower extremity is often affected as a result of being struck by a ball. The most common injuries include strains, sprains, and contusions (Table 47.4). More injuries occur during games as opposed to practice, possibly the result of increased pressure and intensity of game play. Base running is associated with increased risk of injury relative to other activities [5, 7, 8].

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Fig. 47.1 Baseball

Table 47.1 Professional baseball injury location (disabled list) [1]

Body region	
Shoulder or clavicle	14.7%
Upper leg (thigh)	11.7%
Hand, finger, or thumb	10.1%
Elbow	9.5%
Knee	8.5%
Lower back/sacrum/pelvis	5.6%
Ankle	5.1%
Head/face	5.0%
Lower leg/Achilles tendon	4.6%
Foot/toes	4.3%

Table 47.2 Location of injury sorted by fielders versus pitchers [3]

Fielders		Pitchers	
Hand/wrist	17.0%	Shoulder	30.7%
Hamstring	13.7%	Elbow	26.3%
Knee	12.5%	Back/spine	7.4%
Shoulder	9.5%	Upper extremity, other	5.4%
Foot/ankle	8.8%	Groin	4.4%
Lower extremity, other	8.1%	Hand/wrist	4.2%
Back/spine	7.8%	Knee	3.7%
Core	5.3%	Core	3.5%
Elbow	4.1%	Hamstring	3.3%
Groin	3.0%	Foot/ankle	2.9%
Chest	2.5%	Chest	2.3%
Head	2.1%	Lower extremity, other	1.7%
Hip	1.6%	Hip	1.3%
Hernia, abdomen	1.5%	Illness	1.1%
Upper extremity, other	1.5%	Hernia, abdomen	0.8%
Illness	1.1%	Head	0.6%
Miscellaneous	0.1%	Miscellaneous	0.5%

Table 47.3 NCAA men's baseball and women's softball injury by mechanism frequency [5, 6]

Mechanism	Men's baseball	Women's softball
No apparent contact (non-throwing)	19.0%	13.4%
Throwing (pitching)	15.3%	6.3%
Contact with ground	10.9%	13.6%
Hit by batted ball	9.9%	11.2%
Hit by pitch	9.5%	8.7%
Contact with opposing player	9.0%	18.3%
Contact with fixed base	7.6%	8.7%
Other, unspecified	6.1%	6.5%
Throwing (non-pitching)	5.3%	5.0%
Contact with boundary walls, railing, dugout	2.6%	1.8%
Contact with thrown ball (non-pitch)	2.4%	3.6%
Contact with teammate	2.0%	1.7%
Contact with breakaway base	0.6%	1.1%

Table 47.4 NCAA men's baseball and women's softball positions injured by frequency [5, 6]

Position	Men's baseball	Women's softball
Base runner	23.7%	28.8%
Pitcher	20.9%	10.8%
Batter	15.3%	13.4%
Catcher	7.5%	9.3%
Second baseman	4.9%	5.3%
Center fielder	4.9%	4.5%
Shortstop	4.5%	7.3%
Third baseman	4.4%	4.5%
First baseman	4.1%	4.3%
Left fielder	3.8%	4.3%
Right fielder	3.7%	3.4%

NCAA Women's Softball Epidemiology

The majority of injuries in NCAA women's softball players involves the lower extremity, followed by the upper extremity. A large fraction of injuries result from contact with other players or the ground (Table 47.3). Common lower extremity injuries include ankle sprains, knee injuries, and contusions. Similar to NCAA men's baseball, more injuries occur during games as opposed to practice, likely for the same reasons of increased pressure and intensity of game play. Running the bases is associated with increased risk of injury compared to other activities (Table 47.4) [6, 8, 9] (Fig. 47.2).

High School Men's Baseball Epidemiology

Many of the injuries in high school male baseball players involve the upper extremity (hand, wrist, shoulder), especially sprains and strains (Table 47.5). More severe injuries (requiring surgery) commonly occur at the knee, followed by injuries to the head, face, and shoulder. Injury rates are not significantly different during games versus practice [10–12].

High School Women's Softball Epidemiology

High school female softball players experience injury most frequently in the distal upper extremity and proximal lower extremity; the most prevalent injuries include sprains and strains (Table 47.5). Similar to men's baseball athletes at the same level of competition, more severe injuries (requiring surgery) commonly occur at the knee, followed by injuries to the head, face, and shoulder (Table 47.6). Injury rates are not significantly different during games versus practice [10, 11].

Youth Epidemiology

The majority of youth baseball and softball injuries involve the head and face, followed by the upper extremity, mostly the wrist, hands, and fingers (Table 47.7). In softball, upper extremity injuries were more common, followed by the lower extremity and face. Many injuries result from impact with a ball, either while batting or fielding. Eye injuries are disproportionately high in youth baseball relative to other sports [13, 14].

Table 47.5 High school men's baseball and women's softball type of injury by frequency [10]

Type of injury	Men's baseball	Women's softball
Strains	31.2%	32.2%
General trauma (NOS)	30.7%	27.6%
Sprains	20.6%	23.8%
Fractures	8.8%	8.4%
Musculoskeletal (NOS)	6.6%	3.8%
Neurotrauma	1.7%	3.2%

Fig. 47.2 Softball

Table 47.6 High school men's baseball and women's softball location of injury by frequency [10]

Location of injury	Men's baseball	Women's softball
Forearm/wrist/hand	24.6%	22.9%
Shoulder/arm	19.7%	16.3%
High/thigh/leg	14.5%	18.0%
Ankle/foot	12.5%	14.8%
Knee	10.5%	10.8%
Face/scalp	8.9%	8.0%
Trunk	7.2%	5.5%
Head/neck/spine	1.9%	3.2%
Other, unspecified	0.1%	0.5%

Table 47.7 Youth baseball / softball injury by location, ranked [13]

Location of injury	
Facial (not dental)	21.2%
Hand/finger	21.2%
Knee	13.6%
Ankle/foot	9.1%
Wrist/forearm	7.6%
Facial (dental)	6.1%
Leg (no joint involvement)	4.5%
Elbow	4.5%
Head	4.5%
Shoulder	3.0%
Hip/pelvis	1.5%
Back	1.5%
Chest	1.5%

Commotio Cordis (CC)

- Commotio cordis is blunt, non-penetrating trauma to the chest resulting in arrhythmia, like ventricular fibrillation, often leading to sudden death.
- The pathophysiology likely involves an increase in ventricular pressure activating stretch channels, which can trigger ventricular tachycardia and fibrillation.
- Young baseball and softball athletes who receive a direct blow to the chest from a fast moving ball or bat over the area of the cardiac are at risk of CC as their chest walls are more elastic and more easily compressed. CC is the second highest cause of death in athletes younger than 14 years old.
- Early defibrillation is crucial for survival from CC and, unfortunately, resuscitation is often unsuccessful for a number of reasons, including the lack of availability of automatic external defibrillators (AEDs) [14, 15].

Acute Collision Injuries

- Base runners and catchers are at high risk for injuries related to collision during scoring plays at home plate.
- Common collision injuries include concussions, joint hyperextension and dislocation, and foot and ankle fractures.
- One study that focused on collision injuries to catchers, given their particular risk, found that most injuries occurred at the knee and ankle [4, 16].
- Team physicians should be prepared to evaluate and manage injuries ranging from abrasions and concussions to fractures following acute collision injuries.

Contact Injuries with Objects

- Contact injuries with objects, namely, balls and bats, can cause a variety of traumatic injuries in baseball and softball.
- One study, focused on facial injuries by querying the National Electronic Injury Surveillance System (NEISS) from 2009 to 2013, found that the majority of facial contact injuries resulted from contact by a ball in most cases (70%) followed by contact with a bat (12.5%); lacerations and contusions were the most common result (33.2% and 29.7%, respectively) followed by facial fractures (26.9%) and dental injuries (5.1%).
- The majority of facial fractures involved the nose (60.6%), orbit (16.2%), and mandible (6.1%); young children were more commonly injured by a bat [17].
- In one study involving high school-level athletes, batted balls resulted in contusions, fractures, lacerations, and concussions; the majority of injuries occurred to the head and face, including the mouth and teeth [18].
- Among ED visits from softball injuries, those sustained by contact most commonly affected the head, face, and dentition; being hit by a ball accounted for the majority of soft tissue injuries in this cohort [19].
- To prepare for injuries related to contact with objects, medical personnel should be prepared to care for and stabilize facial injuries, including facial fractures and dental injuries, lacerations, contusions, and other soft tissue injuries.

Sliding Injuries

- Sliding is an integral part of baseball and softball and is an important source of injury; an athlete can slide either head first or feet first, each type generally dictating regions of injury.
- Many documented injuries related to sliding have occurred as a result of sliding, including lacerations, contusions, abrasions, shoulder dislocations, fractures, sprains, and strains [20, 21].

- Evidence conflicts on whether or not head-first or feet-first slides cause more injuries [20, 21].
- For sliding injuries, medical personnel should prepare to care for sprains and strains, contusions, lacerations, abrasions, fractures, and dislocations.

Concussion

- Concussions occur at all levels of play, from youth sport to the major leagues, recreational to competitive, and often result from collisions with other athletes, objects, the ground, or surroundings [14, 22–25].
- Catchers may be at increased risk for concussion given the increased probability of contact with a player running toward home base [22].
- A standardized screening tool, like the SCAT5, should be used to evaluate any player who possibly suffered a concussion.

Shoulder and Upper Extremity Injury

Superior Labrum Anterior-Posterior (SLAP) Lesions

- Labral tears are common in throwing athletes; those affecting the superior aspect of the labrum and involving the biceps tendon can be debilitating to overhead throwers.
- The etiology may involve extreme external rotation experienced by a thrower with resultant increased strain at the biceps anchor.
- Pain is often characterized as vague and often localized to the posterosuperior joint line. Symptoms are worsened with throwing and may involve snapping, locking, or instability. Other findings may include pain at the late cocking phase of the pitch and decreased throwing velocity.
- Comprehensive history and physical examination are important in narrowing the differential diagnosis.
- MRI is the modality of choice for diagnosis.
- Physical therapy is often the first-line approach to management and surgery is reserved for refractory cases [26].

Ulnar Collateral Ligament (UCL) Injury

- The medial UCL in the elbow stabilizes the joint from valgus stress during the throwing motion. Repetitive near-failure tensile stresses at the UCL lead to cumulative microtrauma and microtears, occasionally causing failure of the UCL. Even asymptomatic degeneration of the UCL can lead to acute rupture.
- Athletes with UCL injuries often complain of elbow pain and may exhibit decreased throwing velocity and control.
- A positive valgus stress test, milking maneuver, and moving valgus stress test may be found on exam. Furthermore, signs and symptoms of ulnar nerve injury may be elicited, as concomitant injury is common.
- Depending on the clinical scenario, imaging may include plain radiography, ultrasound, or MRI.
- Physical therapy in addition to other routine conservative management is generally the first-line approach, especially for recreational athletes. Competitive athletes may require surgical intervention [27–31].

Proximal Humeral Epiphysiolysis

- Proximal humeral epiphysiolysis, also known as Little League shoulder, is a common injury among young, skeletally immature throwers that affects the throwing shoulder.
- Pathophysiology involves repetitive torsional overload of the proximal humeral epiphysis and resulting microtrauma when the shoulder is nearly maximally externally rotated.
- Athletes complain of diffuse shoulder pain that worsens with throwing.
- Clinical examination may reveal tenderness at the proximal humerus; swelling, decreased range of motion, and atrophy are uncommon findings.
- Plain films demonstrate widening of the proximal humeral physis in the throwing arm relative to the non-throwing arm.
- Treatment involves rest for 6 weeks after the diagnosis, followed by continued strengthening and rehabilitation. Proper pitching mechanics and preseason strengthening may also benefit the athlete [32–34].

Proximal Biceps Tendinitis

- Overhead throwers are at risk for proximal biceps tendinitis, especially softball pitchers. The proximal biceps tendon is also susceptible to fraying, splitting, and tendon rupture.
- Athletes report pain around the area of the bicipital groove, which worsens when throwing.
- Examination reveals tenderness at the bicipital groove; Yergason's and Speed's test may also be positive.
- Treatment involves physical therapy, cryotherapy, and NSAIDs; return to play involves a graded approach [9, 35].

Wrist, Hand, and Finger Injuries

Hook of Hamate Fracture

- Hook of hamate fractures often occurs as a result of impact from the bat in the lead hand or from a direct impact from a ball.
- Athletes usually report localized pain in the region of the hypothenar eminence and hamate.
- Pain with resisted flexion of the ring and small finger and a painful grip with decreased strength may be found on clinical examination.
- Plain radiography provides inadequate visualization (carpal tunnel view) and CT is often required for diagnosis.
- Acute non-displaced fractures can be managed with cast immobilization, while displaced or non-united fractures generally require surgical intervention; competitive athletes often require surgical intervention due to risk of nonunion and persistent symptoms.
- Complications of a hook of hamate fracture may include ulnar neuropathy, flexor tendon irritation and rupture, ulnar artery thrombosis, and symptomatic nonunion [28, 36].

Annular Flexor Pulley Rupture

- Pitchers are at risk of rupturing the A4 annular pulley of the middle finger.
- Pathophysiology involves an actively flexed middle finger resisting the force of a thrown baseball causing a hyperextension moment at the distal interphalangeal joint.
- The athlete may report feeling a pop during the injury.
- Examination may demonstrate swelling, ecchymosis, and pain that is worsened with resisted flexion of the flexor digitorum profundus (FDP). The finger should be examined for vascular and neurologic integrity.
- MRI confirms the diagnosis, indicating increased tendon-to-bone distance secondary to anterior subluxation of the FDP from P2 of the middle finger; other findings may include edema, effusion, hematoma, or cysts.
- These injuries have been treated successfully with rest, cryotherapy, splinting, NSAIDs, and gradual return to pitching.
- Two weeks of rest is recommended followed by an interval throwing program while protecting the finger. Return to play may occur to normal competition levels in 6–12 weeks, only if range of motion is painless and the pitching arm has been properly rehabilitated [28, 37].

Other Hand Injuries

- Extensor mechanism injury at the distal interphalangeal (DIP) can result in mallet finger. Closed, reducible bony or soft tissue mallet fingers are best managed with splints. Bony mallet fingers with articular involvement or open injuries should be evaluated by a hand surgeon [28, 38].
- The triangular fibrocartilage complex (TFCC) may be injured as a result of forces required to swing a bat. MRI is the optimal imaging study for diagnosis. Conservative management can be effective but surgery may be warranted [36, 39].
- Baseball and softball athletes may also be at risk for scapholunate dissociation. MRI arthrogram aids in diagnosis. Low-grade tears may be managed with splinting and PT, while surgical intervention is required for high-grade scapholunate ligament tears [40, 41].

Lumbar Injuries

- Baseball athletes, particularly elite athletes, may complain of acute-onset lumbar pain.
- Pitching, throwing, running, and hitting involve energy transfer through the core and spine increasing the risk of injury.
- The most common etiologies of lower back pain in elite athletes include muscle strains, spondylolysis, annular tears, disc herniation, stenosis, transverse process fracture, facetogenic pain, and sacroiliac joint arthropathy [42].

Abdominal Oblique Muscle Strains

- Trunk rotation plays a critical role in transmitting power during throwing and batting.
- Abdominal oblique muscle strains, also referred to as side strains, are relatively common and characterized by sudden onset, sharp pain in the side. Symptoms often occur after throwing, swinging, or other twisting movements.
- The majority of injuries occur to the contralateral side to the dominant arm in pitchers and contralateral to the dominant batting side in hitters.
- Treatment involves activity modification, cryotherapy, analgesia, and gradual increase in activity [43].

Knee Injuries

- Among professional players, the most commonly coded diagnoses for knee injuries from 2011 to 2014 included knee contusion (30.5%), other knee injury (15.5%), patellar tendinopathy (10.2%), first-degree MCL sprain (4.7%), lateral meniscal tear (4.5%), and first-degree LCL sprain (2.1%) [1].
- The majority of these injuries did not involve contact; other mechanisms included contact with the ball, striking the ground, and contact with another player [1].
- In another cohort involving baseball players from the high school to professional level, the majority of ACL ruptures occurred while fielding followed by base running; ACL ruptures related to fielding were significantly more common among outfielders relative to infielders or pitchers [44].

Vascular Pathology

Paget–Schroetter Syndrome

- Paget–Schroetter syndrome is venous thrombosis of the deep venous system of the upper extremity that occurs without underlying risk factors (e.g., coagulopathy); patients present with localized pain, which worsens with activity, and occasionally edema.
- Overhead athletes may be at particular risk for Paget–Schroetter as the pathophysiology likely involves anatomic variants at the thoracic outlet, including a hypertrophied anterior scalene or a cervical rib, combined with perivascular fibrosis related to recurrent venous compression or local hypercoagulability related to venous stretching.
- The general treatment paradigm involves two principles: reducing thrombus load to reestablish patency and decompressing the thoracic outlet.
- One study found that recurrence of thromboses was greater in younger patients (≤ 28 years), and they should be offered early surgical decompression [45–48].

Axillary Artery Aneurysm

- A variant of arterial thoracic outlet syndrome (TOS) includes axillary artery aneurysm, and this has been documented as occurring in the throwing arm of overhead throwing athletes.
- Pathophysiology involves vessel injury related to repetitive shoulder abduction with excessive external arm rotation causing intimal hyperplasia, stenosis, and/or thrombus formation; more abrupt and substantial axillary artery injury may result in acute thrombotic occlusion or dissection, while progressive degeneration leads to aneurysm formation.
- Presenting symptoms include the following: throwing arm fatigue, finger numbness, Raynaud syndrome, cold hypersensitivity in the hand or digits, rest pain in the hand, and localized fingertip discoloration.
- Arterial insufficiency may be confirmed with brachial-brachial indices or forearm/digital waveforms.
- Plain films may demonstrate the presence of a cervical rib but provide very little further diagnostic benefit.
- Angiography remains the most important and essential diagnostic study.
- Nonoperative treatment involves PT but often fails. Stenting is not recommended in competitive athletic populations. Surgical intervention is often required [28, 49].

Microvascular Trauma and Digital Ischemia

- Baseball and softball players are exposed to repetitive hand trauma related to catching and batting, increasing the risk of ischemia of the hand and digits characterized by pain, numbness, weakness, cold insensitivity, paresthesias, and cyanosis.
- Catchers have been shown to be at increased risk, possibly related to the repetitive impacts to the hand from high-velocity pitches.
- Clinical examination may exhibit a positive timed Allen test and digital hypertrophy.
- Evaluation includes digital brachial indices and Doppler ultrasound.
- Treatment may include cold avoidance, increased protective equipment, short-term vasodilator therapy, and cessation of all nicotine-containing products [28, 50].

Neurologic Injury

Ulnar Neuropathy

- Ulnar neuropathy is a common injury occurring in throwing athletes.
- Softball pitchers may be at particular risk as they often strike their arm on their hip.
- The ulnar nerve innervates the intrinsic muscles of the hand, flexor carpi ulnaris, and medial aspect of the flexor digitorum profundus. Sensory innervations involve the dorsal and palmar surfaces of the 4th and 5th digits and the ulnar border of the hand.
- Etiologies of ulnar neuropathy include compression at the cubital tunnel, UCL injury, traction from valgus stress, compression from adhesions, osteophytes, and friction due to ulnar nerve subluxation.

- Symptoms include medial elbow pain and occasionally numbness on the ulnar side.
- Examination often demonstrates tenderness of the UCL at its proximal origin of the medial epicondyle, and a positive Tinel's sign may be present at the cubital tunnel.
- Intrinsic muscle weakness of the hand may also be seen; Wartenberg's sign (inability to adduct the 5th digit) and the elbow flexion test may be positive.
- Physical examination should include assessment for concomitant UCL injury.
- Plain radiography is of limited utility but may detect a medial epicondyle fragmentation. MRI may also provide information about the exact cause of neuropathic symptoms.
- Electromyography and nerve conduction (EMG/NCS) studies may be useful in diagnosis.
- Nonoperative treatment involves instruction about proper throwing mechanics to reduce elbow strike on the hip for softball players, avoiding irritation of the ulnar nerve, cryotherapy, and NSAIDs.
- In refractory cases, surgical intervention may be required [9, 51, 52].

Axillary Neuropathy

- The axillary nerve innervates the deltoid and teres minor in addition to providing sensation to the lateral shoulder.
- Axillary neuropathy may occur in baseball or softball players as a result of acute shoulder dislocation or trauma to the shoulder. Quadrilateral space syndrome (QSS) may also cause axillary neuropathy.
- History often involves a recent trauma, or, in the case of QSS, dull aching or burning pain at the posterior shoulder may be reported.
- Physical examination may demonstrate evidence of dislocation or trauma; in QSS, tenderness may be present at the quadrilateral space (QS).
- Plain radiographs should be acquired if trauma occurs. MRI and other advanced imaging modalities may assist in the diagnosis of QSS.
- Appropriately managed shoulder dislocations generally preserve axillary nerve function. If QSS is present, rest, NSAIDs, and rehabilitation generally improve symptoms.
- Surgical intervention may be required in complex dislocation fractures; surgery may also be required for QSS refractory to conservative management [52, 53].

Testicular Injury

- Although rare, impact to the scrotum from a ball or bat may result in testicular injury, even when a cup is worn, including testicular hemorrhage, testicular rupture, or torsion.
- Testicular rupture may cause systemic signs, including nausea and vomiting.
- On examination of the scrotum, the testicles may appear asymmetric or "lay" differently; cremaster reflex may be absent.
- Concern for testicular injury should prompt urgent ultrasound evaluation with Doppler.
- Even with equivocal imaging findings, operative management is generally indicated for rupture or torsion. Surgical exploration to salvage the testicle is generally required within 24–72 hours.
- Orchiectomy is reserved for a nonviable testicle.
- A protective cup may reduce the severity of injury [54–56].

Coverage of Baseball Events

- Team physicians and medical personnel for baseball or softball teams should be familiar with common musculoskeletal injuries, especially those affecting the upper extremity, including UCL injury, SLAP tears, and proximal humeral epiphysiolysis.
- Traumatic injuries may result from contact injuries with other athletes, balls, bats, the ground, or surrounding walls/dugouts and result in minor abrasions to fractures or concussion. Team physicians and medical personnel should be prepared to treat and stabilize a wide array of traumatic injuries.
- Vascular and neurologic injuries occur to baseball and softball athletes, and diagnosis may or may not be clear on the sideline.
- Emergency action plans should account for the possibility of commotio cordis, specifically for rapid AED use following a traumatic chest wall blow. This precaution should be especially observed in youth sport.
- Young athletes are also at increased risk for head, face, and dental injuries, and medical personnel should be prepared for this possibility.

Return to Play

- Musculoskeletal injuries may present acutely from repetitive injury or relate to trauma. In any case, a thorough evaluation should be performed, and the athlete should receive appropriate treatment and rehabilitation prior to returning to play.
- Vascular and neurologic injuries are less common, but athletes with these pathologies should receive appropriate therapy and rehabilitation before returning to play.

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Basketball

48

Deepak S. Patel and Britain O'Connor

Key Points

- Basketball is played by pediatric and adult males and females.
- There is no mandatory personal protective equipment in basketball, although ankle taping/bracing, knee bracing, and use of mouthguards are common.
- Injury rates vary between age groups, level of play, and genders.
- Lower extremity injuries are the most common injury in basketball.
- Minor injuries such as ankle and finger sprains are common.
- Injuries are more likely to occur during game time.
- Female players, especially at the college level, are prone to ACL rupture.

Introduction

Basketball is a limited contact sport consisting of five players on each team. Each team attempts to score by putting the ball in their respective basket, while preventing the opposing players from scoring in their own basket. The game is only stopped for fouls, timeouts, ball leaving the court, and end of time (quarters). Substitutions can be made only when the time clock is stopped. The sport requires extensive running (often explosive sprints) and jumping (Fig. 48.1).

D. S. Patel (✉)
Rush Copley Sports Medicine, Rush Medical College, Yorkville, IL, USA
e-mail: deepak.patel@rushcopley.com

B. O'Connor
Family Medicine Residency, Rush Copley Medical Center, Aurora, IL, USA

Injury Epidemiology

According to the Agency for Healthcare Research and Quality (AHRQ), team sports accounted for 33.7% of emergency department (ED) visits in 2013. Of these visits, 9.6% were sustained while participating in basketball [1]. Primary mechanisms of injury in basketball include contact with other players, contact with objects on/around the court (the ball, the court, standards, rarely the hoop rim), and noncontact [2, 3].

A general trend appears between both collegiate and high school age participants regardless of gender. Injuries occur most frequently during games [2–4]. Lower extremity injuries are the most common injuries associated with the sport, regardless of whether the injury is acute/traumatic or overuse. This is due largely in part to the maneuvers required to play the game, including jumping/landing, rapid changes in direction, lateral motion (cutting, pivoting), and sprinting [3, 5]. Of these lower extremity injuries, ankle and foot are the most commonly injured anatomic locations in basketball across gender and age groups.

Beyond the injuries outlined above, the rates of specific basketball-related injuries vary to a degree between pediatric/adolescent, high school, and collegiate/professional athletes. Gender disparities in injury rates can also be noted. For example, at the collegiate level, male players are more likely than female players to sustain contact injuries. Female college athletes are more likely than male athletes to sustain noncontact as well as overuse injuries [6]. The variability of injury patterns also depends on the setting of the injury (i.e., game time vs. practice).

Pediatric/Adolescent

Sports injuries are more likely to occur in older children as there is a trend toward more practice hours and more intense training as children age [7]. There is a growing trend in

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Fig. 48.1 A National Basketball Association (NBA) game. (Courtesy of Morteza Khodaee, MD, MPH)

young athletes training and participating in sports year-round. Data on rates of specific injuries in pediatric patients is limited, especially in children younger than 12 years of age [7]. Younger players seem to be most susceptible to traumatic injuries such as fractures. Overuse injuries are common at this age. Upper extremity injuries also tend to be more common in younger players. These injury reports, however, are not sports specific [7]. Pediatric patients with injuries at specific body sites are more likely to injure the same site again [8].

Trends in injury rates change once players reach high school level. Students at this level often follow more vigorous training schedules than younger participants. Regardless of gender, players in guard position are more likely to sustain injury [4]. The most common injuries while playing this position are ligamentous injuries, muscle/tendon strains, and foot and ankle injuries. Players in forward and center positions also sustain similar injuries with lesser frequency [4].

Overall, the most commonly reported injuries are ligament strains and muscle/tendon injuries at the high school level [4]. Gender disparities, however, do exist in regard to injury rates and types. Among high school players, female players tend to have more reported injuries than male players. They are more likely to sustain ligament/tendon injuries than male players. Male players reportedly seem to sustain more fractures [4]. While upper extremity injuries are less common in basketball, they do still occur. Mallet finger is often a contact-related injury common in ball sports [9].

Concussions are also common among high school participants in basketball. Female players tend to experience concussions more frequently than male players. Rebound and defense maneuvers seem to be the most common activities leading to concussion [4].

Collegiate Level

Injury trends evolve again once players graduate to collegiate level. The most common mechanism of injury in practice in females was noted to be noncontact, while males tend to have higher injury rates resulting from contact (most commonly with other players) [2, 3].

Lateral ligament compartment sprain of the ankle is the most common lower extremity injury reported by NCAA across all collegiate sports regardless of gender and setting [5]. Basketball has the highest rate of this type of injury, with

game times being the most common setting. Knee injuries follow second in commonality across both genders on both practice and game times. Practice time injuries are similar across genders. After ankle/foot, knee injuries, upper leg, and patellar injuries are common (Table 48.1) [2, 3, 10].

However, game time injury rates diverge between male and female players. Game time injuries most common in male collegiate athletes include upper leg contusions, concussion, and patellar tendon injuries [3]. Practice time injuries most common in male players include hip/pelvis tendon strains, patellar injuries (direct patellar injury and patellar tendon injury), and low back muscle/tendon strains. After knee and ankle injuries, concussion and patellar injuries follow in frequency among female players during games. Muscle/tendon injuries and patellar injuries follow internal knee and ankle injuries in frequency during practice sessions for female players (Table 48.1) [2]. While knee injuries occur frequently in both male and female players, female athletes also tend to have higher rates of ACL rupture than male players [10]. Other common knee problems in female athletes include patellar tendinopathy and meniscal tears.

It should also be noted that concussions occur more frequently in this age group as compared to high school athletes. This is likely due to faster, more aggressive game play at this level [11].

Table 48.1 Most common injury categories in collegiate basketball participants [2, 3, 10]

Male	Practice	Ankle/foot	Ankle sprain
		Knee	Internal derangement
			Patella/patellar tendon
		Thigh/hip	Muscle/tendon strains
		Back	Low back muscle/tendon strains
	Game time	Ankle/foot	Ankle sprain
		Knee	Internal derangement
			Patella/patellar tendon
		Thigh/hip	Contusion
		Head/neck	Concussion
Female	Practice	Ankle/foot	Sprain
		Knee	Internal derangement
			Patella/patellar tendon
		Thigh/hip	Muscle/tendon strain (hamstring, quadriceps)
		Head/neck	Concussion
	Game time	Ankle/foot	Sprain
		Knee	Internal derangement
			Patella/patellar tendon
		Head/neck	Concussion
			Nasal fracture
		Thigh/hip	Contusion

What Is Unique About Basketball Injuries?

- Basketball is a limited contact sport without any specific protective equipment required.
- Injuries can occur from contact with other players or the court. Noncontact injuries can also occur (e.g., ACL rupture and ankle sprain).
- Typically when there is notable blood, the athlete is required to be substituted immediately.
- Lacerations need to be stabilized and secured against reinjury prior to return.
- Lower extremity injuries can affect a player's running and jumping ability and upper extremity injuries can affect ball dribbling, passing, and shooting.

What Do the Physicians Need to Know While Covering a Basketball on the Sideline

- Concussion testing skills/protocols, laceration repair, braces for ankle, knee, elbow, and finger injuries may be required.
- Prepare for dislocation (finger, shoulder, elbow, ankle, patellar) reduction.
- Identify imaging options for fracture evaluation.

General Rule About Return to Play

Consider how the injury can limit the effectiveness of the athlete. Taping and bracing is allowed and may be used to stabilize the injured part for return. To limit deconditioning, encourage cross-training during the recovery from an injury. Consideration of reinjury especially after ankle sprains, ACL rupture, and concussions is essential.

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Boxing

49

Joshua V. Okon, Christine A. Marschilok, and Adam M. Cooper

Key Points

- Boxing is an ancient and unique combat sport.
- Continued competition in the sport remains controversial.
- Injury patterns differ between professional and amateur boxing, given the differences in protective equipment.
- Common injuries include those to the head/neck region and upper extremities.
- The ringside physician plays the role of both health-care provider and health advocate for the athlete.

Introduction

History

Boxing has a long and rich history, dating back to between 1500 and 3000 BC, when few rules existed and combat was bare-fisted. After spreading throughout the Mediterranean, boxing first appeared as an Olympic event in 688 BC where boxers wore headgear and leather hand wrappings for protection. With the collapse of the Roman Empire, the sport largely disappeared until the eighteenth century when it gained popularity in Britain. In 1743, John Broughton, commonly considered the "father of modern boxing," created new rules, further developed by the introduction of the Queensbury Rules in 1867. These rules converted boxing from bare-fisted contest into the timed, gloved sport similar to that which we recognize today [1, 2]. Boxing appeared in the modern Olympic Games in 1904, with women participating in exhibition games in the same year. Despite its long history, boxing as a sport remains controversial. This comes as no surprise given direct blows and therefore infliction of injury upon one's opponent is fundamental to success (Fig. 49.1).

J. V. Okon (✉)
Department of Family and Community Medicine, Sports Medicine Section, Thomas Jefferson University, Philadelphia, PA, USA
e-mail: Joshua.Okon@jefferson.edu

C. A. Marschilok
Thomas Jefferson University, Family & Community Medicine, Philadelphia, PA, USA

A. M. Cooper
Department of Family and Community Medicine, Sports Medicine Section, Thomas Jefferson University Hospital, Philadelphia, PA, USA

Sports Medicine in Boxing

Medical personnel have been an integral part of boxing since its early days. The role of the ringside physician has evolved over time, culminating with the Professional Boxing Safety Act in 1996 mandating on-site physician coverage at each event. Given the combat nature of boxing, the ringside physician has a particularly important role, not only as a medical provider but also as a true advocate for the safety of the athlete.

Professional Versus Amateur Boxing

Amateur and professional boxing differ in areas of protective equipment, timing, refereeing, scoring, and allowed medical interventions.

Amateur boxing is governed by the International Amateur Boxing Association (AIBA). Males and females age 15–40 are eligible for competition as amateur boxers [3]. Professional boxing has no universal governing body. Rules and regulations are specified by individual countries and even differ within one country [4].

Protective Equipment and Injury Prevention

In amateur boxing, the use of certain protective equipment is mandated to ensure the health and well-being of boxers. Ring size and protective padding is standardized. Amateur

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Fig. 49.1 Boxing. (Reprinted with permission by iStock# 591825788)

boxers wear 10 or 12 ounce (283–340 g) padded gloves (depending on weight category); use hand wraps of specified length (2.5 m) and width (5.7 cm); must dress in light boots, shorts, and singlet/top; use a cup protector (males); and use a form-fitted gumshield. Female boxers may wear a form-fitted breast protector [3]. All amateur boxers also wore protective headguards up until 2013 when the AIBA removed headguards for elite amateur boxers citing internal evidence that it would reduce the rate of concussion in this groups [3]. The vast majority of studies on injury patterns in boxers is prior to this change. It was written as of 2018, the AIBA plans to ban headguards for all amateur boxers [5].

In contrast, professional boxing ring size varies depending on location, the use of headguards and wearing of singlets/tops is prohibited, and glove size depends on the jurisdiction.

Injury Epidemiology

Boxing is often considered a dangerous sport with high injury rates. High-quality prospective research on the epidemiology of boxing injuries is limited and studies vary greatly in reported injury statistics.

Injury risk is much higher during competition for both professional and amateur boxers (Table 49.1). During training, more protective hand wrappings and heavier gloves may be utilized; punches are thrown in a more controlled setting; sparring is less intense than competition; and many punches are "pulled" (i.e., not making full contact) [6]. Despite this, training hours vastly outnumber those of competition, so both settings contribute significantly to injuries with 42.9–65% of injuries occurring during training [7, 8].

Table 49.1 Injury rates in competition and training between amateur and professional boxers [9–12]

	Training (per 1000 hours)	Competition (per 1000 hours)
Amateur	0.5	920–1221.4
Professional	0.7–1.8	250.6–1081

Table 49.2 Injury patterns in amateur and professional boxing [6]

	Amateur (%)	Professional (%)
Head/neck	9–75	74–96
Upper extremity	14–55	0–22
Lower extremity	4–24	0–2
Trunk/other	0–16	2–5

The overall injury rate for combined amateur and professional boxing in both training and competition has been reported as 2.0 per 1000 hours [11].

Injury patterns differ between professional and amateur boxing, likely due to differences in protective equipment (e.g., headguard prior to 2013) and stricter refereeing in amateur boxing (Table 49.2). Studies show a wide range of injury patterns likely due to vast differences in methods and injury definitions. Overall, there is a trend toward higher rates of head/neck injuries in professional boxers, with very few upper extremity, lower extremity, or trunk injuries. In contrast, while head/neck injuries still predominated in amateur boxers, there was a higher proportion of upper and lower extremity injuries as compared to professional boxers [6]. Again, this is thought to be due to headguard use in amateur boxing prior to 2013, stricter refereeing in amateur boxing, and more liberal handwrapping allowed in professional boxing [9].

Head/Neck Injuries

The most common injuries within the head/neck region include facial and scalp lacerations and contusions, and concussions. Facial/scalp lacerations and contusions are the most common head/neck injuries, although rates vary widely between studies – 7–93% in amateur boxers and 12–96% in professional [6, 10]. These injuries are traditionally more common in professional boxing than in amateur boxing secondary to longtime use of headguards in amateur boxing [8, 9, 12]. This appears to be changing with the aforementioned removal of headguards in amateur boxing [6, 9].

The second most common head/neck injury in boxing is concussion. Reported rates of concussion vary in the literature (12–52% of total injuries in prospective studies) [9]. Studies suggest differences in concussion criteria/diagnosis as contributing greatly to the variability in rates [6]. Concussions occur more often in competition than in training. The removal of headguards from amateur boxing was done to hopefully decrease rates of concussion, the theory being that removal of protective headguards will decrease the severity with which opponents throw punches and therefore reduce the force transmitted to the brain. This is reportedly based on internal evidence within the AIBA [5]. Further research is needed to follow concussion rates.

Less-frequent injuries in the head/neck region include periorbital hematoma, tympanic membrane rupture, epistaxis, nasal bone fractures, retinal detachment, intracerebral hematoma, epidural hematoma, and subdural hematoma.

There have been case reports of death in boxing, thought likely due to vertebral artery dissection. These are exceedingly rare, such that prospective epidemiologic studies of injury risks have failed to capture these.

Upper Extremity Injuries

The majority of upper extremity boxing injuries occur at the hand (11–53%) and wrist (19–49%) according to prospective studies [6]. The types of upper extremity injuries do not appear to differ significantly between amateur and professional boxing.

One of the most common acute injuries is "boxer's knuckle," which is a traumatic injury to any of the second through fifth MCP joint capsules with injury to the extensor apparatus, comprising up to a third of upper extremity amateur boxing injuries [12]. The third MCP joint is most at risk for this injury, given its prominence in a clenched fist. Symptoms include arthralgia, swelling, limitation in range of motion at the MCP joint, and tendon subluxation. Boxer's knuckle can be career threatening if not properly diagnosed. Management depends on injury severity, degree of instability, and athlete level [13]. Conservative management involves buddy taping or splinting in extension, often employed for injuries without instability [13]. The use of intra-articular corticosteroid injections for this injury has not been studied in the literature. Surgical management is often required for injuries with instability, those that have failed conservative management, and high-level athletes for whom quick return to play is necessary [13].

Tearing of the ulnar collateral ligament (UCL) of the MCP joint of the thumb ("skier's thumb") comprises 13–30% of upper extremity injuries [6]. In boxing gloves, the thumb lies adjacent to the PIP joint of the second digit, which is a particularly vulnerable position for adduction injury [14]. Skier's thumb often presents with pain at the ulnar aspect of the first MCP joint and laxity with valgus stress at the joint.

Fractures account for 9–47% of upper extremity injures in boxing [6]. The classic "boxer's fracture," (transverse fracture of the neck of the fifth metacarpal); Bennet's fracture (fracture of the base of the first metacarpal bone with extension in the CMC joint); other metacarpal fractures, and proximal phalangeal fractures of the fingers are most common [8, 9, 12].

Other upper extremity injuries in boxing include contusions; wrist, elbow, or shoulder sprains; shoulder impingement; and biceps muscle or tendon tears [8, 9].

Lower Extremity and Trunk Injuries

Acute lower extremity and trunk injuries in boxing are uncommon. The most common lower extremity injuries include thigh tears, adductor tears, meniscal or ligamentous tears of the knee, ankle sprains, and lower extremity contusions [8, 12]. Fractures are rare [8].

The most common trunk injuries are rib contusions [8, 9]. There is one report of pneumothorax due to boxing [15]. Few reports of splenic rupture in boxing exist, though those have been reported in the setting of infectious mononucleosis [16, 17]. These are rare, so not well documented in epidemiologic studies of boxing injuries.

What Is Unique About Boxing Injuries?

- Unlike most sports, the goal of a boxer is to inflict pain, bodily harm, head blows on their opponent.
- Blows to the head and bodily fluid exposure are expected during each bout.
- Any knock-out and technical knock-out incurs an automatic medical suspension of at least 1 month.
- Unlike most other sports, physicians have the authority to stop a fight.

- Women must sign disclaimer stating they are not pregnant to the best of their knowledge [18].
- Boxers require pre- and post-fight physicals.

What Physicians Need to Know About Boxing?

The role of the ringside physician is different from that of other sports. He or she is responsible not only for the health of the athlete but also for the safety of the athlete during competition. This involves evaluations both before and after the fight, as well as care during the fight. The ringside physician should possess a valid state license for the state in which the fight occurs, and must possess experience and abilities in high acuity triage and trauma assessment. He or she must have a solid fund of orthopedic knowledge, head injury/concussion assessment experience, and airway management skills. The physician can stop a fight beforehand based on the pre-fight physical or during the fight at any time by mounting the ring apron.

The ringside physician must continuously follow the action of a bout. If called into the ring by the referee, a physician approaches the ring at the neutral corner and attends to the injured boxer. The physician should be on high alert for cervical spine injuries. Common injuries for which the ringside physician is summoned include lacerations, nosebleed, head injuries/disorientation, or joint injuries. Physicians need to know which injuries would disqualify fighters from competition and act accordingly, forcefully if necessary (Tables 49.3 and 49.4) [18].

Covering physicians should have certain medications readily available ringside. These would include albuterol, epinephrine 1 mg/ml, morphine, IV diazepam or buccal midazolam (for seizure activity), thrombin and avitene (to accelerate clot formation), and an anti-emetic [18].

Physicians covering boxing matches should have their emergency action plans and protocols in place at the time of the event. Having established relationships with local hospitals and appropriate providers is recommended (Table 49.5).

Cutman

Cutmen play an important role for boxers. They are tasked with controlling and stopping bleeding within 1 minute from nosebleeds or lacerations that would otherwise disqualify the boxer. Use of medications in controlling bleeding is barred in amateur boxing, but any topical treatment is allowed in professional boxing. Petroleum jelly is often applied pre-bout in attempts to prevent lacerations. An enswell, a smooth metal instrument often chilled, is used to control facial swelling or hematomas. In professional boxing, various topical medications, often mixed with petroleum jelly, such as avitene, epi-

Table 49.3 AIBA Medical handbook- disqualifying conditions prior to competition [18]

Severe chronic infections
Severe blood dyscrasias (e.g., sickle cell disease)
Hepatitis B/C, or HIV infection
Refractive and intraocular surgery, cataract, retinal detachment
Myopia of more than −5 diopters
Recorded visual acuity in each of: Uncorrected worse than 20/200 Corrected worse than 20/50
Exposed open infected skin lesions
Significant congenital or acquired cardiovascular, pulmonary, or musculoskeletal deficiencies or abnormalities
Unresolved post-concussion symptoms, which will need clearance from a neurologist
Significant psychiatric disturbances or drug abuse
Significant congenital or acquired intracranial mass lesions or bleeding
Any seizure activity within the last 3 years
Hepatomegaly, splenomegaly, ascites
Uncontrolled diabetes mellitus or uncontrolled thyroid disease
Pregnancy
Any implantable device that can alter any physiologic process
Women's breast protector, which protects legitimate scoring areas beside the breast

Table 49.4 AIBA disqualifying injuries during competition [18]

Excessive swelling of face or eyes that impair vision
Deep cuts (subcutaneous tissue visible) in the Inverted Bell Zone[a]
Suspected/proven fractures of nose, face, or metacarpals
Presence/history of retinal detachment
Lacerations or wounds requiring dressings for control of bleeding
Nose bleeds complicated by arterial bleed, septal hematoma, facial fracture, or excessive bleeding
Concussion
Knockout

[a]Zone including important facial structures of eyes, lacrimal ducts, nose, lips, mouth, naso-ethmoidal bones

Table 49.5 Recommended medical equipment: ringside and medical room [18]

Ringside	Medical room
Penlight	Venous cannulas
Oral/nasal airways	Infusion sets
Gloves	Wound cleansing equipment
Blood pressure cuff	Wound glue
Petroleum jelly	Absorbable/nonabsorbable sutures
Sponges	Suturing instruments
Adhesive tape	
Stethoscope	
Stretcher	
Oxygen tank	
Cervical collar	
Defibrillator	
Clean towels	
Two buckets, one with ice and one empty	
Ice and ice bags	
Scissors/trauma shears	

nephrine, thrombin, or surgical glues are used. Cutmen commonly treat aforementioned injuries prior to involvement of the ringside physician.

General Rule About Return to Play

Return to play for boxing is clearly injury specific. Following a knockout or a technical knockout, the boxer will incur a mandatory medical suspension for at least 1 month, maybe longer depending on whether there is loss of consciousness. No specific protocols are currently in place for most injuries. The physician and athlete should keep in mind the extensive demands on the body with repeated physical contact associated with boxing, and they should wait until recovered and able to withstand the repeated trauma. Whenever possible, physicians should follow standard guidelines from governing bodies, such as the NCAA, where appropriate.

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Cheerleading

50

Stephanie Chu

Key Points

- Cheerleading is the most dangerous female sport when looking at the number of catastrophic injuries.
- Direct catastrophic injuries in cheerleading include closed-head injury, skull fractures, and cervical spine injuries resulting in permanent brain injury, paralysis, or death.
- The most common musculoskeletal sprains and strains in cheerleading are in the wrist and ankle.
- The most common mechanisms of injuries are basing/spotting, tumbling, and falls from heights.
- Concussions and closed-head injuries primarily occur from stunting (primarily flyers) and pyramid stunts and are responsible for the majority of head/neck injuries.

Introduction

Cheerleading popularity and participation has increased from 600,000 to 3.6 million from 1990 to 2003 in the US in ages 6 years and older. In addition to the increased participation, cheerleading as a sport has changed dramatically since its origination in the late 1800s, when the sport was primarily to lead a crowd in cheering on sports teams using pompoms, clapping, splits, and jumps (Fig. 50.1). In the past century, the sport has evolved into a competitive sport that is year-round, where cheerleaders execute a routine that is timed and performed within a certain area consisting of tumbling, dance, jumps, stunting, and pyramids [1]. Most high schools and colleges have cheerleaders; however, only 29 state high school athletic associations recognize cheerleading as a sport and only three colleges consider cheerleading a sport. Since it is not an NCAA championship sport, there is no information available as to whether or not the other colleges consider it a sport as it is usually not under the umbrella of athletics [2, 3]. With this growth in participation, the physically demanding routines, and the lack of governance come increased injuries presenting to clinicians.

Injury Epidemiology

The overall injury rate in cheerleading across all age groups is 1.0 per 1000 athletic exposures. An athletic exposure is defined as an athlete's participation per practice or competition. College cheerleaders have the highest injury rate (2.4) and middle school and recreational cheerleading the lowest (0.5) (Table 50.1) [1]. Most acute injuries sustained in cheerleading can be categorized into catastrophic injuries, sprains and strains (primarily to the ankle and wrist), concussion, abrasion/contusions/hematomas, fractures, and laceration or puncture (Table 50.2) [4]. Cheerleading is included in the National Center for Catastrophic Sports Injury Research (NCCSIR) data collection system for high school and collegiate sport. Considering the number of catastrophic injuries from the data, cheerleading seems to be the most dangerous female sport [2].

Catastrophic Injuries

Catastrophic injuries can be classified into two categories, direct or indirect, where direct is trauma related to participating in the skills of the sport and indirect resulting from body system failure resulting from exertion while participating in a sport (Table 50.3 and Table 50.4). The overall risk of catastrophic injury in cheerleading is lower than in other sports; however, the risk of *direct* catastrophic injury is considerably

S. Chu (✉)
Department of Family Medicine and Orthopedics, Division of Sports Medicine, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: stephanie.chu@cuanschutz.edu

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Fig. 50.1 Cheerleading in an NBA game

Table 50.1 Comparison of the overall rates of injury among cheerleading levels [1]

Cheerleading level	Injury rates per 1000 athletic exposures
College	2.4
All-star	0.8
High school	0.9
Middle school	0.5
Elementary school	1.5
Recreational	0.5

Table 50.2 Estimated incidence of types of injuries in cheerleading [4]

Type of injury or illness	Incidence of all cheerleading injuries (%)
Sprains and strains	53
Abrasions/contusions/hematomas	13–18
Fractures/dislocations	10–16
Lacerations/punctures	4
Concussion/head injuries	3.5–4

higher. From 1982 to 2009, cheerleading accounted for 65.0% of all high school direct catastrophic injuries in girls and 70.8% in college [1].

Table 50.3 Direct and indirect injuries in cheerleading [1, 5, 6, 7]

Traumatic	Closed-head injury
	Subdural versus epidural intraparenchymal hemorrhage
	Concussion
	Skull fracture
	Cervical spine injuries
	Dislocation
	Fracture
	Nerve injury
	Spinal contusions
	Major ligament injuries
Nontraumatic	Cardiac collapse
	Severe dietary restriction
	Heat stroke

What Is Unique to Cheerleading Injuries?

- As in other sports, cheerleading does not have a "game" equivalent. Cheerleaders "perform" at pep rallies, athletic events, and cheerleading competitions with each performance being a unique routine. Each routine is also performed on various surfaces (natural grass, artificial turf, wood gym floor, concrete or vinyl tile floor, mat on foam floor, spring floor), which can affect injury pattern [1, 3].

Table 50.4 Most common injuries sustained in cheerleading [4, 5, 8–10]

Sprains and strains	Ankle ligamentous injury
	Neck strain/sprain
	Low back strain
	Knee injuries
	  Ligamentous injuries (ACL, MCL)
	  Meniscus injuries
	Wrist strain/sprain

Dermatologic/soft tissue	Contusions Lacerations Hematomas Puncture wounds
Fractures/dislocations	Shoulder dislocations/subluxation Wrist/forearm fractures (physeal fractures) Finger fractures Patellar dislocations
Head injuries	Concussion

- Cheerleading coaching and associations have not kept up with the skill level and safety of the sport. Only in the past 10 years have cheerleading coaching certification programs become available. In many instances, coaches who do not have the expertise to teach the current skills in the sport are the cause of safety issues seen within cheerleading [2].
- Higher BMI can be a risk factor for cheerleading injuries [1].
- Despite catastrophic injury and concussion being of heightened concern in cheerleading injuries, the organizations that control cheerleading do not take an active role in safety [2].
- Concussions sustained in cheerleading cannot always be removed from "play," given the short nature of cheerleading competition routines. Unless the concussion is accompanied by loss of consciousness, the athlete often will complete the entire performance.

What Do Physicians Need to Know While Covering a Cheerleading Event?

- A cheerleading "event" is not only during a cheerleading competition but can also be at a pep rally, basketball, football, volleyball, soccer, and even ice hockey game.
- Pyramids that are over two people high should not be performed with mats and proper spotting.
- An emergency action plan should be in writing and available to everyone involved including athletes and coaches.
- Cheerleaders who show signs of concussion should be removed immediately and obtain medical attention. Coaching staff and athletes should be educated on signs and symptoms of concussion [2].

General Rule About Return to Play

- Return to cheerleading after a concussion may need practice of stunts and tumbling in a pool to assess for return of symptoms prior to returning to full competition on a variety of surfaces in which recurrent injury could be sustained.
- Any athlete with a suspected acute fracture or concussion should be held from cheerleading.
- Taping and bracing of ankle and wrist injuries can aid in return to play for cheerleading during an event in which they are cheering at another sporting event; however, it is not typically done during a cheerleading competition routine.

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CrossFit

51

Armando F. Vidal

Key Points

- CrossFit is a popular fitness program and is performed by athletes of all ages and fitness levels
- Injury rates are poorly defined but appear to be consistent with other common forms of exercise
- Shoulder, back, and knee injuries are the most commonly seen and are typically acute and mild
- Preexisting injuries can predispose an athlete to reinjury
- Skin injuries are common and often underreported
- Rhabdomyolysis is rare, but the same consideration should be given by providers as with any other at-risk sport

Introduction

Since its introduction in 2000, CrossFit has experienced exponential growth nationally and internationally. CrossFit defines itself as "constantly varied functional movements performed at high intensity" [1]. With over 13,000 affiliates worldwide and hundreds of thousands of athletes, it is quickly becoming a very common exercise program for athletes of all ages (Fig. 51.1).

As a strength and conditioning program, CrossFit incorporates a wide variety of exercises including running, biking, rowing, Olympic lifting (snatch, clean and jerk), power lifting (squat, deadlift and bench press), kettle bell training, and gymnastics (pull-ups, muscle-ups, handstand pushups, toes to bar, etc.). These exercises are combined in a myriad of different permutations to generate a high-intensity workout, where these movements are done in rapid succession and are scored for either time or repetitions. The competitive nature of CrossFit and the passionate enthusiasm that its athletes express have historically given CrossFit a bad reputation for creating a disproportionately high number of injuries.

Epidemiology

Injury rates from CrossFit are difficult to definitively establish because of a lack of well-controlled studies. Media reports of rhabdomyolysis and severe injuries have amplified the perception that CrossFit is a dangerous sport with high rates of injury [2, 3]. Several retrospective, survey-based studies have been performed but they lack any control and have the potential for recall and sampling bias.

In general, reported injury rates from CrossFit are consistent or lower than many traditional fitness activities such as distance running, weightlifting, track and field, and other field and court sports [4–9]. Reported injury rates range from around 2.3 to 3.1 injuries per 1000 h of participation [4, 6]. Published, online surveys of CrossFit athletes have documented gross injury rates between 19.4% and 73.5% for responders [4, 9]. However, when the criteria is more tightly defined as missing at least 1 week of training, the rates are closer to the 20–30% range [7, 9]. Most injuries are minor and self-limiting and are frequently an exacerbation of a preexisting condition [8, 9].

Shoulder injuries are consistently the most commonly reported injuries [4, 6, 8, 9]. Most athletes describe general inflammation/pain and strains as the most common complaints. Ruptures and dislocations can and do occur but are relatively rare [9]. Summitt et al. sought to specifically examine the rate and epidemiological characteristics of shoulder injury among CrossFit athletes [8]. They surveyed 187 athletes and revealed a 23.5% shoulder injury rate that at a minimum required total removal from training for more than a week. Gymnastics and weightlifting movements equally accounted as the primary cause for injury. Many of these injuries were

A. F. Vidal (✉)
The Steadman Clinic, Steadman Philippon Research Institute, Vail, CO, USA
e-mail: avidal@thesteadmanclinic.com

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Fig. 51.1 Athletes performing CrossFit in a gymnasium. (Courtesy of Armando Vidal)

exacerbations of previous shoulder complaints prior to starting CrossFit. They did not report diagnosis or injury severity, but most injuries resolved within 2 months and only one required surgery. This was consistent with the findings of Weisenthal et al. who reported that "rupture" and "dislocation" only represented 6.2% of the shoulder injuries that they observed [9].

Lumbar spine and knee injuries are also commonly reported in this athletic population [6, 7, 9]. Like the shoulder, most of these are mild and self-limited. Lumbar spine injuries appear to be linked more closely to powerlifting (deadlift and squat) in this population rather than to gymnastics [9].

Despite the intense media attention that has been given to CrossFit and rhabdomyolysis, there is no evidence that there exists an elevated risk with this activity over any other high-intensity fitness program [4, 10]. Although, creatine kinase (CK) elevation is relatively common in CrossFit, it is very rare for these athletes to develop acute kidney injury and full rhabdomyolysis [11]. In fact, despite media reports and rare case reports [2, 3, 12], the incidence of rhabdomyolysis appears to be very rare and less common than in other fitness activities such as football, marathon training, and military fitness [10, 13]. It should be noted that risk factors for rhabdomyolysis such as low fitness level, age, hydration, temperature, and humidity can exist during CrossFit training and/or competitions, and as such, this should be considered by any sports medicine specialist covering these events.

Lastly, although underreported, the prevalence of skin compromise, specifically hand and tibial lacerations, with CrossFit is very high. This is not captured well in the literature as it often is not reported as an injury, as it rarely impacts the training schedule of an athlete. "Hand Rips" from high-frequency pullups or muscle-ups often occur through previously callused hands. Additionally, pre-tibial abrasions and lacerations can occur from missing a landing on a box jump or from direct trauma of the knurl of a barbell along the tibia during Olympic lifts. Although athletes are typically well versed in the management of these injuries, the presence of blood on weightlifting and gymnas-

tic equipment creates a hazard and should be addressed. Additionally, rope climbs can create rope burns along the legs of participants. Acutely, these typically do not require medical attention, but they can easily become infected and should be treated appropriately.

Injury Prevention

Avoiding injury with sports such as football and soccer is difficult given the traumatic nature of those activities. Injury prevention and counseling with CrossFit is easier given the more predictable nature of the sport. In general, injury rates appear to be lower in athletes who undergo formal beginner or "on ramp" programs and in those who have better supervision [5, 9]. Inappropriate form and technique is often cited by athletes as a common cause of injury and this should be considered [8]. Experience level does not appear to be a major consideration, as experienced athletes seem to get injured at the same rate as beginner athletes.

Additionally, aggravation of preexisting injuries represents between 20% and 40% of the self-reported injuries by athletes [8, 9]. Educating patients about which movements are most likely to aggravate their preexisting condition can be very useful. For instance, a patient with a preexisting shoulder condition should be counseled that gymnastics and overhead lifting are more prone to injure shoulders, and as such, they should focus on form and technique before applying heavy loads or technical movements.

What Is Unique About CrossFit Injuries?

- Most CrossFit injuries are acute and mild with limited time lost from training or other athletic activities.
- Shoulder injuries are the most common and usually result from gymnastic movements or weightlifting.
- Lumbar spine issues are more common in powerlifting (deadlift and squat).
- Lacerations and skin abrasions are common during training and competition. "Hand rips" from gymnastic movements and shin lacerations from missing box jumps and occasionally from Olympic lifting are common and often need medical attention.
- Rope burns from rope climbing can create significant abrasions on the lower extremity. These can readily become infected and should be monitored for infection.
- Although the elevation of creatine kinase is very common, the actual rhabdomyolysis is extremely rare.

What Do Physicians Need to Know About Covering a CrossFit Event?

- Prepare for basic first aid for skin wounds and lacerations.
- Overuse injuries are common, and athletes need to be counseled about avoidance of activity if they cannot safely participate.
- Rarely, acute fractures/dislocations can occur, and reduction or splinting may be necessary.

General Return-to-Play Guidelines

- CrossFitters are a passionate and motivated group of athletes. They generally do not like to be told that absolute abstinence from their sport is necessary for recovery. CrossFit is scalable and many movements are modifiable. Finding reasonable accommodations is typically easy to accomplish.

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Cycling

52

Jonathan T. Bravman and Robin H. Dunn

Key Points

- Cycling injuries can be broadly categorized as acute traumatic and chronic overuse.
- Traumatic injuries usually occur as the result of a fall or a collision with another cyclist or a motor vehicle.
- Superficial abrasions/contusions are the most common traumatic injuries sustained by cyclists, followed by fractures of the clavicle, wrist, forearm, and olecranon.
- Chronic overuse injuries usually result from poor positioning on the bicycle or contact pressures at the three athlete–bicycle interfaces: handlebar, pedal, and saddle.
- Assessment of bicycle fit is a key skill for the team physician.
- Knee pain is a common complaint by cyclists, and the specific location about the knee can indicate the diagnosis and etiology.

Introduction

Competitive cycling and mountain biking have increased in popularity in recent years among recreational and competitive athletes of a wide range of ages in the United States and abroad (Fig. 52.1). Despite significant improvements in bicycle design, safety equipment, and training regimens, cyclists and mountain bikers remain at significant risk for both traumatic and overuse injuries. In order to effectively assess and treat an injured cyclist or mountain biker, the clinician should be aware of the most common types of injuries and should have a working knowledge of bicycle design and fitting principles.

The initial evaluation of an injured cyclist should consist of a thorough history, a focused physical examination, and – especially with chronic injuries – a static and dynamic assessment of bike fit [1]. In assessing an acute injury, the initial timing and mechanism of injury should be noted, along with any associated symptoms. For chronic injuries, a more extensive history of the onset, character, and frequency of symptoms is often necessary, including aggravating and relieving factors as well as treatment modalities tried thus far. The history should also include details regarding the athlete's training volume, intensity, duration, and frequency. The exam should focus on the area most symptomatic and should include inspection, palpation, range of motion testing, neurovascular assessment, and special tests – comparing the injured and uninjured sides. The injured area should also be assessed with regards to overall limb alignment, strength, and flexibility. Finally, the clinician should assess the athlete's static and dynamic position on the bicycle, paying attention to the errors in bike fit that can contribute to overuse injuries [2].

Injury Epidemiology

Epidemiologic studies of cycling injuries vary substantially with regards to injury incidence, pattern, and severity. This variation is likely due to differences in study populations, methodologies, and injury categorization [3]. In the general population of recreational cyclists, injuries – like many other types of trauma – exhibit a bimodal age distribution, with peaks in the second and seventh decades. Regardless of age, men are disproportionately more likely than women to sustain traumatic injuries [4, 5].

J. T. Bravman (✉)
Department of Orthopedics, Division of Sports Medicine and Shoulder Surgery, University of Colorado School of Medicine, Denver, CO, USA
e-mail: jonathan.bravman@cuanschutz.edu

R. H. Dunn
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: robin.dunn@cuanschutz.edu

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Fig. 52.1 Leadville 100 is a popular mountain bike race in Leadville, Colorado. (Courtesy of Morteza Khodaee)

Injury mechanism and type are largely dependent upon the intensity and level of competition. Recreational cyclists who ride occasionally and for short durations have less overall exposure to threats of traumatic injury and less repetitive loading of musculoskeletal structures than competitive cyclists who spend dozens of hours on the bike and accumulate hundreds of kilometers of training per week.

There are few studies in the literature focusing on the incidence of traumatic injuries in competitive cyclists. A 1997 study by Barrios et al. reported the incidence of traumatic and overuse injuries in a cohort of 65 professional Italian cyclists over a 12-year period from 1983 to 1995 [6]. Fifty-six (86%) of the 65 athletes reported traumatic injuries, including 21 fractures, and 37 reported overuse injuries. Only nine (14%) of the cyclists reported being free of injury during the study period. On average, each cyclist sustained a traumatic injury every other year [6]. In general, traumatic injuries are the results of falls, which occur most commonly because of rider error, irregularity in terrain, collision with another cyclist, or collision with a motor vehicle [7].

In 2012, de Bernardo et al. catalogued the traumatic and overuse injuries sustained by a group of 51 elite-level road cyclists over the course of a 4-year period [8]. Forty-three (84%) of the 51 athletes sustained a total of 103 cycling-related injuries during the period, with roughly half being traumatic and half being overuse [8]. Traumatic injuries most commonly affected the shoulder girdle (34%) and the lower extremity (26%). The most common traumatic injuries were lacerations/abrasions (20%) and fractures of the clavicle (22%), wrist (8%), ribs (8%), olecranon (6%), and facial bones (6%). Overuse injuries involved knee pain (32.1%) and lower extremity muscle contracture/tightness (26.4%) [9].

In 2016, Decock et al. reported the details of 777 acute traumatic injuries occurring among competitive cyclists of various levels in Flanders in the calendar year 2012 [7]. By far, the most common cause of injury was collision with other riders (52.2%). The most common types of injuries were abrasions/lacerations (58.7%), contusions/hematomas (50.5%), and fractures (26.6%). Of the fractures, the most common locations involved the shoulder girdle (30.3%), the hand/wrist (29.4%), and the forearm (23.5%) [7].

In general, traumatic injuries tend to affect the upper extremities while chronic overuse injuries tend to occur in the lower extremities [3]. The following sections will elaborate some of the most common acute and chronic injuries sustained by cyclists.

Acute Traumatic Injuries

Abrasions/Contusions

Abrasions and contusions, usually of the extremities, are the most common injuries sustained by cyclists that require medical attention [7, 10]. The severity of these injuries can range from relatively minor abrasion (road rash) to severe degloving injury (Morel-Lavallée Lesion) requiring surgical debridement (see Chap. 40).

Head Injuries

The main risk factors for head injury during cycling are lack of helmet, collisions with motor vehicles, and male gender [11]. Despite the mandatory use of helmets in cycling, concussions remain a common injury sustained by competitive cyclists. They usually are the result of direct contact between the athlete's helmeted head and the road. Head injuries are cited as the most common severe injury warranting hospital admission. In the review by Bostrom and Nillson, 46% of patients requiring hospital admission after bicycle accident were admitted for closed head injuries, including concussion, cerebral contusion, intracranial hemorrhage, or skull fractures [5].

Fractures

The reported incidence of fractures resulting from falls and crashes in cycling is heterogeneous. The most common locations of fracture include the clavicle, wrist, forearm, elbow, ribs, hip, pelvis/acetabulum, and tibia/fibula. In the report by Barrios et al., fractures of the upper extremity (71%) were more common than those of the lower extremity (29%) in their cohort of elite cyclists, with clavicle fractures accounting for 43% of all fractures [6]. In the review by Bostrom and Nillson, however, 20% of all patients admitted to the hospital after a bicycle accident had lower extremity fractures (hip and tibia/fibula), while only 10% had upper extremity fractures [5]. The latter study likely overstates the incidence of lower extremity fractures because isolated upper extremity fractures are less likely to require hospital admission. In a 2003 review of bicycle injuries, Rosenkranz and Sheridan found that of 13,485 patients who presented to their Level 1 trauma center in Massachusetts after a bicycle collision, 174 presented with a fracture. The most common locations were, in order of decreasing frequency, vertebral, femur, forearm, clavicle, tibia/fibula, pelvic, ankle, humerus, hand/wrist, scapula, and foot [12].

The location of upper extremity fractures results from the manner in which the athlete attempts to break their fall. For instance, reaching with outstretched hands to brace oneself can cause carpal, distal radius, forearm, and elbow fractures. Attempting to roll onto the ground can cause a direct blow to the lateral shoulder, resulting in clavicle fractures.

Throacoabdominal Injuries

Injuries to the torso can result from direct contact with the handlebars or stem or as a result of high-energy contact with motor vehicles or the ground. Blunt abdominal injury can cause lacerations of the spleen or liver as well as hollow viscus rupture. A blow to the chest wall can result in rib fractures, flail chest, and pneumothorax. Of note, these injuries are more common in mountain bikers than road cyclists [10].

Nontraumatic Injuries

Training and competition in cycling involves a significant repetitive movement (pedal stroke) as well as constant contact between the athlete and the bike at relatively small interfaces (handlebar, pedal, and saddle). Sudden or drastic increases in training or competition volume can predispose an athlete to injuries that can interfere with competition. In addition, high-altitude illness should be considered among participants of races that are above 2500 m of elevation (Fig. 52.2).

Bicycle Fit

It is crucial for the clinician covering a race to be able to assess bicycle fit because errors in bicycle fit can cause acute exacerbations of preexisting atraumatic injuries during competition [13]. There are three primary contact points between the cyclist and the bike that must be addressed: shoe/cleat–pedal, pelvis–saddle, hands–handlebar.

- Cleat: Modern clipless pedal systems use cleats that are installed into the cycling shoe, and the location of this cleat is customizable. The cleat should be positioned within the shoe so that the first metatarsal head lies directly over the axle of the pedal, and rotation should be adjusted to avoid excess in-toeing or out-toeing.
- Saddle: The saddle can be adjusted up or down, forward and backwards. Changes in these two dimensions can alter the knee flexion angle and degree of ankle plantar/dorsiflexion during the pedal stroke. Saddle height should be adjusted so that, at the bottom of the pedal stroke, the knee is flexed to 25–30°. The fore-aft position of the saddle should be adjusted so that, when the pedals are horizontal,

Fig. 52.2 An ultra-endurance mountain biking race. (Courtesy of Morteza Khodaee)

a plumb line dropped from the tibial tubercle of the anterior knee should intersect the axle of the pedal.

- Stem and Handlebars: Should be positioned so that the torso is flexed to ~45° relative to the top tube (horizontal) with hands on the brake hoods and ~60° with hands in the drops.

Epidemiology of Overuse Injuries

In 2010, Clarsen et al. published a study of the nontraumatic injuries sustained by a cohort of 109 professional cyclists over a one-year period. Sixty-three athletes (58%) reported a total of 94 injuries [9]. The most common chronic injuries causing athletes to seek medical attention were low back pain (46%), knee pain (23%), and neck pain (10%). Of the injuries causing cyclists to miss training or competition, knee pain predominated (57%) followed by low back pain (17%) [9].

Upper Extremity

The most commonly reported chronic injury in the upper extremity is compression of the ulnar nerve, dubbed "cyclist's palsy." This is a compressive mononeuropathy of the ulnar nerve either proximal, within or distal to Guyon's canal caused by inadequate handlebar padding and infrequent hand position changes. Athletes usually complain of pain and paresthesias in the ring and small fingers [14, 15].

Lower Extremity

According to a 2007 review of cycling injuries by Wanich et al., the knee is the most common site of nontraumatic injury in cyclists [16]. The etiology often correlates with location of pain about the knee.

Anterior knee pain is one of the most common complaints by cyclists, and it can be caused by patellofemoral pain syndrome (including patellofemoral chondromalacia) or tendinitis of the quadriceps and/or patellar tendons [1]. Patellofemoral pain is caused by excess contact pressures between the undersurface of the patella and the trochlea of the distal femur. Risk factors include athlete anatomy (increased Q-angle, *patella alta*, trochlear dysplasia), as well as errors in bicycle fitting, especially a saddle that is too low or too far forward. The resulting excess knee flexion increases the resulting forces in the patellofemoral

joint [1, 8, 13, 17, 18]. These same errors in bike fitting can also cause quadriceps or patellar tendinitis [16].

Medial knee pain is often due to pes anserine tendonitis/bursitis or, less often, medial plica syndrome. The pes anserine bursa can be irritated by repetitive friction from the overlying hamstrings tendons. Medial plica syndrome is caused by irritation of an embryonic remnant of a synovial septum over the medial femoral condyle [16]. Cyclists often experience a painful popping sensation during knee flexion.

Lateral knee pain is often caused by Iliotibial Band (ITB) Friction Syndrome. Here, repetitive sliding of a taught ITB (specifically, the posterior fibers) over the lateral femoral condyle leads to inflammation and fibrosis of the ITB and the underlying fat. Contributing factors include the athlete's anatomy and improper bike fit. If the saddle is too high or too far back, the knee overextends and is in the "impingement zone" (<30° flexion) throughout much of the pedal stroke [19–21].

Achilles tendonitis is a very common nontraumatic injury affecting cyclists. Excess plantarflexion and/or dorsiflexion of the ankle during the pedal stroke causes microtrauma to the tendon, resulting in tenderness and pain with resisted foot plantarflexion [1, 11].

Saddle Problems

Saddle sores are a very common complaint of cyclists, especially during periods of increased duration of training. Moisture, pressure, and friction at the interface between the saddle and the ischial tuberosities leads to skin irritation and chafing, which can eventually lead to furuncles, and nodular induration. Perineal numbness, sometimes called "cyclist's syndrome," is caused by pudendal nerve entrapment from prolonged time in the saddle [13, 14, 19, 22, 23].

Road Cycling vs. Mountain Biking

Although road cycling and mountain biking share several fundamental common characteristics, the two are very different sports and pose different risks to athletes during both training and competition [24]. The only epidemiologic study directly comparing mountain vs. road biking injuries was published by Kotlyar in 2016 [10]. In this retrospective study, injuries sustained by 304 patients presenting with acute injuries sustained while road cycling (101) or mountain biking (203) were reported during a 3-year period [10]. The most common injuries sustained by both groups were abrasions/lacerations/contusions (64%) and upper extremity fractures (26%). Road cyclists sustained head injuries more often than mountain bikers (16% vs. 6%) while mountain bikers sustained thoracoabdominal injuries more often than road cyclists (8% vs. 2%) [10].

What Is Unique About Cycling Injuries?

- As a nonimpact sport, cycling is popular among athletes of all ages and a wide variety of skill levels.
- Traumatic injuries to cyclists are often the result of crashes that occur at high speeds, usually involving other cyclists or motor vehicles [25, 26]. The severity of these injuries varies widely, but these high-energy mechanisms can cause severe, life-threatening injuries.
- Most fractures resulting from crashes occur in the upper extremity, and the fracture type depends on how the athlete attempts to break their fall.
- Older cyclists involved in crashes may be at higher risk of hip and lower extremity fractures.
- Chronic overuse injuries can occur at specific athlete–bicycle interfaces due to the long hours that cyclists spend in the saddle during training and competition.
- Poor bicycle fit, as well as variations in anatomy, can predispose athletes to chronic repetitive-use injury to the lower extremities, especially the knee and ankle.

What Do Physicians Need to Know While Covering a Cycling Competition?

- The team physician should be able to assess and treat simple abrasions.
- The initial assessment of upper extremity fractures involves thorough inspection of the skin and soft-tissue envelope, assessment of distal neurovascular status, and provisional stabilization with slings or splints.
- During competition, acute exacerbations of nontraumatic injuries can hinder performance. Assessing bicycle fit is a key component of assessing these injuries.
- Lower extremity pain, especially about the knee, is an extremely common complaint among cyclists, and it usually improves with proper bike fit and adequate rest.

Return to Competition

- When a cyclist is injured during competition, the decision of whether to continue the race is multifactorial and should involve a discussion with the athlete regarding:
  - The severity of the injury (e.g., minor abrasion vs. long-bone fracture)
  - The level of competition and importance of the race to the cyclist
  - The impact of the injury on the cyclist's performance and ability to continue riding safely
  - The risk of reinjury or injury exacerbation during the remainder of the race

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Dance

53

Rajwinder S. Deu

Key Points

- There are multiple genres of dance, including ballet, modern, jazz, tap, hip-hop, musical theater, folk, and ethnic. Each placing the dancer at different risks.
- Factors that play a part in types of injuries includes: dance style, footwear, poor training, technical errors, unfamiliar choreography or style, lack of time for rest, and environmental factors.
- Lower extremity injuries are the most common in dance.
- Majority of participants are female.

Introduction

Dance continues to grow in popularity. There are a multitude of factors that play a part in the type of injuries that dancers sustain. The diverse styles (i.e. ballet, modern, jazz, tap, hip-hop, musical theater, folk, and ethnic) place different stresses on the body. Each style uses different footwear ranging from barefoot in modern, pointe shoes in ballet, and character shoes in musical theater. The combination of poor training, technical errors, unfamiliar choreography or style, and environmental factors including dance surface can contribute [1, 2]. The lack of time for rest and recovery due to rigorous rehearsal schedules, lengthy show runs, and intensive summer dance programs increase the risk as well [1, 2]. Dancers are particularly vulnerable when their technical mechanics break down due to muscle fatigue [1, 2] (Fig. 53.1).

R. S. Deu (✉)
Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: rdeu1@jhmi.edu

Injury Epidemiology

The majority of dance injuries are from overuse; however, traumatic injuries are not uncommon. A review of emergency room visits showed that 113,084 children were treated in emergency rooms for dance-related injury between 1991 and 2007 [3]. A systematic review of MSK injury in ballet showed that amateur dancers had a higher proportion of overuse injuries as compared to professionals [4]. Male professional dancers suffered a higher proportion of traumatic injuries, which accounted for half of their injuries [4]. Acute injuries are more likely to occur during rehearsal and performances [5]. Numerous studies have shown that the lower extremity is affected the most, with the ankle and/or foot as the most frequent [4, 6, 7, 8]. The most common injury locations for different styles appear in Table 53.1.

Ankle and Foot

Ankle and foot injuries are the most common traumatic injuries in dance [5, 9, 10, 11, 12, 13]. Majority of these injuries are secondary to ankle inversion while in plantar flexion, either due to missed landings from jumps or rolling over the outer border of the foot while on demi-pointe [1, 14, 15]. Ankle sprains are the most common result with the anterior talofibular ligament as the most commonly injured structure. Ankle sprains can also result in acute cuboid subluxation. The medial border of the cuboid subluxes in a plantar direction, causing a dorsal displacement of the fourth metatarsal and plantar displacement of the fourth metatarsal head [16]. The dancer will complain of lateral midfoot pain and may have difficulty bearing weight or difficulty achieving demi-pointe or pointe position [1, 13, 15, 17]. Cuboid subluxation can be treated with manual reduction or passive mobilization of the rear and midfoot joints [1, 15].

Even though the majority of complaints regarding the Achilles involves chronic tendinosis, tendon ruptures can

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Fig. 53.1 Ballet dancing

Table 53.1 Most common dance injury location categorized by style

Dance style	Most common	Second most common	Third most common	Fourth most common
Ballet [8]	Foot/ankle	Lumbar spine	Hip	Knee
Modern [8, 9]	Foot/ankle	Lumbar spine	Hip	Knee
Hip hop [10]	Foot/ankle	Knee/hip	Wrist/forearm	Shoulder
Irish [11]	Foot	Ankle	Knee	Hip
Revue [12]	Ankle	Knee	Foot	Lumbar spine

occur in any jumping dancer. They are more common in male dancers over age 30 and can occur when landing in hyper-dorsiflexion or with eccentric loading of the foot during push-off [1, 14, 15].

Dancers can suffer midfoot sprains and injuries to the Lisfranc joint. Multiple mechanisms leading to hyper-plantarflexion have been documented, ranging from a fall off pointe, missed jump landings, during spins, or during take-off for a jump [1, 14, 15]. Prompt diagnosis is critical as delay can limit surgical options and possibly make it a career-ending injury.

There are multiple fractures involving the foot that dancers are susceptible to. The fifth metatarsal is vulnerable to fracture with an inversion injury. A dancer's fracture consists of an oblique spiral fracture involving the distal fifth metatarsal shaft. This fracture can be treated nonoperatively even if displaced. Fracture of the base of the fifth metatarsal can include a simple avulsion of the tuberosity, which can be treated with a stiff-soled shoe or removable fracture boot. The most difficult to treat are fractures that occur at the metaphyseal–diaphyseal junction, which are known as a Jones fracture. These are most common in modern dancers and are at risk for nonunion due to poor blood supply in the area. It is treated with 6–8 weeks of nonweightbearing in a short-leg cast or fracture boot. High-level dancers may opt for surgical treatment to avoid prolonged immobilization.

Fracture and dislocation of the phalanx is rare but can occur from striking the foot against scenery [2]. A fracture can be buddy taped to the adjacent toes and the toe of the pointe shoe can be padded with lamb's wool to relieve pressure [2]. Dislocation should be reduced by hyperextending the joint and applying traction to pull the phalanx distally and then flexing the toe to reduce the deformity [2].

Knee

Patellar dislocation can occur when the knee is in full extension and sustains a valgus stress. This can be the result of a direct blow to the knee, twisting awkwardly on a planted foot, or attempting to compensate for poor turnout [18]. Dancers with poor turnout often hyperpronate and maximally externally rotate their feet placing torque on the extended knee and leading to malalignment of the patellofemoral joint [18].

This can also lead to lateral patellar dislocations and medial meniscal tears. Reduction of lateral patellar dislocation can be done by providing pressure medially on the lateral patellar margin while gently extending the leg.

ACL injuries are rare in dancers, particularly in comparison to athletes participating in team sports. Leiderbach et al. found an incidence of 0.009 per 1000 exposures [8]. In contrast, a review of NCAA athletes suffering ACL injury showed a range of 0.02–0.24 per 1000 exposures [19]. Female modern dancers had a three to five times greater risk that female ballet dancers and male dancers [8]. It is thought that the rigorous jump and balance training that dancers receive from a young age reduces their risk.

Shoulder

Shoulder dislocations are rare in dancers but can be the result of a direct blow to the shoulder, either from a fall on outstretched arm or having the arm pulled forcefully. Many dancers have increased laxity and may suffer from chronic shoulder instability.

Head

There are very few published papers regarding concussion in dance. Dancers can suffer a concussion from stunting, diving, flipping, an unintentional drop while partnering, a slip, fall, or direct blow to the head [20]. Table 53.2 contains a summary of location and traumatic dance injury.

Dance Floors

A dance floor has two components, a surface and a subfloor. The subfloor provides the shock absorbing properties while the surface provides the grip to prevent dancers from slipping as they glide and turn completing maneuvers. Wanke found that 12.8% of all accidents were caused by dance floor surfaces [21]. Almost half of the professional dancers (53.1%) and dance students (42.5%) claimed that the floor was too slippery or that they got stuck as cause for injury [21].

Table 53.2 Traumatic dance injuries categorized by location

Location	Injury
Ankle	Ankle sprain, Achilles tendon rupture
Foot	Midfoot sprain, Lisfranc injury, dancer's fracture, Jones fracture, phalangeal fracture, cuboid subluxation
Knee	Patellar dislocation, meniscal tear, ACL rupture
Shoulder	Shoulder dislocation
Head	Concussion

What Is Unique About Dance Injuries?

- Majority of dance injuries involve the foot and ankle.
- Dance organizations do not require pre-participation physical examinations.
- Dancers are generally unable to wear braces in performances due to their bulk which not only inhibit movement but do not fit in the shoe.

What Do Physicians Need to Know While Covering Dance Performance on the Sidelines?

Typically, physicians are not present during dance performances. Unlike other athletic activities, dance organizations do not require the presence of health-care providers. Many larger dance companies will have an athletic trainer and/or physical therapist present. Some physicians will run a clinic at the dance studio similar to an athletic training room.

General Rule About Return to Play

There are no specific guidelines regarding return to play. Most injuries do not require complete restriction from activity. The ballet barre is often used to progress an injured dancer to full activity. Shah has described the steps as follows: (1) Both legs with feet flat on the floor, (2) single, unaffected leg flat on the floor, (3) single, affected leg flat on the floor, (4) releve (weight-bearing on the metatarsal heads) both legs, (5) small jumps, (6) releve single, unaffected leg, and (7) releve single, affected leg [17]. The dancer must be pain-free to advance through the stages and once the ballet barre is mastered, the dancer can progress to center work using a similar progression. The final phase will include jumps and then pointe work [17]. Early evaluation and initiation of therapy (preferably by a dance-specific physical therapist) is key to early return to dance.

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54 Extreme Sports

Matthew J. Kraeutler, Lior Laver, and Omer Mei-Dan

Key Points

- Participation in extreme sports is associated with risk of injury or even death and therefore the extreme athlete – amateur or professional – as well as the medical personnel treating these athletes must consider the risk of injury and measures for injury prevention.
- Medical personnel treating the extreme sports athlete need to be aware that there are numerous differences that must be appreciated between the common traditional sports and this newly developing area.
- The highest injury rates in extreme sports are found in two groups: new and inexperienced athletes who have just started engaging in extreme sports and experienced extremists.
- Comparison of injury rates across extreme sports is difficult given the large variance in terrain and environmental conditions – often changing variably during a single competition or event.

Introduction

The definition of extreme sports (ES) inhabits any sport featuring high speed, height, real or perceived danger, a high level of physical exertion, highly specialized gear or spectacular stunts, and elements of increased risk [1]. These ES activities tend to be individual and can be pursued both competitively and noncompetitively [2]. They often take place in remote locations and in variable environmental conditions (weather, terrain) with little or no access to medical care [3], and even if medical care is available it usually faces challenges related to longer response and transport times, access to few resources, limited provider experience due to low patient volume, and more extreme geographical and environmental challenges [4].

Examples of popular ES include BMX, mountaineering, hang-gliding and paragliding, free diving, surfing (including wave, wind, and kite surfing), personal watercraft, whitewater canoeing, kayaking and rafting, bungee jumping, BASE jumping, skydiving, extreme hiking, skateboarding, mountain biking, in-line skating, ultra-endurance races, alpine skiing, snowboarding, ATV, and motocross sports [5].

In the last two decades, there has been a major increase in both the popularity and participation in ES, with dedicated TV channels, internet sites, high-rating competitions, and high-profile sponsors drawing more participants [6–8]. The popularity of ES has been highlighted in recent years by the success of the X-games, an Olympic-like competition showcasing the talents in ES.

Participation in ES is associated with risk of injury or even death and therefore the extreme athlete – amateur or professional – as well as the medical personnel treating these athletes must consider the risk of injury and measures for injury prevention. Recent data suggest that the risk and severity of injury in some ES is unexpectedly high [9].

Medical personnel treating the ES athlete need to be aware that there are numerous differences that must be appreciated between the common traditional sports and this

M. J. Kraeutler
Department of Orthopedics, St. Joseph's University Medical Center, Paterson, NJ, USA

L. Laver
Head of the Hip Preservation and Sports Hip Service, Sports Medicine Unit, Department of Orthopaedics, Hillel Yaffe Medical Center, Hod-Hasharon, Israel

O. Mei-Dan (✉)
Department of Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: omer.meidan@cuanschutz.edu

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newly developing area. These relate to the temperament of the athletes themselves, the particular epidemiology of injury, the initial management following injury, treatment decisions, and rehabilitation.

The purpose of this chapter is to provide an epidemiologic overview of the available literature on common injuries affecting extreme athletes, the risk of their occurrence and available prevention measures in this athletic population.

Epidemiology of Injuries in Extreme Sports

Despite great evolution in traditional sports epidemiology, injury mechanisms in ES are poorly understood, particularly the injury pattern in many sports. The highest injury rates in ES are justifiably found in two groups: new and inexperienced athletes who have just started engaging in extreme sports and experienced extremists [10].

Reported injury rates in ES may be expected to increase during competition rather than training, a trend well recognized in common team sports [11–13]. Although injury rates for various ES have been determined and published, in some disciplines, the injury and fatality rates are hard to establish due to lack of formal recorded events.

In addition, unlike expected terrain and environmental conditions which are similar in most traditional sports (e.g., soccer is played on a real or synthetic grass field), comparison of injury rates across extreme sports is difficult given the large variance in terrain and environmental conditions – often changing variably during a single competition or event.

Specific Extreme Sports and Their Associated Injuries

Skydiving

Skydiving is a major air sport of parachuting from an aircraft. It is practiced both competitively and recreationally. The International Parachuting Commission (IPC) reported in 2009 approximately 5.5 million jumps, made by almost one million jumpers in 40 countries [14], including tandem jumps. The reported number of jumpers self-operating their equipment added up to some 220,000 skydivers performing 4.7 million skydives [15]. Since the late 1980s, few epidemiological studies have been conducted in order to establish the injury and fatality rates associated with in the sport. Focusing on sport skydiving, most reports are incidence and injury statistics from local governing bodies in the USA and Europe and report relatively high injury rates [16–20]. The majority of jumps are performed by a significantly smaller number of sports skydivers, whereas a larger number of participants perform fewer jumps.

The 2006 international safety report states 1 fatality per 16,300 jumpers, which increases to 1 fatality per 4000 skydivers when tandem jumper data is excluded [19]. Sixty percent of fatalities were categorized as expert jumpers. Students accounted for about 20% of fatalities [19]. In 2009, 62 skydiving fatalities were reported worldwide by the IPC in 40 countries [15], indicating 1 fatality per 88,000 jumps. Data also showed that 44 of the 62 fatalities (71%) occurred with the skydiver having at least one good parachute on and that the majority – 49 fatalities (79%) – have been caused by human errors. The fatalities were related to low or no pull of the parachute (30%), malfunctions of the parachute system (15%), reserve canopy problems (15%), midair collisions (20%), and landing errors (20%).

Fatality rates, causes, and profile of jumpers tend to be consistent worldwide. A study from Sweden showed that the fatality rate decreased 11-fold for the periods 1994–2003 compared to 1964–1973 [19]. United States Parachute Association (USPA) data from 2008 showed most victims were experienced jumpers in their fourth to fifth decade of life with an average 11 years in the sport [21]. Half of these jumpers held the highest parachute license (USPA D-License) and had averaged 1,954 jumps (median 665).

Barrows et al. [16] documented jumping incidents during two consecutive world free fall skydiving conventions in Illinois in 2000–2001. The event is considered to be the world's largest annual skydive gathering with participants from 55 countries. They followed 8,976 skydivers making 117,000 skydives in 20 days, indicating a total injury rate of 170 per 100,000 jumps, while only 30% of those required a visit to an emergency department and as few as 10% continued to hospital admission. Sixty-six percent of the injuries were considered minor; 32% of these were abrasions and contusions, and 22% were lacerations. Of the jumpers who visited the emergency department for follow-up treatment, half suffered from extremity trauma which was related to the lower extremity in 80% of patients. Twenty fractures were diagnosed (15 limbs, 5 spinal) indicating a rate of 0.5 fractures per 100,000 jumps.

Westman et al. [22] evaluated the skydiving injury rate during five consecutive years and more than 500,000 jumps in Sweden. The incidence of nonfatal events was found to be 48 per 100,000 jumps (or 2,100 jumps per incident in total or 3,200 jumps per licensed jumpers) and 88% of those occurred around the landing with 51% of injuries involving the lower extremities, 19% involving the upper extremities, 18% involving the back and spine, and 7% involving the head. Forty-one percent of the injuries were categorized as minor, 47% as moderate, and 12% as severe. Most serious injuries were experienced by licensed skydivers, while students in training had a six-times higher injury rate. Interestingly, women were overrepresented with injuries in this study (RR 1.4–2.7) and they also had a higher proportion of landing injuries than men.

Although many parameters and participants have changed over the last 20 years, injury rates remain similar. Modern equipment has decreased overall morbidity and mortality, but it has also led to faster landings with increased limb injuries.

BASE Jumping

BASE (**B**uilding, **A**ntenna, **S**pan [a bridge, arch, or dome], and **E**arth [a cliff or other natural formation]) jumping is a sport that developed from skydiving and uses specially adapted parachutes to jump from fixed objects (Fig. 54.1). Thought to involve only about 2,500–3,000 active members worldwide, it is considered among the most dangerous adventure sports in the world and significantly more dangerous than skydiving, as BASE jumps are performed from much lower altitudes (often less than 150 m above ground level). Lower falling speeds with far less aerodynamic control and a high risk of losing flying stability leave little room for error. If the parachute is deployed while the jumper is unstable, there is a high risk of entanglement or malfunction.

Fig. 54.1 BASE jumping. (With permission and courtesy of Dr. Omer Mei-Dan)

In such cases, the single canopy used may also be facing the wrong direction. Off-heading opening resulting in object strike is the leading cause of serious injury and fatality in BASE jumping.

Few studies have been conducted on this unique population. Soreide et al. [23] determined that BASE jumping is associated with a five- to eight-fold risk for fatality or injury when compared to regular skydiving. The fatality rate associated with BASE jumping was found to be 0.04% [23]. In a study by Monasterio and Mei-Dan [24] among 35 experienced BASE jumpers, an estimated injury rate of 0.4% was found in 9,914 jumps, a finding similar to Soreide's results [23]. Twenty-one (60%) jumpers in this study were involved in 39 accidents. The majority of accidents (28 accidents, 72%) involved the lower limbs, 12 (31%) involved the back/spine, 7 (18%) the upper limb, and 1 (3%) was a head injury. The sport attracts predominantly male participants. In Monasterio and Mei-Dan's study [24], 75% of injuries were categorized as moderate or severe, as opposed to Soreide's series where most injuries were considered minor [23]. This could be explained by the fact that the single high site (1,000 m) studied in Soreide's series offered relatively safe jumping conditions allowing greater speed generation before parachute deployment and controlled landing. The rate of injuries requiring hospitalization in Monasterio and Mei-Dan's study was 294 per 100,000 jumps, 16-times higher compared to the rate of such injuries in freefall skydiving found by Burrows et al. (18 per 100,000 jumps) [16]. A more recent study by Mei-Dan et al. [24] analyzing fatality rates associated with wingsuit use in BASE jumpers showed a growing pattern of wingsuit-related fatalities. Most fatalities occurred in the summer period in the northern hemisphere and were attributed to cliff or ground impact, being mostly the result of flying path miscalculations [25].

Climbing

Climbing is an adventure sport which has developed from alpine mountaineering. Its popularity has vastly grown in the past three decades, with the introduction of indoor climbing gyms and climbing walls, becoming globally spread and evolving to new categories such as ice climbing (Fig. 54.2), bouldering, speed climbing, and aid climbing (see Chap. 66). The evolution in the sport also developed competition between individual climbers, progressing in time to formal competitions and world championships gaining growing popularity with an ever increasing number of participants, large audiences, and live media coverage. Estimations of the worldwide number of rock climbers are significant, with over two million in Europe and about nine million in the USA. Mountaineering numbers are estimated to be even higher.

Fig. 54.2 Ice climbing. (With permission and courtesy of Dr. John Hill)

Rock climbing, considered the purest form of the sport, has several subdisciplines, differing by the level of risk and difficulty, the climber's experience and skills, route difficulty, equipment used, and environmental factors (surface, remoteness of location, altitude, weather). Many climbers regularly practice more than one subdiscipline, making analysis and risk determination difficult. This also increases the time involvement in the sport and, as a consequence, the risk of injury.

Strength-to-weight ratio is one of the most important factors in climbing, and climbers' anthropometric data is very similar to that found in gymnasts. Anorexia athletica, especially in top female climbers, is not an uncommon finding.

A cross-sectional survey on rock climbing showed a lower injury rate compared to football and horse riding [26]. However, these sports rarely result in catastrophic events or fatalities. Most studies show that the incidence of overuse injuries is associated with climbing frequency and difficulty [27, 28]. Most injuries are sustained by the lead climber, with falls being the most common mechanism of acute injuries [29]. Overall, most registered injuries in climbing studies are of minor severity. The fatality rate reported in climbing ranges from 0% to 28% [30]. This wide range could be explained by varying methodology and data collection techniques in different series.

In indoor climbing, injury rates are much lower with 0.027–0.079 injuries/1,000 h of participation and fatalities are rare [31]. Overuse injuries are more common in this discipline, most commonly involving the upper extremities – mainly finger injuries. Although climbing relies on the synchronized and optimal function of the whole body, activity and performance are primarily limited by finger and forearm strength. Various gripping techniques lead to transmission of extremely high forces to the fingers, making overuse injuries of the fingers and hands the most common complaints in rock climbers [27, 28, 31–34]. Some injuries, such as flexor tendon pulley ruptures or the lumbrical shift syndrome, are unique to the sport and are rarely seen in other patient populations [27]. Little data exists for ice climbing, and although severe injuries and fatalities occur, most recognized injuries are of minor severity and are comparable with other outdoor sports [30, 35].

Most studies on mountaineering report fatality/injury rates per 1,000 climbers or 1,000 summits, making it difficult to compare to the more common 1,000 h of sports participation used in other disciplines. Monasterio et al. [35] surveyed 47 mountaineers (mean age 33 years) from 8 different countries and 23 climbers (49%) reported a total of 33 accidents. In mountaineering, additional environmental factors (e.g., avalanches, crevasses, high altitude illnesses with neurological dysfunction) can directly influence injuries and fatalities [36, 37].

It is clear that every climbing subdiscipline contains different levels and types of risks, injury, and fatality rates, with characteristic objective dangers and physical hazards for each subdiscipline. Nevertheless, dangerous situations and predictable injury patterns or accident circumstances can still be avoided or successfully managed with adequate preparation, training, and experience. In contrast, in indoor and sport climbing, these objective and external dangers are greatly reduced, although the risk of a fatal injury is still present.

Skiing and Snowboarding

Skiing and snowboarding are the two main piste-based snow sports. With roots in Nordic (cross-country) skiing, alpine skiing gradually evolved over time from a method of transportation in Scandinavia thousands of years ago into the present recreational and competitive sport, becoming a winter Olympic sport in Garmisch in 1936. Snowboarding developed in the 1960s and its popularity took off in the mid-1980s, leading to the development of terrain parks at ski

areas offering features such as half- and quarter pipes, rails, and big air jumps, allowing snowboarders to perform aerial tricks and maneuvers (Fig. 54.3) difficult to perform on traditional alpine skis.

As with most outdoor activities, skiing and snowboarding are associated with a risk of injury (see Chaps. 71, 72, and 73). The current risk of a recreational snow sport-related injury is between two and four injuries per 1,000 participant-days [38], a risk much lower than in popular sports such as football and rugby and has decreased steadily over recent years [39] due in part to improvements in equipment, ski area design and maintenance, and piste preparation [39]. The risk of injury from recreational alpine skiing is between 1–2 injuries per 1,000 participant-days [38, 40]. Prior to release bindings, fractures of the lower leg were common from twisting forces transmitted unmitigated from the ski up to the lower leg, and their introduction significantly reduced these fractures. The fracture rate from alpine skiing is approximately 19% [41], and common sites include the clavicle, proximal humerus, and tibia. Nevertheless, alpine skiers are still more likely to injure their lower rather than upper limb, with the knee joint being the single commonest site of injury among skiers and most of these injuries are soft tissue/ligamentous in nature, as illustrated in Figs. 54.4 and 54.5 [42].

Fig. 54.3 Snowboarding. (With permission and courtesy of David Carlier)

The risk of injury from snowboarding is generally estimated at about twice that of alpine skiing and currently stands at between 2–4 injuries per 1,000 participant-days [40]. Snowboarders are more likely to injure their upper limb than their lower limb [41]. Unlike skiers, when losing balance, snowboarders cannot step out a leg to regain balance. As a result, falls due to loss of balance are frequent. This commonly results in falls on an outstretched hand and places the upper limb, especially the wrist joint, at high risk of injury [43]. The fracture rate among snowboarders is twice that of alpine skiers [41], caused largely by the high rate of wrist fractures (up to 33% of all injuries) [44].

Muscle and ligament strains/sprains are still common as are contusions from off-balance falls. Snowboarders suffer a higher rate of shoulder joint injuries due to an increased tendency to fall onto the upper limb [41]. Jumps and other aerial maneuvers, commonly performed in snowboarding, are associated with a relatively small but definite risk of injury to the spine [45–47]. Figure 54.6 illustrates injury types in snowboarding.

The injury risk among professional skiers and snowboarders is approximately three-times that of recreational participants [48] and has been calculated to be 17 injuries per 1,000 ski runs [49]. Almost one-third of injuries among professional athletes were classified as severe, leading to an absence from participation of more than 28 days [50].

The fatality risk in snow sports is even lower at one death per 1.57 million participant-days [51]. This equates to approximately 39 traumatic deaths per year in the USA out of a total of almost 60 million participant-days [51]. These fatality rates are much lower compared to other popular recreational activities such as swimming and cycling [51]. The commonest cause of a traumatic snow-sport-related death is a high-speed collision with a static object (tree, pylon, or another person) [52, 53]. Many of these deaths involve head injuries [53]. Nontraumatic causes of death on the slopes include ischemic heart disease, hypothermia, and medical events such as acute severe asthma attacks [52]. A less frequent but important mechanism of death is the so-called non-avalanche-related snow immersion death (NARSID), also known as a “tree well death” [53, 54], when skiers/snowboarders fall into a hidden pit underneath a tree. Unless the event is witnessed, self-extraction from the tree well is nearly impossible. The trapped individual tends to cause more snow to fall into the pit as they struggle to try to extract and death usually resulting from hypothermia or asphyxiation from snow falling in [55].

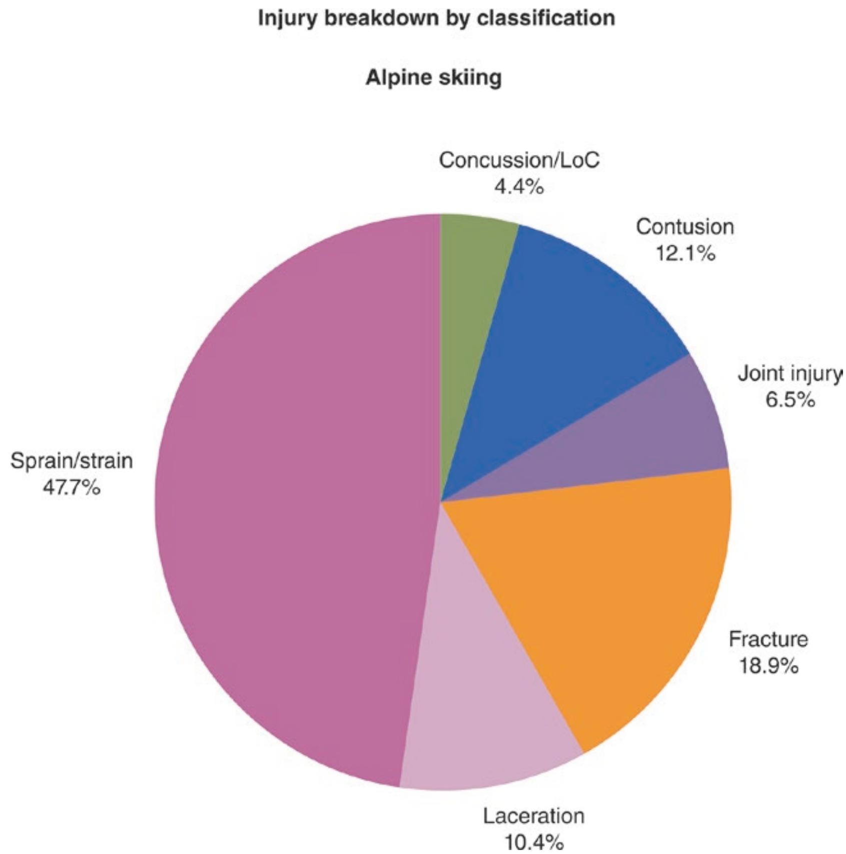

Fig. 54.4 The breakdown of alpine skiing injuries by types [42]. (Modified with permission)

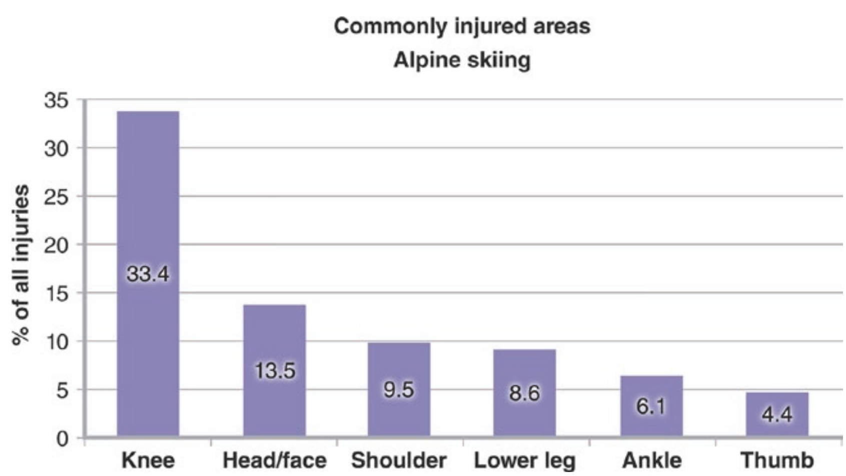

Fig. 54.5 Commonly injured areas in alpine skiing [42]. (Modified with permission)

Return to Sports

Published reports on extreme sports athletes suggest that they return to active participation once rehabilitation is completed, even after life-threatening and disabling injuries [56]. Although half of the injured white water kayakers sought medical care for their injury, and almost one-third missed more than 1 month of kayaking because of their injury, almost all (96%) reported a complete or good recovery with the best outcomes associated with impact injuries and the worst with overuse injuries [56]. Specialized rehabilitation is frequently required as sports tend to feature one predominant key maneuver or action. A gradual return to extreme sports activity may not be possible as most require full commitment and an "all or nothing" level of performance is needed.

Simulation exercises can be performed in a reduced risk environment prior to full return. Confirming that functional restoration has returned may be something that only the athlete themselves can determine. Resumption of their sport before body and mind are fully ready may result in

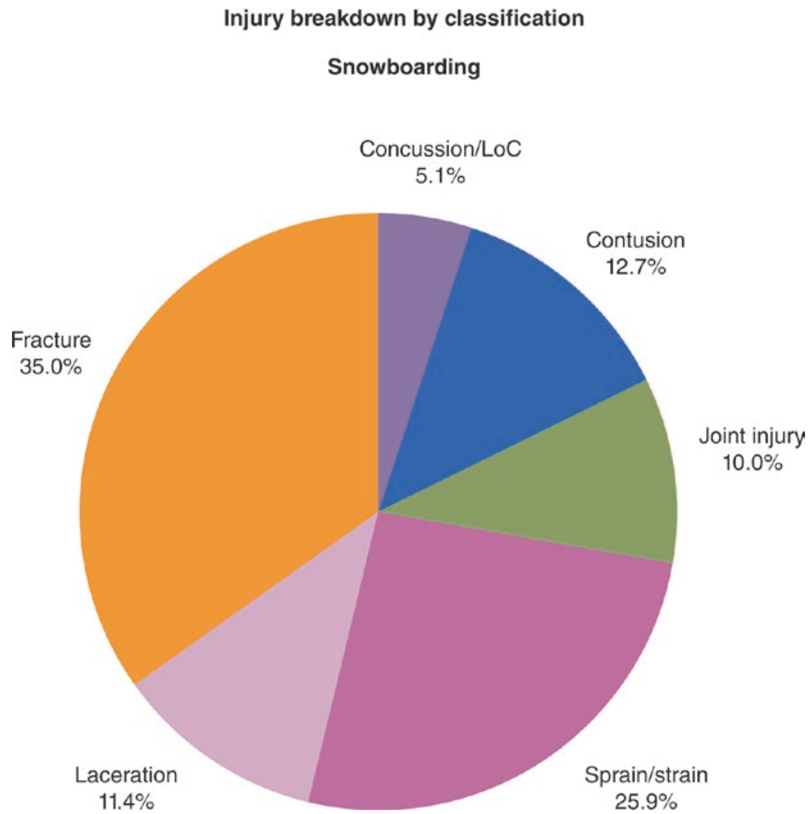

Fig. 54.6 The breakdown of snowboarding injuries by types [42]. (Modified with permission)

life-threatening injuries rather than "just" a reinjury for other traditional sports. A shoulder prone to dislocation for the skydiver or BASE jumper can result in inability to deploy the parachute on time or at all. The athlete would be better off testing his shoulder stability and function primarily in a wind tunnel environment rather than off the nearest 200 m cliff.

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Field Hockey

55

Katherine S. Dahab

Key Points

- Field hockey is played by pediatric, collegiate, and adult male and female athletes.
- Protective eyewear, shin guards, and mouthguards are the protective equipment used by athletes.
- Lower extremity is the most common injury location.
- Head and facial injuries have decreased significantly with mandated use of protective eyewear in high school athletes.
- Injuries are more likely to occur during games than practices.

Introduction

Field Hockey is one of the most popular team sports worldwide [1]. In North America, most participants are women (Fig. 55.1). The game is played in two halves, which are 35 min each in collegiate and elite competitions and 30 min at the high school level. Each team has 11 players, including the goalkeeper. The sticks have a curved head and are flat on one side and rounded on the other. Players use only the flat face of their sticks to dribble and hit the ball down the field or pitch. The ball is made of solid plastic and is slightly larger than a baseball. The ball weighs between 156 and 163 g (5.5 and 5.75 oz). Points are scored by shooting the ball in the opponent's net past the goal line. Only shots taken within the striking circle can count as goals. Players are not allowed to kick, hold, or carry the ball.

Recent advances in stick construction have enabled players to generate more power and velocity. High school players are able to generate speeds >80 kmh (>50 MPH) [2] and elite players have recorded shot speeds close to (100 MPH) [3]. Over the past two decades, more play is on artificial turf, which makes for a smoother, faster playing surface. In addition, the ball tends to be elevated off the playing surface more frequently on turf compared to natural grass [4]. For the 2011–2012 school year, the National Federation of State High School Associations (NFHS) mandated that high school field hockey players wear protective eyewear.

Current International Field Hockey Federation rules do not require the use of protective eyewear, but most will allow players to wear approved protective eyewear if they choose to do so. Regardless of level of play, the current rules recommend that players have only minimal personal protective equipment including mouthguards, shinguards, and ankle guards. Surveys of players show that many players do not wear recommended mouthguards regularly [5].

Within the United States alone, there are a number of different field hockey organizations. Starting with youth leagues, there are clubs throughout the country. The NFHS is the governing body for high school athletes. At the collegiate level, women's field hockey is played at NCAA division I, II, and III schools. For men, NCAA field hockey is a club sport. At the national level, USA Field Hockey has both men's and women's teams and the Pan American Hockey Federation is an international league [6].

Injury Epidemiology

Despite the popularity of the sport, injury data epidemiology is limited. Most available studies focus on collegiate women athletes. One of the major sources of field hockey injury epidemiology looks at injury surveillance in collegiate women's field hockey from 1988–1989 to 2002–2003 (Table 55.1) [4]. This study shows that injuries are more likely to occur during games than in practice. Looking at

K. S. Dahab (✉)
Department of Orthopedics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO, USA
e-mail: Katherine.dahab@childrenscolorado.org

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Fig. 55.1 Women's field hockey. (Reprinted with permission from iStock# 139972828)

Table 55.1 Most common injuries in women's NCAA field hockey game [4]

Diagnosis	1989/1990 through 1995/1996		1996/1997 through 2002/2003[a]	
	Percent of all injuries	Rate per 1000 athletic exposure	Percent of all injuries	Rate per 1000 athletic exposure
Ankle sprain	10.5	0.9	8.3	0.6
Knee internal derangement	7.0	0.6	7.5	0.5
Finger fracture	5.1	0.4	3.8	0.3
Concussion	5.1	0.4	9.0	0.6
Upper leg strain	4.3	0.4	5.8	0.4

[a]Placement of corner shots and no offside (new rules) were implemented in NCAA women's field hockey in 1996

practice injuries alone, injury rates were three times higher during preseason compared to regular season practices.

Location of Injury

Within the pediatric age group, the most common site of injury is the hand/wrist (34%), followed by the face (21.3%) and the lower leg/foot (19.2%) [7].

In collegiate women, over 40% of all game injuries and 60% of all practice injuries were to the lower extremity [4]. During games, ankle ligament sprains and knee internal derangement injuries were the most common injuries. During practices, upper leg muscle strains accounted for nearly 27% of all reported injuries. In addition to the lower extremities, injuries to the head and neck and upper extremity are common [4].

Among elite international women field hockey players, the head/face is the most common location of injury (40%), whereas for men it is reported to be the head/face (27%) as well as the thigh/knee (28%) [8]. Severe injuries are rare in elite international settings [8].

Mechanism of Injury

During a field hockey game, there are three primary potential sources of injury. These include player-to-player contact, other contact (with balls, sticks, and the ground), and no contact. The majority of game injuries reported (approximately 60%) were from contact with the stick or ball, which has also been shown in other studies [8] Just over one quarter of injuries (26%) were noncontact injures and only 13% were due to player-to-player contact. In contrast, most injuries during practice (64%) were noncontact injuries [4].

During games, 77.4% of above-the-neck game injuries were caused by contact with the ball or stick [4, 8]. Most hand/finger/thumb game injuries were sustained by defensive players and nearly all of these injuries (94.9%) were caused by contact with the ball or stick [4].

Injuries by Player Position

Most game injuries were reported in midfielders (28%), though other positions were not far behind with 22% among forwards, 24% in defensive players, and 20% in goalies (Table 55.2) [4].

A different study reports that goalies have the highest risk of injury [1]. Common injuries to goalies include concus-

Table 55.2 Injuries in women's NCAA field hockey game by position, location on the field, and injury mechanism (1996–1997 through 2002–2003) [4]

	Above the neck[a] (%)	Hand/finger[b] (%)
Player position		
Forwards	33	29
Midfield	31	24
Defense	29	39
Goalie	3	0
Location at time of injury		
Goal	30	25
Within 25 yard-line	39	46
Other	31	29
Mechanism of injury		
Contact with another player	20	0
Contact with ball	47	49
Contract with stick	31	46
Contact with goal	1	0
Contact with surface	2	5

[a]Includes head, face, eye, ear, teeth, nose, mouth, and jaw injuries
[b]Includes hand, fingers, and thumb injuries

sions, wrist fractures, ankle sprains, thigh muscle strains, and knee ligament sprains. Most injuries to goalies occur from contact with another player, the field or the goal. Among field players, those who play multiple positions seem to be at highest risk for injury [1].

Elite Male Field Hockey Injuries

Injury patterns during the men's field hockey junior world cup 2009 are interestingly different from those reported in collegiate women athletes [9]. In addition to the different population, this could to be due to the short duration of the study and different methodology. In this population, most of the head and face injuries (54%) were from player-to-player contact, while 33% were from contact with a stick and 13% were noncontact injuries. Most of the head and face injuries were contusions (42%), lacerations (21%), and concussions (21%). Most of the reported injuries (79.2%) were in the second half of match play compared to 20% in the first half [5].

Traumatic upper limb injuries during the 2009 men's field hockey junior world cup were from contact with the ball or stick and most injuries were to the left arm/wrist/hand compared to the right. Despite these injury risks, the use of protective gloves in field hockey is rare and most players play without them [10].

Injury Patterns After Mandated Eye Protection

Epidemiological studies assessing eye, head, and facial injuries in high school field hockey players before and after the mandated use of eye protection show a significant decrease in eye, head, and facial injuries with the use of protective eyewear [11]. In addition, the concussion rates did not significantly change before and after the use of protective eyewear, showing that using the protective eyewear did not likely increase aggressive play and more player-to-player contact injuries [11].

What Is Unique About Field Hockey Injuries?

- Field hockey is a limited contact sport with most injuries occurring from contact with the ball or stick.
- Injuries can occur from player-to-player contact, contact with the ball or stick, or noncontact injuries.
- A large percentage of injuries occur to the head and face.

What Do the Physicians Need to Know While Covering Field Hockey Games on the Sideline?

- Physicians should be competent in concussion management, laceration repair, bracing/splinting/taping for ankle, knee, hand, wrist, and finger injuries.
- Prepare for assessment and referral for possible fractures.
- Have plenty of ice on hand for contusions.
- Prepare for dental and ocular injuries.

General Rule About Return to Play

Assess if the athlete is able to demonstrate full range of motion and strength in the injured body part. Assess for mechanics and altered gait with running after a lower extremity injury. If the athlete is near-normal in strength and motion, consider taping/bracing for support and return to play (limited evidence exist). If the athlete shows deficits in strength and or/motion, consider removal from play and further evaluation. If there is any concern for a concussion, fracture, or other significant injury, the athlete should not return to play until he/she has further evaluation.

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Football

56

Matthew S. Leiszler, Sourav K. Poddar, and Bjorn C. Irion

Key Points

- Football is one of the most popular sports in the United States.
- It is a high-velocity collision sport.
- Football has one of the highest reported injury rates of any sport.
- There are similar injury patterns across all levels of competition.
- Physicians should be aware of emergency action plan during sideline coverage.
- The most common injuries during game competition are to the knee, ankle, and shoulder.
- During practice, injuries to the knee, trunk, upper leg, and shoulder account for the majority of injuries.
- The most common mechanism of injury is player-to-player contact.
- Injuries based on position vary across competition levels.
- At the collegiate level, running backs and quarterbacks are the most injured offensive players followed by wide receivers and offensive lineman.
- At the collegiate level on defense, linebackers are the most frequently injured players, followed by defensive backs and defensive lineman.
- At the professional level, wide receivers and tight ends have the highest injury rates per exposure followed by defensive backs, running backs, linebackers, defensive lineman, offensive lineman, quarterbacks, and specialists.

M. S. Leiszler (✉)
University Health Services, University of Notre Dame, Notre Dame, IN, USA
e-mail: mleiszle@nd.edu

S. K. Poddar
Department of Family Medicine and Orthopedics, CU Sports Medicine Center, University of Colorado School of Medicine, Denver, CO, USA
e-mail: sourav.poddar@ucdenver.edu

B. C. Irion
Western Orthopedics & Sports Medicine, Grand Junction, CO, USA

Introduction

Football is one of the most popular sports in the United States with an estimated 3 million athletes participating in youth football, 1.1 million in high school football, and 100,000 in collegiate and professional football (Fig. 56.1) [1]. Football is a physical, high-velocity collision sport involving intermittent sprint activity and high-energy impacts often between large, powerful players. Consequently, there is a high risk of injury associated with the sport, and participation in youth football has actually decreased in the United States largely due to parental fear of injury [2, 3].

Injury Epidemiology

Football has one of the highest reported injury rates of any sport with an estimated 1.2 million football-related injuries occurring annually in the United States [4]. Injuries range from minor sprains, strains, and concussions to career-ending tears, ruptures, and fractures. Injury rates in football are high across all levels of competition with injury rates as high as 8 injuries per 1000 athlete exposures (AE) in high school athletes, 36 per 1000 AE in collegiate athletes, and 65 per 1000 AE in professional football [5–7]. Hootman et al. showed that football had the highest rate of injuries per exposure in both competitions and practices when compared to 14 other collegiate sports, with a rate over double that of all sports except men's wrestling [8]. Game injury rates have been shown to be six times higher than in-season practice injury rates [9] and spring football injuries are twice as common as in the fall [8].

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Fig. 56.1 An NCAA football game. (Courtesy of Matt Cashore)

The most common injuries during game competition are injuries to the knee, ankle, and shoulder (19.1%, 15.7%, and 15.2%, respectively) with sprains, strains, and contusions accounting for the majority of each specific injury types (36%, 15.7%, and 12.5%, respectively) [9]. During practice, injuries to the knee, trunk, upper leg, and shoulder accounted for the majority of the injuries, primarily including strains and sprains (24% each) [9]. The most commonly injured knee ligaments are the MCL followed by the ACL. Lateral ankle sprains are the most common ankle injury.

Similar injury patterns occur across all levels of competition with some slight variation in frequency. As seen in Table 56.1, lower leg/ankle/foot, knee, and shoulder injuries are the most common at the high school level. Lower leg/ankle/foot, knee and hip/thigh are the most common areas injured in collegiate athletes. Hip/thigh, lower leg/ankle/foot, and knee injuries are the most common injuries at the professional level [6, 11, 12]. Table 56.1 summarizes and further breakdowns of injuries at each level of competition.

The most common mechanism of injury is player-to-player contact. This is true across all levels of football [6, 9, 11], accounting for 74.5% of the injuries at the collegiate level [9]. This is followed by noncontact injuries and overuse injuries.

Table 56.1 Injury percentages by the body part injured across high school, collegiate, and professional football [6, 11, 12]

	High School (%)	NCAA (%)	NFL (%)
Head/face	11.5	8.3	7.5
Shoulder	12.4	12.7	8.4
Arm	7.4	2.6	3.1
Hand	9.3	5.9	6.0
Torso/neck/spine	11.6	9.8	12.4
Hip/thigh	9.3	16.6	21.3
Knee	15.2	16.9	17.8
Lower leg/ankle/foot	22.4	22.2	20.5
Other	1.1	5.0	3.0

Injuries based on position vary across competition levels. In a 15-year injury surveillance study of NCAA football players by Dick et al., running backs and quarterbacks were the most injured offensive players followed by wide receivers and offensive lineman. On the defensive side, linebackers were most commonly injured followed by defensive backs and defensive lineman [6]. At the professional level, wide receivers and tight ends had the highest injury rates per exposure followed by defensive backs, running backs, linebackers, defensive lineman, offensive lineman, quarterbacks, and specialists [12]. Concussion rates in professional football

players followed a similar trend with tight ends, wide receivers, defensive backs, and running backs sustaining the highest concussion rate per exposure followed by quarterbacks, offensive lineman, linebackers, defensive lineman, and specialists [12].

Football accounts for the largest proportion of sports-related concussions with recent data estimating that almost one-third of the 105,600 annual estimated sports-related concussions in NCAA athletes are sustained during participation in football [13]. However, the incidence per athlete-exposure was lower in football than some other sports in NCAA Injury Surveillance Data, with a rate of 6.71 per 10,000 AE, trailing men's wrestling (10.92), men's ice hockey (7.91), and women's ice hockey (7.50). Player contact is the main mechanism for sports-related concussion. In order to improve safety, numerous concussion policies and education programs have been created to attempt reduce the incidence of concussion in football. Rules changes in the professional and collegiate game have been implemented, which can have an effect on other sites of injury [10].

What Is Unique About Football Injuries?

Football players sustain a wide variety of injuries, but the following injuries are examples of those that are seen more commonly in football than most other sports.

- Exertional heat illness includes a range of conditions related to physical exertion in hot or humid conditions and accounts for 5.6% of all preseason football injuries [14]. Heat illness is particularly common in the preseason or early fall sports season when athletes are not yet acclimatized to physical exertion in the heat. Football places additional risk to athletes due to the multiple layers and protective equipment worn during practices. The greatest risk of heat illness is in the first 14 days of the season [14]. The NCAA has instituted policies limiting practice time for the first 5 days of the season to allow for acclimatization of the athletes. USA Football has made similar recommendations for other football players at all levels.
- Turf toe is an injury to the first metatarsal–phalangeal joint, commonly caused by a hyperextension mechanism to the joint. In football, this commonly occurs when an athlete sustains an axial load to a plantar-flexed foot with a dorsiflexed hallux, often due to another player falling on the back of the foot. The severity of the injury ranges from capsular or ligamentous sprains to chondral injuries, plantar plate injuries, or dislocations [15].
- Lisfranc joint injuries account for up to 4% of injuries in football players. These injuries involve damage to the bony or ligamentous structures of the tarsometatarsal (TMT) joints of the foot. Football players are especially prone to these injuries, with lineman accounting for 29% of Lisfranc joint injuries [16]. The associated mechanism of injury results from an axial longitudinal force to a plantarflexed foot, which can occur with tackling, blocking, or other collisions.
- Brachial plexus traction injuries are the most common injury of the cervical spine and peripheral nerves in football [17]. Commonly referred to as "stingers" or "burners," these injuries describe a constellation of pain, burning, and paresthesias that radiate down the arm typically in the C5 and C6 distributions. Most of these injuries occur during direct contact during tackling and are thought to be due to nerve root compression, brachial plexus stretch or direct blows to the superficial fibers of the brachial plexus [16]. These injuries are usually self-limited resolving in seconds to minutes.
- Acromioclavicular (AC) joint injuries are common shoulder injuries accounting for up to 41% of all shoulder injuries in football [18]. Injuries to the AC joint commonly result from falling onto the point of the shoulder usually while tackling or being tackled.

What Do Physicians Need to Know About Covering the Game on the Sidelines?

- Football is a collision sport, and physicians covering a game on the sidelines should be prepared to handle traumatic injury. This can include stabilizing fractures and dislocations of the extremities.
- Being cognizant of and able to appropriately identify and stabilize possible cervical spine injury is an important skill for physicians covering football games.
- Physicians should develop a pre-game awareness of resources available on-site and nearby as part of an emergency action plan. These may include availability of paramedics and emergency transportation at the event, accessibility of urgent imaging (X-ray, CT, ultrasound), and readiness of on-site splinting, bracing, and spine boarding.
- Diagnosis and recognition of concussion in the acute setting is a key skill for the sideline physician.
- Prior discussion with coaches, team medical staff (athletic trainers), and administrators regarding best practices of medical care can preclude confusion and possible confrontation during evaluation and management of injury in-game, whether orthopedic or non-orthopedic in nature.

General Rule About Return to Play

- A principle statement when considering return to the football playing field hinges on the athlete possessing a level of function to be able to contribute positively to team play while simultaneously being able to protect themselves in a collision sport.
- Variables involved in this decision may include the athlete's position (interior line has different demands than a skill position, such as wide receiver or running back) as well as the ability to perform sports and position-specific skills adeptly.
- There are certain absolutes that are universal, such as no return to play on same-day with a concussion.
- Return to play after injury resulting in significant time-loss or surgery should include reestablishment of strength and function of the injured body part, consideration of the athlete's mental readiness to return to a contact/collision sport, and restoration of sports-specific skills.

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Gymnastics

57

Stephanie Chu

Key Points

- Majority of gymnastics injuries are sudden in nature and occur in competition versus practice settings. This is thought to be due to exposure rates being higher in competition and better and more protection and padding in practice.
- The most common musculoskeletal injury in club-level female gymnastics is in the lower extremity, specifically the ankle, followed by the upper extremity and then the spine/trunk. The most common area in the spine/trunk is the low back and the common upper extremity locations are the wrist, elbow, and hand/fingers.
- In young male gymnasts, the shoulder is injured most often followed by the wrist and then the ankle, differences between female and male injuries likely have to do with the different apparatuses.
- Comparing men's and women's gymnastics injuries reveal that females are more likely to sustain injuries requiring surgical intervention.
- Gymnastics is both an upper extremity and lower extremity weight-bearing sport frequently requiring athletes to bear significant loads to their upper extremities which can compound their injuries to these joints.
- Lower extremity injuries, ankle ligament sprains, and internal knee derangements are the most common in women's gymnastics and upper extremity, wrist, and shoulder injuries are the most common for men.

Introduction

Gymnastics rises in popularity every 4 years during the Olympics. A basic understanding of the events, equipment, and biomechanics of gymnastics is important in understanding the mechanism of injury and their return to sport following an injury (Table 57.1). However, external factors such as rule change every 4 years, new skills being developed all the time and multiple and very different events make gymnastics unlike any other sport [1]. Research has shown that the majority of female gymnastics injuries are reported in the lower extremity, sprains, strains, and overuse injuries as well as the lower back with most injuries occurring during the floor exercise and the uneven bars [2]. The sport of gymnastics requires a level of flexibility, conditioning, and complete

Table 57.1 Types of gymnastics and events associated with each type

Gymnastics type	Events
Men's and women's acrobatic	Women's pairs
	Men's pairs
	Mixed pairs
	Women's group
	Men's group
Men's artistic gymnastics	Floor exercise
	Pommel horse
	Still rings
	Vault
	Parallel bars
	High bar
Women's artistic gymnastics	Vault
	Uneven parallel bars
	Balance beam
	Floor exercise
Rhythmic Gymnastics (women ONLY)	Rope
	Hoop
	Ball
	Clubs
	Ribbon
Tumbling and trampoline	Double mini-trampoline
	Synchronized trampoline
	Trampoline
	Tumbling

S. Chu (✉)
Department of Family Medicine and Orthopedics, Division of Sports Medicine, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: stephanie.chu@cuanschutz.edu

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body recruitment and presents a unique diagnostic and treatment challenge to the medical practitioner caring for a gymnast or covering a gymnastics event [1].

Injury Epidemiology

Gymnastics is a popular and very injury-prone sport in the United States with more than five million individuals older than 6-year-olds who participate in gymnastics annually (Fig. 57.1). Seventy five percent are female and more than 900,000 of these individuals participate in gymnastics more than 100 days per year. USA Gymnastics (USAG) is the main governing body of competitive gymnastics and according to the USAG safety manual, a gymnastics injury is defined as "any injury sustained during gymnastics participation that results in the gymnast missing any portion of a workout or competitive event" [3]. Injury rates have been reported as 3.6 injuries per gymnast per year with 2.5–3.3 injuries per 1000 h of training and 2.1–8.5 injuries per 1000 athletic exposures [4]. The largest proportion of injuries occurs during floor and vault exercises and involves the lower extremity, whether it is during practice or competition it does not matter. An explanation for increased injuries during competition may be related to athlete fatigue from performing a full routine. A significant difference in men's artistic gymnastics injuries is that they occur more frequently in the upper extremity than the lower [3]. Most studies are on injuries to collegiate or elite gymnasts. A notable study on artistic gymnastics skills and/or routines but not during organized competitions in young children showed that every child (age 5–10 years) that participates in an hour of recreational gymnastics weekly for a year could have an incidence of injury 8.5 times per child per year [3].

What Is Unique About Gymnastics Injuries?

- Many gymnasts are skeletally immature, so growth plate injuries can be more common.
- Complex aerial and acrobatic nature of gymnastics places athletes at risk for catastrophic neck injuries.
- Most injuries are due to trauma occurring during landings both from an apparatus and from the floor exercise.
- Although lower extremity injuries are more common, it is important to remember that gymnasts are upper extremity weight-bearing athletes (Table 57.2).

What Do Physicians Need to Know While Covering a Gymnastics Event?

- Two times more injuries occur during competitions.
- Due to cervical spine and head catastrophic injuries, an emergency action plan needs to be in place prior to the event.
- Injuries that should be attended to as soon as possible include (but are not limited to): concussion, eye injuries, nasal fractures, elbow fractures/dislocations, grip lock injuries, blisters and rips, dislocations, and cervical spine injuries.

Fig. 57.1 Balance beam. (Reprinted with permission from iStock#165054566)

Table 57.2 Common acute gymnastics injuries [3, 5–12]

Lower extremity injuries (most common)	Foot	Lisfranc injury
		Turf toe
		Calcaneal apophysitis (Sever's disease)
		Calcaneal contusion
	Ankle	Lateral ankle sprain
		Os trigonum fracture
		Distal fibula physeal fracture
		Talar fractures
	Knee	ACL tear
		MCL/LCL sprain/tear
		Meniscal injuries
		Patellar subluxation/dislocation
		Tibial tubercle avulsion fracture
	Hip/pelvis	Pelvic apophyseal injuries
		ASIS
		AIIS
		Ischial tuberosity
		Lesser trochanter
		Greater trochanter
		Iliac crest
Upper extremity injuries	Hand	Metacarpal fractures
		Phalanx fractures
	Wrist	Gymnast wrist (distal radial physeal injuries)
		Scaphoid fracture
		TFCC tear
		Scapholunate dissociation
		Scaphoid impaction syndrome
		Grip lock injury (forearm fractures)
		Ulnar abutment
	Elbow	Ulnar collateral ligament injuries
		Medial epicondyle apophysitis
		Elbow dislocation
	Shoulder	Rotator cuff tear
		Labral tears
		Acute shoulder dislocations
Head injuries	Concussion	
	Facial fracture	
	Eye injuries from chalk	
Spine injuries	Cervical spine fracture, subluxation, dislocation	
	Lumbar spine facet syndrome	
	Lumbar discogenic back pain	
Dermatologic injuries	Facial laceration	
	Hand and wrist rips and blisters	

- Equipment that should be considered include pediatric and adult cervical collar, an eye kit, splinting material, crutches in pediatric and small adult adjustable heights, skin care, and equipment for a thorough neurological exam for proper evaluation of a concussion.
- Medical staff will need to be aware of multiple apparatuses going on simultaneously and very different routines occurring at the same time.
- Knowledge of the various landing surfaces is important for medical coverage and a covering physician should be familiar with all the different types.

General Rule About Return to Play

- Any gymnast with a suspected cervical spine injury should be withheld from competition and likely need emergency room attention.
- A concussion should be evaluated thoroughly. Given the acrobatic nature of gymnastics, return to play following a concussion might be difficult due to inherent spinning of gymnastics skills which can exacerbate symptoms.
- A gymnast with any suspected fracture, upper or lower extremity, should not return to competition.
- Taping and bracing of ankle and wrist sprain/strains may afford return to competition.

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Ice Hockey

58

Mark F. Riederer

Key Points

- Ice hockey is a fast-paced, collision sport where up to 90% of all injuries are acute in nature.
- Concussions are one of the most common acute injuries in ice hockey athletes, thus prompt recognition and removal from play is critical.
- Lacerations of the head and face are another common acute injury, and the team physician should be comfortable in laceration repair. The use of full face shields/cages significantly reduce this risk.
- Acromioclavicular joint sprains are the most common upper body injury.
- Sprain of the medial collateral ligament of the knee is the most common lower body injury.

Introduction

Ice hockey is a fast-paced, collision sport played on an ice surface enclosed in a rink enclosed by hard boards and plexiglass (Fig. 58.1). It is becoming one of the most popular sports in North America, with an increase in 143% of participants at all levels in the United States alone between 1990 and 2010 [1–3]. In 2014–2015, approximately 358,000 players under the age of 18 were registered with the USA Hockey program [4]. Additionally, female ice hockey participation has increased sevenfold since 1993 [3, 4]. The International Ice Hockey Federation (IIHF) recognizes 77 countries at the international level. It is played by six players on either team: three forwards (two wingers and one centerman), two defensemen, and a goalie. The objective is to score a puck made out of galvanized rubber past the goalie into a net measuring 180 × 120 cm with the use of a stick. The game is played over three 20-minute periods. Although modern ice hockey athletes wear protective padding including a helmet, shoulder pads, elbow pads, gloves, padded shorts, shin guards, and skates, the very nature of the sport makes the athlete prone to acute injuries. All youth, collegiate, and women's ice hockey rules require the use of a full face shield or metal wire shield that protects their face from injury. In men's rules, the use of any type of face protection is optional. Risk factors to acute injuries include body checking, unintentional collisions, rapid, high-velocity changes in skating, and collision with the boards, stick, or puck [5].

Epidemiology of Acute Injuries

There have been a number of recent studies reporting injury rates (IR) in men's, women's, and junior international ice hockey participation. The overall injury rate for men's ice hockey was 14.2 per 1000 player-games (52.1 per 1000 player-game hours), 6.4 per 1000 player-games (22.0 per 1000 player-game hours) for women, and 11.0 per 1000 player-games (39.8 per 1000 player-game hours) for juniors [5–7]. As expected with most sports, injuries occur more commonly during competition than practice, and with higher levels of competition. Between 1990 and 2006, the annual incidence of injury in youth ice hockey athletes between 9 and 14 increased 163% and 85% in those 15–18 years of age [8]. From 2008 to 2013, boy's high school ice hockey had an IR of 23.2 per 10,000 athlete exposures [9]. Acute injuries in youth hockey are dramatically influenced by leagues that prohibit body checking. Many studies have linked body checking to higher rates of injury, especially concussions [10, 11]. Currently in the US and Canada, body checking is not allowed in those 12 years and younger (Mites/Novice, Squirt/Atom, Pee Wee leagues). The average IR for concussion was 1.1 per 1000 player-games at the international hockey level, and 1.58 per 1000 exposures within a youth hockey sample. However, the rates were 2.4 times higher in 12–14 years of age than 15–18 years of age [12].

M. F. Riederer (✉)
Department of Orthopaedic Surgery, University of Michigan, C.S. Mott Children's Hospital, Ann Arbor, MI, USA
e-mail: mriedere@med.umich.edu

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Fig. 58.1 A national hockey league game

Injuries of the Head and Face

- Injuries to the head and face are the most common anatomic sites of injury in men's and junior ice hockey international leagues (40% and 39%, respectively) and third most common in women (21%) [5–7].
- Concussions in women are the most common head and face injury, accounting for 74% of these injuries. Unintentional collision and body checking are the most common mechanisms of injury [6, 16].
- In the U18 junior league, concussions also represented the most common head injury, accounting for 46% of head and face injuries. Checking to the head and body checking were the most common mechanisms of injury [7, 16].
- In men's leagues, concussions were an infrequent diagnosis of head and face injuries, accounting for only 10% of all injuries [5, 16].
- In men's and junior leagues, lacerations represented 74% and 44–80% of all facial injuries. In women's leagues, facial lacerations were only 89%. This is due to the fact that full face shields are required for all women's leagues and U18 leagues [5–7].

Injuries of the Upper Body

- The shoulder is the most common anatomic location for acute upper body injuries in ice hockey (50% in men, 58% in junior, and 33% in women of upper body injuries [5–7].
- Elbow and wrist injuries account for approximately 20% of the upper body injuries [5–7].
- Sprains to the acromioclavicular (AC) joint are the most frequent diagnosis, accounting for 51% in men's, 59% in junior, and 50% in women's upper body injuries [5–7].
- Subluxations/dislocations of the glenohumeral (GH) joint were an infrequent diagnosis in elite Swedish ice hockey athletes, representing only 8% of all injuries. However, they represented about 40% of the upper body injuries in men's leagues, second only to AC joint sprains [5, 13].

Injuries of the Lower Body

- The knee is the most common location of acute lower body injury in ice hockey, representing 50% of the acute lower body injuries in men's, 33% in juniors, and 49% in women's leagues. Collisions are the most common mechanism of injury [5–7].
- Sprains to the MCL are the most common diagnosis, representing 57% in men's, 40% in juniors, and 37% in women's leagues [5–7].
- ACL ruptures are infrequent injuries, representing 11% in men's, 6% in juniors, and 11% in women's leagues [5–7].
- Ankle injuries are the second most common lower body injuries. Due to the fairly rigid ice skate covering around one-third of the lower leg, lateral ankle sprains are uncommon. However, high ankle sprains do occur in higher frequency if there is an eversion and dorsiflexed force upon the ankle.
- Hip adductor strains represent only about 9% of all acute injuries. The most common mechanism is from noncontact injuries [14, 15].

What Is Unique About Ice Hockey Injuries?

- Most ice hockey injuries are a result of intentional or unintentional collisions.
- Lacerations, one of the most common acute injuries in ice hockey, are frequently a result of contact with the stick, puck, or skate.

What Do the Physicians Need to Know While Covering an Ice Hockey Event?

- Physicians need to have suture kits and supplies available to quickly repair lacerations, a common acute injury in ice hockey athletes.
- Physicians must be prepared for catastrophic head and neck injuries, and have an Emergency Action Plan for safely removing injured athletes on an ice surface.
- Physicians should understand how to recognize concussions in ice hockey athletes, promptly removing them from play.

General Rules About Return to Play in Ice Hockey

- Lacerations are commonly repaired immediately for athletes to return to play later in the game.
- Athletes with minor sprains and contusions can generally return to play with very little time off sport.
- Athletes with concussion should go through standard return-to-play protocol (Table 58.1).

Table 58.1 Most common acute injuries in ice hockey by anatomic location

Anatomic site	Diagnosis
Head/face	Concussion
	Facial laceration
Upper body	AC joint sprain
	GH joint subluxation/dislocation
Lower body	MCL sprain
	Hip adductor strain
	High (syndesmotic) ankle sprain

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Ice Skating

59

Rajwinder S. Deu

Key Points

- The majority of participants are female.
- Majority of acute injuries are secondary to falls and occur in pairs skating, ice dancing, and synchronized skating.
- Singles skaters commonly suffer from contusions to the knee and hip from falls.
- The female partner is at risk of suffering a head injury from a fall during a lift or throw.
- The close proximity of ice dancers places them at risk for tripping and suffering a laceration, contusion, or fracture.
- The high number of skaters and close proximity place synchronized skaters at high risk for traumatic injuries due to collisions.

Introduction

Figure skating continues to be a popular sport. In 2015–2016, the United States Figure Skating Association (USFSA) reported 181,703 members and 700 skating clubs [1]. Women make up the majority of participants, forming 74% of the membership [1]. There are four major disciplines: singles skating (Fig. 59.1), pairs skating, ice dancing, and synchronized skating. Each discipline requires different maneuvers and expose the skater to different risks. Multiple factors can contribute to injury including the boot, age and developmental stage of skater, technique, training regimen, environmental factors, and scoring system that rewards high-risk moves [2, 3]. Elite figure skaters train between 4 and 7 h per day (2–4 h in on-ice training plus 1–3 h in off-ice training), 6 days per week, and up to 11 months per year [2, 4].

Fig. 59.1 Single skating. (Reprinted with permission from iStock # 147623658)

Injury Epidemiology

Singles Skating

Most injuries in singles skaters are overuse injuries. Acute knee injuries are uncommon likely due to lack of fixation of

R. S. Deu (✉)
Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: rdeu1@jhmi.edu

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the blade on the ice and the protective effect of co-contraction of the quads and hamstrings when the skater lands gliding backwards [2, 5]. Contusions to the knee and hip are common due to repetitive falls incurred while learning new jumps [3]. For those landing on an outstretched arm, fractures involving the distal radius or scaphoid are possible [3].

Pairs Skating

In pairs skating, acute injuries occur more often than overuse [4]. Elements unique to pairs skating include lifts, throw jumps, twist lifts, individual and paired spins, and death spirals. Many injuries are secondary to falls. Competitors are becoming more daring with their lifts including complicated overhead lifts and triple and quadruple throw jumps [4]. This places the female partner at risk for suffering a concussion or craniofacial fracture [5].

Ice Dancing

In ice dancing, acute injuries also occur more often than overuse [4]. This discipline requires intricate footwork on deep edges at a high speed. It does allow for small lifts and spins, but the pair must be in close proximity for majority of the time placing them at risk for tripping and subsequently suffering a laceration, contusion, or fracture [4, 5].

Synchronized Skating

Synchronized skating is a team sport and can range from 8 to 20 skaters. Due to the high number of skaters and their close proximity, there is high risk for traumatic injuries secondary to collisions or falls. Highest risk of injury occurs during the intersection, block, and group lift elements [6, 7]. Dubravcic-Simunjak found that 42.4% of female and 42.9% of male synchronized skaters suffered an acute injury at some point during their career [7]. This was further broken down into different body parts: lower extremity 40.5%, upper extremity 33.0%, head 19.7%, and trunk 6.8% [7]. The types of injuries include fractures, contusions, hematomas, lacerations, sprains, concussions, and ligament injuries. The number of laceration injuries due to the blade and toe pick is greater in synchronized skating [6]. Knee injuries occur more often in synchronized skating due to collisions.

Figure Skate

The figure skate is composed of a leather boot and a blade. Boots tend to be stiff and can cost $200–$700 per pair [2]. The blade has two edges, is curved from front to back, and contains a large front toe pick. Ice dancers and synchronized skaters typically have a shorter blade behind the heel. The boot is high-heeled with the skater's ankle in a plantar flexed position allowing minimal dorsiflexion. Due to the stiffness of the boot, figure skaters have weak peroneal muscles, which places them at higher risk for ankle sprains. Most of these injuries occur during off-ice training [2, 3].

What Is Unique About Ice Skating Injuries?

- Ice skaters do not wear any protective equipment (helmet or pads) and are at risk of serious injury from falls onto the hard ice surface.
- Acute knee injuries are uncommon likely due to lack of fixation of the blade on the ice and the protective effect of co-contraction of the quads and hamstrings when the skater lands gliding backwards [2, 5].
- Contusions are common due to repetitive falls onto the hard ice surface while learning new jumps.
- Figure skaters tend to have weak peroneal muscles due to the stiffness of the boot placing them at risk for ankle sprains during off-ice training [2, 3].

What Do Physicians Need to Know While Covering Ice Skating on the Sideline?

Due to the frequency of lacerations, wound care equipment including suture kits should be available [8]. In addition, it is necessary to have resuscitation equipment, spine boards, and cervical collars [8]. The physician should have a clear line of sight and know where the entrance is should they need to go onto the ice. Shoes with a rubber sole such as a tennis shoe will provide better grip on the ice as compared to a dress shoe with a leather sole. Each organization has developed specific protocols for on-ice emergencies that allow for evaluation of the skater by medical personnel including athletic trainers and physicians. The International Skating Union (ISU) dictates that an injured skater will be evaluated by the team physician or the event's chief medical officer prior to returning to competition [9].

General Rule About Return to Play

There are multiple factors to consider when determining return to play (RTP). Of foremost importance is to do no harm. The key is to ensure that the athlete poses no undue risk to themselves or the safety of other participants [10]. RTP evaluation should include range of motion and functional movement tests assessing agility, balance, strength, and proprioception [11, 12]. There should be determination of psychological confi-

dence and mental readiness to participate [11, 12]. The athlete should be able to perform safely with modifications to their equipment including bracing and orthoses. Once these criteria are met, the athlete can be cleared to return.

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60 Lacrosse

Sameer Dixit

Key Points
- Lacrosse is the fastest growing sport in the United States.
- Men's and women's lacrosse are different sports with different rules.
- Men's and women's lacrosse use different equipment.
- Lower extremity injuries are the most common injuries in lacrosse.
- Head injuries are the second most common injury in lacrosse.

Introduction

Lacrosse is the fastest growing sport in the United States at all levels [1]. The history of lacrosse is traced back to stickball games played by Native Americans and thus it is considered the oldest team sport in North America [1]. Men's and women's lacrosse are different games secondary to deviations in rules and equipment between the two sports (Fig. 60.1) [2]. Body contact and stick checking are allowed in the men's games, creating an environment more conducive to injuries seen in contact sports. As such, protective gear is an important part of the men's game. In the women's game, contact is technically not allowed and there is a 7 inch protective "bubble" around the head which opposing players are penalized if they enter. Additionally, the pocket of the stick is shallower in the women's game. While eyewear was mandated in 2004 in the women's game, helmets are not allowed for field players [1, 2]. Although women's lacrosse is considered a noncontact sport, a significant amount of contact can still occur, and this is an issue the covering physicians should be aware of.

S. Dixit (✉)
Departments of Orthopaedics, Medicine, and Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Lutherville, MD, USA
e-mail: sdixit3@jhmi.edu

Injury Epidemiology

Although lacrosse is a rapidly growing sport, little data exists regarding the epidemiology of injury in the sport [3, 4]. Although lacrosse is considered a collision sport, injury rates are lower in lacrosse than in football and ice hockey [5]. The most common injuries in the sport are lower extremity injuries, specifically ankle sprains, and knee ligamentous injuries [2–4]. Upper extremity injuries are seen more in the men's game [2, 3]. Head injury is the second most common injury [2–4]. Contusions, abrasions, and lacerations are also not uncommon [2–4]. Between studies, how significance of an injury is determined varies perhaps affecting the difference in reported rates between studies.

Lower Extremity Injuries

Lower extremity injuries are the most common injuries in lacrosse. Multiple studies have shown ankle sprains to be the most common injury in lacrosse at multiple levels [3–5]. Although common, ankle sprains generally do not result in significant time loss or complications. More significant knee ligamentous injuries, specifically ACL injuries are also significant injuries in lacrosse and can occur as both contact and noncontact injuries [3–5]. Although ACL injuries are commonly known to have a higher incidence in women than men in comparable sports, there appears to be equal distribution of these injuries between men and women in lacrosse [6, 7].

Head Injury

Rates of concussion in both men's and women's lacrosse are significant [3, 4]. Concussions in the men's game are

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Fig. 60.1 (**a**, **b**) Men's and women's lacrosse. (Reprinted with permission from iStock photo ID:96339432 & 157475751)

generally via player to player contact, whereas in the women's game they are more commonly attributed to stick contact with the head. Recently, Kerr et al. described that in games and practice, respectively, 71.4% and 64.3% of head/neck injuries were concussions [3, 4].

Other Injuries

Upper extremity injuries including AC joint sprains and fractures of the hand are also seen commonly in the men's game [3]. Although not well studied or reported in the literature, heat illness and nutritional issues are probably underestimated and thus appropriate vigilance should be considered when covering games. Case reports of commotio cordis have been reported in men's lacrosse but not in the women's game, thus the emergency action plan (EAP) should be prepared for this rare but potentially catastrophic injury.

Injury Prevention

Injury prevention in lacrosse is primarily a function of proper preparation in practice as well as following the rules of the game as they are intended. Specifically, head injury and lacerations can be avoided if appropriate rules of contact are enforced in the men's game and if contact is avoided as intended by the rules of the women's game. It is hypothesized that as lacrosse has grown, more novice participation had led to an increase in injury [8]. US Lacrosse and the NCAA regulations mandate the use of protective equipment for the head and upper extremity [5].

Equipment

The lacrosse helmet (Fig. 60.2) used in the men's game is different from that used in other sports (e.g. football), and it is imperative that the covering physician become familiar with it prior to coverage. All helmets used must be NOCSAE certified. In the event of a cervical spine injury, the medical team must have familiarity with the helmet as well as other upper extremity equipment. There has been discussion regarding adding a helmet to the women's game, but controversy exists regarding possible ramifications of adding a helmet to the game including an increase in injury patterns of mild traumatic brain injury.

Balls must be of NOCSAE standards as well. Sticks are made of wood, laminated metal, or synthetic material and are a source of significant contact in the men's game and of inadvertent contact in the women's game (Fig. 60.3). Dislodgement of the ball from the stick is an important part of both games, in the men's game this is achieved via stick and body checks while in the women's game only stick checks are allowed [2].

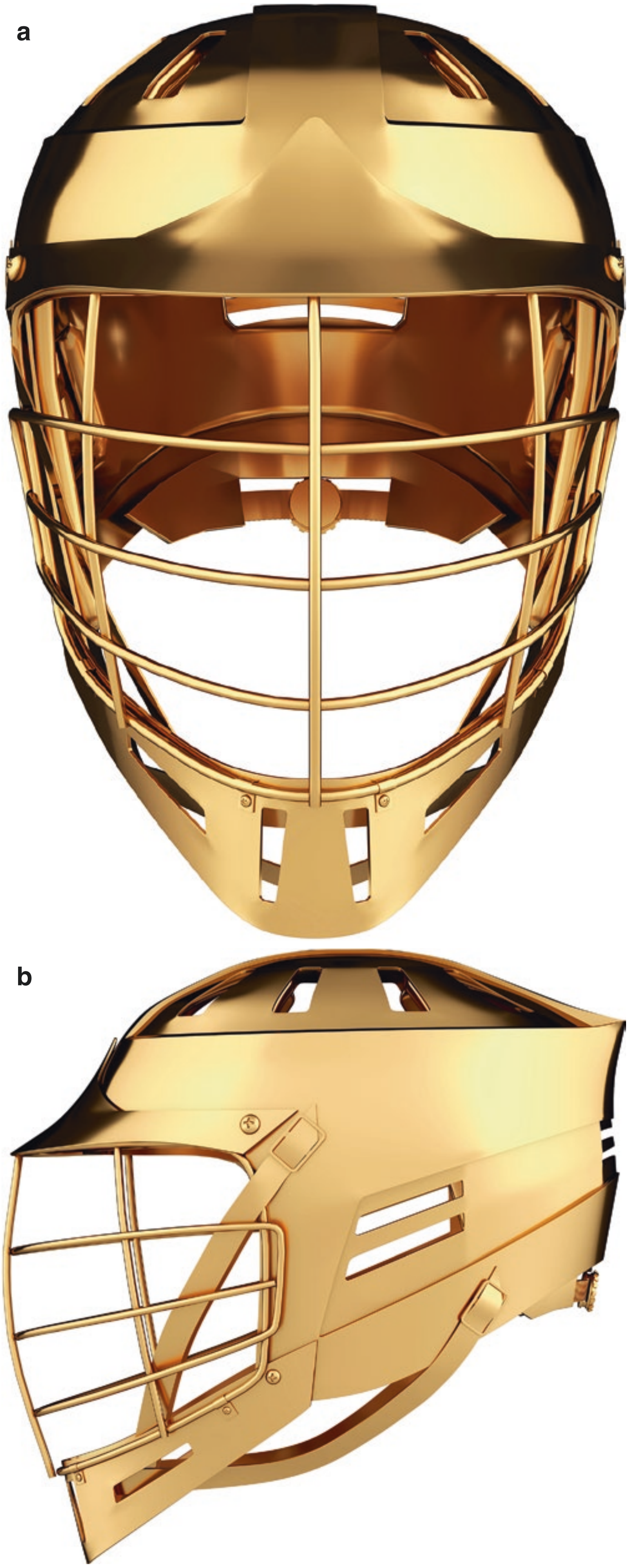

Fig. 60.2 (**a**, **b**) Lacrosse helmet. (Reprinted with permission from iStock photo ID:883069962 and 883070118)

Fig. 60.3 Men's and women's lacrosse sticks. (Reprinted with permission from iStock photo ID:186864702)

In the men's game, additional required equipment include shoulder pads, mouthguards, and arm and rib pads. In the women's game, protective eyewear and mouthguards are required. In both sports, goalkeepers wear appropriate protective equipment as expected.

What Is Unique About Lacrosse Injuries?

- Men's lacrosse is a collision sport with specific required protective equipment for the head and upper extremity.
- Women's lacrosse, while not considered a collision sport, has a significant amount of inadvertent contact.
- Injuries can occur from both contact and noncontact mechanisms.
- Equipment is specific to the men's and women's games and familiarity with this equipment allows for better coverage.

What Do the Physicians Need to Know While Covering a Lacrosse Game?

- Standard concussion protocols should be applied.
- Appropriate equipment for wound and laceration care should be available.
- EAP including consideration for commotio cordis, cervical spine injury, and AED placement should be in place.
- Medical team should be able to do bracing and immobilization for injuries to the joints.
- Consideration and planning for emergent plain radiography should be in place for fracture evaluation.
- Physicians should be familiar with equipment specific to lacrosse.

General Rule About Return to Play

Injuries should always be considered on an individual basis and return to play is based on an athlete's ability to participate at a safe and effective level. Lacrosse allows for frequent substitutions, and thus player movement in and out of the game can be quite fluid. This generally allows for ample time for evaluation of an athlete as well as the opportunity to put the player back in participation for limited portions of the game if that is an appropriate option.

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Marathons

61

Brian J. Krabak and Eric T. Chen

Key Points

- Medical encounters during these events can be categorized into either musculoskeletal, dermatologic, or medical issues.
- The most common acute musculoskeletal injuries are muscle strains followed by ankle and knee sprains.
- Dermatologic issues occur frequently, with blisters being the most common complaint.
- Cardiac arrest, exercise-associated hyponatremia, and severe heat or cold illness are potentially life-threatening conditions that may occur during marathon events.
- Runners who exhibit limping or altered gait may need to be removed from competition.
- Marathon race courses are large and present a unique challenge that requires thorough logistical planning and preparation.

Introduction

The marathon and half-marathon (Fig. 61.1) are long distance running events that require competitors to complete standard distances of 42.195 km and 21.1 km respectively. From 1976 to 2014, the popularity of marathons in the United States has grown rapidly from 25,000 to 550,600 participants annually [1]. More recently, the popularity of half-marathons in the United States has also grown from 303,000 participants in 1990 to over two million in 2014 [1]. This growth in popularity has expanded participation to include runners of all ages, fitness levels, and racing experience.

Injury Epidemiology

Approximately 1% of all half-marathon participants and 8% of marathon participants will undergo a medical encounter during a race (Table 61.1). The majority of medical encounters can generally be categorized as either musculoskeletal injury, dermatologic issues, or medical illness (Table 61.2).

Musculoskeletal Injury

- While far less common than overuse injuries, acute trauma during the half or full marathon may present as muscle strain, ankle or knee sprains, and injuries related to falls such as acute fracture or skin trauma.
- Muscle strains are the most common acute injury, occurring at a rate of 2.7 per 1000 entrants, followed by sprains at 0.25 per 1000 entrants [2].
- Overall, the knee is the most commonly injured joint, followed by the ankle [3–5]. However, when considering acute injuries alone, acute ankle sprains have a higher incidence than acute knee sprains, 10–15% compared to 5%, respectively [6].
- Common overuse injuries may present on race day due exacerbation from repetitive motion. The most common causes of knee pain among half and full marathon runners are patellofemoral pain syndrome (PFPS) and Iliotibial Band Friction Syndrome (IBFS). (Table 61.3).

B. J. Krabak (✉)
Department of Rehabilitation, Orthopedics, and Sports Medicine, University of Washington and Seattle Children's Sports Medicine, Seattle, WA, USA
e-mail: bkrabak@uw.edu

E. T. Chen
Department of Physical Medicine and Rehabilitation, University of Washington, Seattle, WA, USA
e-mail: ericch1@uw.edu

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Fig. 61.1 A half-marathon race

Table 61.1 Comparison of the overall rates of injury and illness in distance running events [2, 3, 5, 9, 12, 28, 32–34]

Overall injury rates	Half-marathon (16–21 km)	Marathon (42 km)
Impacting performance or requiring medical encounter (incidence)	0–4%	1–18%
Self-reported after event (incidence)	40%	65%
Pooled incidence per systematic review	1% (N = 97,490)	8% (N = 82,776)
One year prevalence per systematic review	32% (N = 6206)	52% (N = 1650)

Table 61.2 Estimated proportion of total injuries by type for various distance running events [5, 35]

Types of injuries or illnesses	Half-marathon (%)	Marathon *(%)
Musculoskeletal	46	17
Medical	27	63
Dermatologic	27	21

Table 61.3 Knee injuries: estimated incidence of patellofemoral pain syndrome and iliotibial band fraction syndrome in distance running events [7, 8, 11, 28, 36, 37]

Knee injury	Half-marathon (%)	Marathon (%)
Patellofemoral pain syndrome	6–13	10
Iliotibial band friction syndrome	11	14

- Bone injuries can range from stress reactions to acute fracture. The most common cause of bony pain that may present on race day is Medial Tibial Stress Syndrome, also known as “shin-splints.” The 1-year prevalence of MTSS in half and full marathon runner is 10% and 20%, respectively [7, 8].
- Acute fractures are rare and most commonly related to accidental fall. Stress fractures are more common but still account for less than 1% of encounters in half and full marathons [2, 5]. The tibia is the most common site of stress fracture [6].

Dermatologic Issues

- Skin conditions have an estimated incidence of 21–27% in half and full marathons [2, 5].
- Blisters are the most common of all complaints related to skin. The estimated incidence of blisters ranges from 4–9.4 per 1000 runners in the half-marathons [9] to 3.8–17 per 1000 runners in the full marathons [2, 9].
- Dermatitis due to repetitive friction, also known as chafing, can affect between 2% and 16% of marathon runners [10] and accounts for up to 9% of all medical encounters during a race [11].
- Subungual hematomas are less common, and have been reported in 3% of marathon runners [11].

Medical Illness

Medical conditions, as opposed to purely musculoskeletal conditions, account for up to 63% of all marathon medical encounters [2, 12]. A variety of medical illnesses can present on race day and early recognition of serious illness is critical for preventing morbidity and mortality.

Exercise-Associated Collapse (EAC)

- EAC is a general term used for any illness that causes a runner to collapse during or after a race.
- While post-finish line EAC is generally considered a benign entity, EAC prior to the finish line is associated with more serious medical conditions including cardiac arrest, heat stroke, exercise-associated hyponatremia, and hypoglycemia [13].
- The rates of EAC per 1000 runners are 1.06 in half-marathons and 11.25 in marathons [11, 14].

Cardiac Arrest

- Cardiac arrest is a rare occurrence in marathons. However, it is a life-threatening condition that should be recognized urgently.
- The incidence of cardiac arrest in marathoners is estimated to be 1.0 per 100,000. Incidence of cardiac arrest during the marathon is significantly higher than during the half-marathon [15].
- Male runners are at a significantly higher risk of sustaining a cardiac event as compared to their female counterparts [15].
- Initiation of bystander-administered cardiopulmonary resuscitation and an underlying diagnosis other than hypertrophic cardiomyopathy are strong predictors of survival [15].

Exercise-Associated Hyponatremia (EAH)

- EAH is another potentially life-threatening medical condition that can present on race day as a collapsed runner or a runner with altered mental status and neurologic derangement.
- Most runners with hyponatremia by laboratory exam are clinically asymptomatic; the incidence of some form of EAH has been reported as high as 4% in half-marathons [16], between 0% and 22% in marathons [17–19].
- Runners who actually required medical attention for symptomatic hyponatremia is significantly lower; approximately 0.23 per 1000 per entrant [20].
- Other risk factors for developing EAH include slower race pace, female sex, and low body weight [18, 21].

Gastrointestinal (GI) Complaints

- GI Complaints are a common complaint among long distance runners. Studies with varying methodologies have reported a wide range of incidence rates from 3% to 87% [11, 22, 23].
- One of the most common GI complaints is exercise-related transient abdominal pain (ETAP), more commonly known as cramps or "side-stiches." ETAP has been reported to occur in up to 69% long distance runners [24, 25], and may be related to an individual's level of conditioning.
- Most other GI complaints can be categorized as being related to either the upper or lower GI tract. Causes of upper GI complaints include reflux, heartburn, belching, bloating, nausea, or vomiting. Lower GI complaints may include flatulence, urge to defecate, diarrhea, or intestinal bleeding.
- Risk factors for GI symptoms include having a prior history of GI symptoms [26], female gender [27], younger age, recent illness [28], race-day weight loss [29], and increased carbohydrate intake [26].
- GI bleeds can also occur with some frequency in long distance runners, although do not typically present until well after the race is over. Some studies have found 16% of marathon runners reporting bloody diarrhea after the race [13].

Heat- and Cold-Related Illness

- Runners' abilities to regulate body temperature can be challenging during marathon races. Increased temperature, humidity, sun exposure, ages <15 yrs or >65 yrs and lack of acclimatization are risk factors for heat illness [30].

- Studies have reported incidence of heat illness during half-marathon and marathon races to range between 0–0.5/1000 runners and 0–1.4/1000 runners, respectively [9].
- Prolonged exposure to cold temperatures can also put runners at risk of hypothermia. The incidence of hypothermia ranges from 0–1% to 0–8% in half-marathon and marathon runners, respectively [9].

Race Day Preparation

Organizing Your Medical Team

- Organizing your medical team starts with identifying an experienced medical director. The medical director is the individual responsible for overseeing all aspects of planning and operations of the medical team on race day.
- In addition to other appropriately trained physicians, the medical team may also include athletic trainers, emergency medical technicians, podiatrists, paramedics, physical therapists, massage therapists, and nurses.
- Staffing numbers will depend on the size of the event. Generally, one can plan for 1–2 physicians per 1000 runners. Knowledge of participant demographics and historical data of medical needs at past races will be helpful in planning the size and make-up of the medical team [31].

Supplies

- In addition to basic first aid and medical supplies, there are several items that may be particularly useful in the marathon setting.
- Adequate blister care supplies including elastic tape, moleskin, and blister pads are needed given the high frequency of skin issues during half and full marathons.
- Automatic external defibrillators (AEDs) should ideally be available throughout the race course in the event of sudden cardiac arrest.
- Point of care blood chemistry analyzers (e.g. i-STAT) are useful when hyponatremia, hypoglycemia, or other metabolic abnormalities are suspected.
- Rectal thermometers are essential for measuring accurate core temperature when heat or cold illness is suspected.

Logistics

- The main medical tent should be established and clearly visible near the finish line. Most of the medical team should be stationed here, approximately 75%.
- The main medical tent should consist of a triage area, general medical area, and intensive medical area. A physician with training in sports medicine or emergency medicine should be designated to perform triage duties for runners presenting to the main medical tent.
- In general, medical aid stations should be established approximately every 2–3 km on the race course.
- Water stations can be set up alongside medical aid stations, but these should remain physically separate to respect space and avoid congestion in and around the medical aid stations.

Communication

- Two-way radio or cellular communications can be used to maintain open lines of communication between the main medical tent, on-course medical aid stations, and mobile medical team members on patrol.
- The chain of command and protocol for communication on race day should be distributed to all medical team members prior to race day.

Transportation

- Endurance events can involve numbers of participants and spectators on the order of thousands to hundreds of thousands. Races of this magnitude often require closing streets and shutting down of thoroughfares to accommodate for the massive numbers of runners and spectators during the event.
- Medics on bike patrols can be used to monitor for down or distressed runners on the race course in between aid stations.
- Designated road cars and mobile medical vans can be used for transit of supplies and noncritically ill or injured runners to and from the race course and main medical tent.

What Is Unique About Marathon Injuries?

- Most musculoskeletal injuries in marathon runners are due to overuse injuries. However, acute injuries, such as sprains, strains, and acute fracture may also occur.
- Dermatologic issues are particularly common among marathon runners.
- Exercise-associated collapse is relatively unique occurrence to marathon participants and may reflect a benign or serious medical condition.

What Do the Physicians Need to Know While Covering a Marathon Event?

- The size and physical area that a marathon race course covers presents a unique logistical challenge in allocating medical resources on race day.
- There are typically much fewer runners requiring medical assistance early on in the race than in the later stages, and medical team members should be distributed appropriately.
- Cooperation with local fire and police departments is essential and an ambulance should be available and on call at the main medical tent in the event of ill or injured runner requiring transfer to a higher level of care.

General Rule About Return to Play or Continue the Race?

- If a muscle strain causes limping or altered gait, one should consider removing the athlete from competition to avoid further injury. Athletes with suspected full thickness tears of a muscle should be removed from competition and referred for further evaluation.
- Athletes who sustain an acute ankle sprain and present with instability, ecchymosis, or high suspicion for fracture (based on Ottawa ankle rules) should be removed from competition.
- Any athlete with a suspected acute fracture should be held from competition immediately.

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62 Martial Arts

Vicki Nelson and Robert Masocol

Key Points

- Martial arts are a diverse group of disciplines with injury rates varying greatly depending on style.
- Injuries are uncommon among practitioners that do not participate in combat-based competition or sparring.
- Common injuries include contusion, abrasion, sprains, and strains.
- Team physicians should familiarize themselves with the rules of competition and martial arts style to anticipate injuries.

Introduction

Martial arts comprise a group of codified combat-based disciplines practiced throughout the world for sport, self-defense, and physical and emotional fitness. The martial arts are diverse but generally can be divided stylistically into those that emphasize striking (such as karate and taekwondo), grappling (such as judo or jujitsu), and mixed martial arts that blend several techniques. Martial arts practice is based in the repetitive practice of stylized movements and techniques without contact. Some practitioners progress to include contact-based practices including combat sparring or competition. Martial arts have been shown to improve muscle strength and coordination, balance, and flexibility as well as mental benefits on cognition and self-esteem.

V. Nelson
Department of Family Medicine, Greenville Health System, Greenville, SC, USA
e-mail: Vicki.Nelson@PrismaHealth.org

R. Masocol (✉)
Department of Family Medicine, Greenville Health System/University of South Carolina School of Medicine, Greenville, SC, USA
e-mail: Robert.v.masocol@kp.org

Disciplines

Karate is a striking martial art that originated in Japan based on self-defensive blocks and quick strike offensive attacks (Fig. 62.1).

Taekwondo is a striking art form that originated in Korea. It is an Olympic sport. Taekwondo is based heavily on kicks as well as hand techniques and takedowns.

Judo originated in Japan and has a basis in grappling focused on throwing and takedowns. It is an Olympic sport.

Mixed martial arts (MMA) includes components from several other combat-based sports including striking, grappling and takedowns. A bout is won by the opponent's submission or knockout/takedown or determined at the end of a match by the judge.

Brazilian Jiu-jitsu (BJJ) originated in Brazil and is based on grappling and the use of joint locks to submit an opponent to achieve victory.

Aikido is a Japanese martial art focused on redirecting an attack away from the practitioner.

Krav Maga originated in Israel and is focused on real-life combat scenarios and defending against attack.

Muay Thai is a Thai martial art focused on powerful kicks and knee and elbow strikes.

Kung Fu originated in China and focuses on hand/arm strikes, kicks, and weapons training.

Tang So Do is a Korean martial art similar to Taekwondo and Karate.

Injury Epidemiology

There are more than 100 martial arts styles practiced worldwide. Approximately 10–20 million people in the United States participate in martial arts including more than 6.5 million children. Martial arts injury is difficult to quantify due to a lack of reporting and paucity of studies [3]. Available studies also offer wide variability in definition of

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Fig. 62.1 Karate

injury, setting, and martial arts style. Martial arts accounted for more than 35,000 emergency room visits in 2011 [1]. In traditional martial arts practices, estimates of injury risk range from 29.9 to 133 per 1000 athlete exposures with higher risk, 85.1–228.7 per athletic exposure, in mixed martial arts [13, 16, 17].

Types of Injuries Most martial arts injuries are acute musculoskeletal injuries including contusion, sprains, strains, and abrasions. Sprains and strains are most common in the lower extremity including ankle sprain, hamstring, or groin strain (Table 62.1). Fracture constitutes at least 10% of injuries with toe and finger fractures being the most common. Chronic or overuse injuries are rarely reported. The risk of serious injury is low but neck injury, dental injury, and concussion are most frequent. This risk is higher in disciplines that emphasize head contact. Death is extremely rare but there are case reports of death after head, neck, and chest traumas.

Table 62.1 Region of martial arts injuries by sport [17]

Sport	Most common injury	Second most common injury	Third most common injury
Karate	Lower extremity	Upper extremity	Trunk
Taekwondo	Lower extremity	Upper extremity	Head/neck
Judo	Upper extremity	Lower extremity	Head/neck
Mixed martial arts	Head/neck	Upper extremity	Lower extremity

Sex The number of female martial arts participants, particularly among youth and adolescents, continues to increase, and the male predominance in martial arts is narrowing with recent estimates that about 40% of participants in the United States are female. Males are more frequently seen for injury [16]. However, most studies of injury epidemiology have not had significant female participation and

this may be a representation of a historically male-dominated sport rather than risk differences attributable to gender [2, 16].

Age Age has not been a reliable predictor of injury risk. Studies suggest that younger participants are at lower risk for injury [17]. However, these results are confounded by the role of exposure time, competition, and skill levels.

Experience Experience and hours of participation appear to associate with higher rates and more severe injury. Across five styles, individuals with more than 3 years of training had twice the risk of injury than less-experienced participants [17]. Injuries that result from delivering rather than receiving a blow are typically associated with poor technique.

Setting The risk of injury is related to the amount of time spent on full-contact or limited-contact training or competition. A contact-based competition setting is associated with lower risk than training but higher severity of injury. Noncontact practices focused on technique have a lower risk of injury.

Weight Weight has not been a reliable predictor of injury across disciplines. Lightweight judo participants have a higher incidence of injury, while heavyweights had more severe injuries [9]. Muay Thai kickboxing heavyweights have a higher incidence of injury [8].

Protective Equipment Utilization of safety equipment for sparring or competition is determined by the individual style of martial arts practiced. A mouthguard and protective cup is utilized in most combat-based styles. Several styles will allow or require head and body gear for sparring or competition. Hand and foot padding is required for some styles such as karate. Use of a mouthguard has correlated with fewer dental injuries during sport [10]. No protective equipment has correlated with reduction of concussion, neck injury, or fracture. MMA generally does not require protective gear, but participants may wear lightweight gloves with exposed fingers. Use of protective gear when appropriate should be encouraged and may reduce the risk of contusion, abrasion, and laceration. The protective equipment required varies by sport. The US National Collegiate Taekwondo Association requires use of foam head gear, shin protectors and forearm guards, open-finger gloves, groin protection (males), and mouthguard. The US National Karate Federation similarly requires foam shin/foot protectors, groin protection (males), and mouthguard but in contrast requires body protector, chest protector (females), foam gloves, and optional foam head gear. In contrast, Olympic judo does not require protective gear.

Discipline

The rate and type of injuries experienced vary greatly by discipline (Table 62.2).

Karate

Much of karate practiced in the United States is time spent in noncontact training. In youth competition, focus is placed on technique just short of contact. As such, injury rates are lower than seen in other martial arts with most injuries (90%) occurring during sparring practice [2, 18]. Injury risk is low in competition and severe injury is extremely uncommon (<1%) [7].

Taekwondo

In comparison to other disciplines, more injuries are seen which require time away from training, and more practitioners report multiple injuries [6, 17]. Head injury and concussion are also more frequent than in other disciplines [14]. The injury rate is three times higher than Shotokan karate. Increased injury risk may be related to the emphasis on kicking techniques, which provide a more forceful blow to the opponent.

Judo

Sprains, strains, and contusions remain the most frequent injuries, with most injuries resulting in less than one week of time loss from training [15]. Injury to the upper extremities is more common in judo than in striking styles such as karate and taekwondo. Injury is most likely while being thrown. Inclusion of joint-locks and takedowns may increase the likelihood of dislocations, subluxations, and fractures. A study of national-level South Korean athletes reported four injuries per athlete annually [9].

Table 62.2 Rate of martial arts injuries by sport [17]

Sport	Injury (%)
Taekwondo	59.2
Aikido	51.1
Karate	29.8
Kung Fu	38.5

Mixed Martial Arts

Injury risk is higher than in other martial arts and more similar to boxing [11]. The most frequent injuries reported were lacerations, fracture, head injury, or concussion. Head injury, neck injury, and concussion are more frequent than in other martial arts.

Losing fighters are at 2.53 times the risk of injury relative to winning fighters. One study found match-ending head trauma in 31.9% of matches and match losers sustain an average of 18.5 strikes, 92.3% of them to the head, in the final 30 s of the match. Age, weight, and fight experience are not reliable predictors of injury risk. Amateur fighters are at twice the risk of professional fighters [5, 11, 12].

Brazilian Jiu-Jitsu

Limited injury surveillance studies show that elbow and knee joints are the most common injuries at tournaments. Hyperextension injury of the elbow from the use of an arm bar appears to be the most common mechanism. Risk of joint injury appears to be similar among belt ranks.

What Is Unique About Martial Arts Injuries?

- Minor wound care constitutes the majority of martial arts injuries during competition including abrasions, contusions, and epistaxis.
- Matches generally require a decision by the judge or referee to suspend a match in the event of medical injury. Participants must be granted permission prior to medical treatment, and limits imposed on treatment time vary by sport.

What Do the Physicians Need to Know While Covering a Game on the Sideline?

- Event coverage needs will vary based on the style and nature of competition. Ensure that the physician is present when competition rules are reviewed. Familiarity with the particular style techniques, targets, equipment, and scoring should be ensured to best anticipate potential injuries.
- Physicians should be aware of the governing rules that each martial art is subject to (e.g. Taekwondo follows the World Tae Kwon Do Federation medical code).
- Medical equipment should be available to control bleeding related to abrasion, laceration, or epistaxis, including disposable gloves, gauze, bandages, alcohol swabs, betadine, silver nitrate, nasal plugs, and suture kits. Splinting materials, slings, oral airway, and cervical collar should be available.
- Cervical spine injury or severe head trauma is rare, though an emergency action plan should be in place to address these concerns. Control of access to a participant should be ensured to prevent inappropriate movement of a participant with an unstable injury.

General Rule About Return to Play

Martial arts injuries are as diverse and varied as are the styles themselves. Decisions about return to play should be made based on the unique needs of the individual athlete, style, and nature of injury. When returning to combat-based training or competition, an athlete should be able to demonstrate adequate mobility and strength to avoid placing themselves at increased risk of additional or more severe injury.

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Motor Sports

63

Luke Widstrom and Jodi M. Blustin

Key Points

- A medical director should oversee medical care of events. Specific protocols and emergency action plans should be outlined and reviewed prior to events.
- Physicians covering motor sports events should have training in trauma care and appropriate supplies as well as established access for timely transport to definitive care should be available.
- Advances in personal protective equipment and vehicle design as well as rule changes and course design have been key to reducing the frequency and severity of injuries. Physicians covering motor sports events should familiarize themselves with event equipment and regulations.
- In motorcycle racing, using specifically designed courses, rather than road racing, appears to reduce the risk of injury significantly.
- In providing coverage for events involving watercraft, physicians should have training in care for drowning victims.
- ATV use poses a uniquely elevated risk of injury and death to the pediatric patient. Physicians should discuss this risk with adolescent patients and parents of children who use or are considering ATV use.
- Helmets significantly reduce the risk of head trauma in ATV users.

L. Widstrom
Department of Orthopedics, Essentia Health-Duluth Clinic, Duluth, MN, USA

J. M. Blustin (✉)
Department of Family Medicine, North Memorial Health and University of Minnesota, Minneapolis, MN, USA
e-mail: blust010@umn.edu

Automobile

Introduction

Automobile sports include a wide range of activities, ranging from multiple variations of car racing, to demolition derby, to monster truck events. Risks of participation include musculoskeletal injuries, heat illness, and noise exposure. Varying levels of personal protection and prevention are provided by the vehicle itself and other preventive measures depending on the type of vehicle, the level of competition, and regulations. Protection can range from vehicle design, to personal protective equipment (PPE), to physical conditioning, to rules of competition. However, a high degree of injury and even fatality is seen due to the speed and force at which the events take place.

Injury Epidemiology

In professional automobile races, approximately 14 drivers die on average per year in the United States [1].

Upper extremity injury is more common than lower extremity injury in stock car racing, estimated at 43% and 17.5% of drivers having experienced each type of injury, respectively [1]. Among upper extremity injuries, the most common are neuropathies such as hand-arm vibration syndrome or carpal tunnel syndrome, followed by distal radius and scaphoid fractures [2]. Neuromas may also develop on pressures points of the hands from steering [2].

A total of 45%of drivers in one study reported having back or torso injuries, while only 25% reported neck injuries, and an additional 25% reported having experienced a concussion [1]. Ongoing back and neck pain is an issue as well, with reports of 22.5–26% of race car drivers reporting continued back pain and 15–18% having persistent neck pain [1, 3]. Interestingly, among demolition derby drivers participating in an average of 30 career events and 52 crashes per

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event, only 7.5% reported chronic neck pain, while 85% reported having at least some neck pain after a typical event [4]. The greater frequency of neck pain but lower incidence of chronic neck pain compared with stock car racing may be explained by a greater number of crashes, but much lower crash speed. Overall, reports suggest that head and neck injuries may be decreasing since the implementation of Head and Neck Support (HANS) devices in 2001 [5]. Prior to 2001, craniovertebral junction injuries were the leading cause of death in high-speed motor sports; however, after new rules in professional racing mandated HANS, there have been no fatal craniovertebral junction injuries [6].

Temperatures in cars during events may rise to as high as 66 °C [1]. In addition, protective suits significantly reduce evaporative heat loss [7]. As a result, heat illness is a potential risk and heat stroke has been reported in Grand Prix racing [8]. However, no studies have been published, which report the incidence of heat illness.

Motion sickness is a common occurrence in rally car co-drivers. This occurs more frequently during reconnaissance ride (15.3% of co-drivers) compared with competition (2.3%) [9].

Both temporary (less than 24 h) and permanent noise-induced hearing loss may be associated with motor sports, and have been studied in stock car racing in particular. A single exposure to 140 dB has the potential to cause permanent hearing loss, while repeated exposure in the 85–140 dB range may cause permanent hearing loss due to metabolic changes in the cochlea. Peak noise levels have been measured at 130 dB just outside the car and 125 dB inside the car, suggesting that crew members are at even higher risk than drivers. Noise levels at 150 ft may range from 96.5 to 104 dB. Occasional exposure is unlikely to result in permanent hearing loss for spectators (though temporary hearing loss is possible), but hearing protection is recommended for crew members and drivers due to cumulative effects of repeated exposure [10].

What Is Unique About Automobile Injuries?

- In addition to drivers, injuries are also common with pit crew members. In a review of upper extremity injuries in NASCAR, only 16% were incurred by drivers, while the remaining 84% constitute pit crew injuries [2].
- The risk associated with automobile sports extends to spectators. A quarter of the fatalities at racing events are to spectators, journalists, or track workers [11].
- Various forms of unique protective equipment are used or required in automobile sports. These include strategically padded gloves to prevent neuromas, helmets, the Head and Neck Support device, ear protection, five or six point harnesses, flame retardant suits, fuel cells, roll cages, soft walls, and more.

What Do Physicians Covering Automobile Events Need to Know?

Due to the potential for significant trauma and catastrophic injury, training in Advanced Trauma Life Support (ATLS) is recommended for physicians covering automobile sports. EMS should be ready on site and an emergency action plan should be reviewed prior to all events.

Return to Play

Decisions on returning to competition should pertain to the specific injury and level of demand required for the involved region or organ system. One particular difference between motor sports and most other sports is consideration not only for the competitive ability and safety of the participant but also for the safety of other participants. Athletes should not be cleared unless able to appropriately control the vehicle throughout the duration of the event without increased risk of harm to self or others.

Motorcycle

Introduction

Motorcycle sports include racing on pavement, motocross which involves racing on dirt with jumps included, and freestyle events which involve performing both tricks and jumps. As with automobile sports, the speed and force in motorcycle sports can result in a high degree of injury due to trauma. This compounded by the fact that the vehicle itself offers little protection. As such, more importance is placed on personal protective equipment.

Injury Epidemiology

Only one death occurred in the top class of motorcycle racing between 1995 and 2006 [12].

In professional racing, single vehicle accidents predominate, while collisions are quite rare [13, 14]. When using racetracks specifically designed for the sport, motorcycle racing appears relatively safe, especially when compared to racing and recreational riding on regular streets [13–15]. In motoGP track racing, there were only three deaths in a 20-year period, whereas in the Isle of Man street race, there were 16 deaths out of 2500 riders [14, 15].

There are four basic types of crashes: “lowside” (falling toward the inside of a turn), “highside” (falling away from the direction of turn), “topside” (going over the handlebars of the motorcycle), and collision. Highside crashes are thought to occur due to overcorrection when there is loss of

traction on a turn. In a study of elite MotoGP racers, 74% of crashes were lowside, while 17% were highside, 3% were topside, 0% were collisions, and 6% were indeterminate. Lowside injuries tended to be much less severe than the other types of crashes, with about two-thirds of riders being able to continue the race and only 1 rider out of 58 requiring any medical attention at all. In comparison, in highside crashes only about one-fourth of riders were able to continue the race, nine of thirteen required trackside medical care, and three required hospitalization. There were only two topside crashes. Neither racer was able to continue the race and one rider required hospitalization [14].

In a study of motorcycle racers evaluated at a trauma hospital, 48% required operative intervention, 12% required a hospital stay of 5 days or more, and 9% required ICU admission. The most common injury observed was clavicle fracture (14%), followed by long bone fracture (11%), forearm/distal radius fracture (9.7%), and other shoulder injuries. Two or more injuries were seen in 18% of racers. Most of the injuries occurred in males age 11–20. Time of the year also seemed to be a factor, as the majority of injuries occurred in March and April, perhaps indicating that racers were out of practice after the winter months [13].

What Is Unique About Motorcycle Injuries?

- According the NHTSA, per vehicle mile traveled, ordinary motorcycle riders are 35 times more likely than passenger car riders to die in a traffic crash [16]. However, rates of death in motorcycle racing and automobile racing are similarly low.
- The majority of injuries and deaths in noncompetitive motorcycle riding result from collisions with other vehicles or stationary objects. On courses specifically designed for motorcycle racing, these objects are either absent or protected by sandbags.
- Specialized protective equipment include fitted carbon fiber full face helmets, full protective kangaroo leather armor, back braces, boots, gloves, and CO_2-powered self-inflating shoulder, chest, and neck protectors, which deploy when they sense a rider falling violently.

What Do Physicians Covering Motorcycle Racing Need to Know?

Due the high acuity and trauma involved in many motorcycle injuries, onsite medical facilities are highly encouraged. Additionally, track design and conditions play a key role in safety, so careful consideration of these factors should be undertaken. Lastly, more injuries result from highside and topside crashes, so riders should be taught to avoid overcorrection when traction is lost in a turn and providers need to be alert to the crash mechanism.

Return to Play

Like in most sports, the timeframe for returning to participation depends on the type of injury sustained. However, in motorcycle racing, one must consider the safety of not only the injured individual but also the other riders. In order to be cleared to compete, a rider must demonstrate complete physical and cognitive recovery. Even slight delays in processing speed, when combined with the high speeds of racing, can have catastrophic results.

All-Terrain Vehicle (ATV)

Introduction

ATVs consist of a group of four-wheeled motorized vehicles optimized for dirt trail or off-road use (Fig. 63.1). They typically have an open design similar to motorcycles, although newer designs may include bars or cages to provide additional protection. ATVs are typically used for recreational purposes more so than competition. Additionally, ATVs may be used for commercial used such as farming and ranching [17].

Injury Epidemiology

There is a considerable risk of death associated with ATV use, with a reported 7188 ATV related deaths in the United States between 1982 and 2005. Thirteen percent of these incidents occurred in children under 12 years old [18]. Of all ATV-related deaths in the United States between 2000 and 2007, 23% were under the age of 16 [14]. The rate of death is estimated at one in 10,000 uses [18]. More children in the United States die from ATV accidents than bicycle accidents every year [19]. A greater number of fatalities occur due to roadway crashes than off-road crashes [20].

In addition to death, ATV injuries have a high rate of serious injury. A review of emergency room visits over a 10 year period from 1993 to 2002 at a tertiary pediatric trauma center in Canada revealed that 30.8% of ATV-related injuries resulted in admission to the ward or ICU [21]. This rate seems to be typical as another study reported 35% of patients under 16 years old with ATV injuries were admitted to the hospital [22]. There is no difference between pediatric patients and adults in the risk of sustaining moderate to serious versus minor injury [22].

Helmeted patients are at a decreased risk of traumatic brain injury (TBI). A review of all ATV injuries presenting to a Level I trauma center from 2008 to 2012 revealed 8% of

Fig. 63.1 ATV. (Reprinted with permission from iStock #ID:610252554)

helmeted patients suffered TBI versus 26.6% of un-helmeted patients. In addition, helmeted patients had lower ICU admission rates and shorter ICU and hospital stays [23].

Of all ATV-related injuries presenting to a select group of Canadian emergency rooms during a 20 year period from 1990 to 2009, 58% of patients were youth and 42% were adults [22]. The majority of injuries (60%) were mild, while 22% were moderate, 14% were severe, and 0.16% were fatal, with the most common injury type being fractures at 39% [22].

In a study of emergency department visits in the United States from 2007 to 2012, fractures were most common in the shoulder at 27.2%, followed by wrist (13.8%) and lower leg (12.4%) [24].

In order of reported frequency of body area affected in pediatric ATV-related injuries, hip/leg is the leading region at 42.3%. This is followed by shoulder/arm at 36.2% of injuries, head/neck at 26.1%, trunk at 12.3%, spine at 2.3%, and in 4.6% of cases multiple injuries were sustained [21].

What Is Unique About ATV Injuries?

- Children may operate ATVs in the United States and Canada, which has resulted in a rising rate of pediatric injuries and death [21].
- A significant amount of injuries in ATV sports occur to passengers, with a reported rate of 29% in one study [21].
- Male participants and drivers are at increased risk of serious injury compared with females and passengers [22].
- Up to 60% of ATV injuries may be due to loss of control, while 15.4% occur in collisions and 6.2% are from falls [21].

What Do Physicians Covering ATV Events Need to Know?

Physicians who provide care for pediatric patients especially in rural areas should be aware of the considerable risks of ATV use and educate parents and patients about these risks. The anticipatory guidance component of well child visits may be an appropriate time to discuss this information and could play a large role in promoting ATV safety [25]. Helmet use should be encouraged, as this has been shown to significantly reduce the risk of TBI [23]. As with other motor sports, physicians covering ATV events should be well versed in trauma care, and appropriate services should be available for the trauma victim

Return to Play

Patients should be cautioned from returning to ATV operation until they are able to safely control the vehicle. In the setting of concussion, this includes restriction from operation until all symptoms have resolved. Patients should also be cautioned to avoid participation as a passenger or driver until there is little to no increased risk of reinjury to an affected body part in the event of even a low speed crash or fall.

Watercraft

Introduction

Motorized watercraft injuries are much more common with recreational use than in sanctioned events, as recreational use far outnumbers participation in races. In both cases there is risk of both trauma and drowning. Motorized watercrafts can be categorized into personal watercrafts (PWCs), also known as jet skis, and multiple passenger watercrafts such as speedboats and pontoon boats. Injury patterns vary depending on the type of watercraft.

Injury Epidemiology

PWCs appear to put riders at a much higher risk than other recreational watercrafts. The incidence of injury with PWCs has been estimated to be 8.5 times higher than with other motorized watercrafts. Collisions are the leading cause of injury with PWC use [26–28]. Common nonfatal injuries encountered with PWCs include contusions, fractures, lacerations, head injuries, and internal injuries [26, 27, 29]. The most common anatomical locations injured are the head and lower extremities [26, 29]. One study estimated that two-thirds of the injured drivers were male, while 70% of the injured passengers were female [26]. This same study noted that 22% of injured drivers and 38% of injured passengers were under the age of 15 [26].

Not surprisingly, the leading cause of death related to the vast majority of recreational watercrafts is drowning. PWCs, however, are a notable exception, with the leading cause of death being blunt trauma [30]. Alcohol consumption appears to play a significant role in powerboating fatalities. The risk of death while boating has been found to increase with blood alcohol content (BAC). In one study, odds ratio (OR) of death while powerboating was 1.3 with a BAC of 10 mg/dL, but increased to an OR of 52.4 with a BAC of 250 mg/dL [31]. The same risks were noted for both passengers and operators of the powerboats, regardless of whether the boat was stationary or moving [30].

What Is Unique About Watercraft Injuries?

Watercraft use presents both the risk of trauma and drowning, with trauma being more common with PWC use. While alcohol is known to increase the risk of injury and death in a number of recreational activities, this appears especially true with watercraft use, likely related to the risk of drowning. Personal floatation devices are important in preventing fatality due to drowning.

What Do Physicians Covering Watercraft Racing Need to Know?

With PWC use, collisions are the leading cause of injury, so limiting the number of watercrafts in a space appears especially important. Special consideration needs to be given to the unique challenges that events taking place on water present. This includes knowledge of the immediate treatment of drowning victims. Prior lifeguarding experience could prove valuable. If physicians do not have lifeguarding experience, the presence of trained professional lifeguards at events is recommended. As with other motorsports, training in trauma management is important.

Return to Play

Return to play decisions in motorized watercraft sports are similar to other motor sports and should take into consideration the ability to safely operate the vehicle in terms of both cognitive ability and musculoskeletal function.

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Racket Sports

64

Kylie Adamek and Bradley G. Changstrom

Key Points

- Tennis is the most popular racket sport worldwide.
- Common racket sports include tennis, squash, racquetball, and badminton.
- Tennis and badminton are considered noncontact sports, whereas squash and racquetball are considered limited-contact.
- Racket sports are played on a variety of surfaces including asphalt, clay, grass, artificial turf, rubber, wood, and carpet. Court surfaces are known to impact injury patterns.
- The skill level and age of the athlete significantly impacts the risk of injury.
- Across most racket sports, lower extremity injuries are more likely to be acute injuries; upper extremity injuries are more likely to be chronic injuries.
- Racket sports have the risk of eye injuries due to the ball making contact with the orbit. Sport-specific rules exist to help reduce this risk in some racket sports.
- Racket sports are associated with lower all-cause and cardiovascular mortality compared with most other sports.

Introduction

Racket sports such as tennis (Fig. 64.1), squash, badminton, and racquetball are considered some of the most popular activities in the world. Tennis is the most popular racket sport worldwide, and over 200 national tennis federations are affiliated with the International Tennis Federation (ITF), the governing body for tennis [1, 2]. Racket sports are often considered "lifelong sports" as individuals participate across all age levels. Accordingly, the style of play and level of training can vary drastically, and injuries tend to reflect the physical ability and intensity of the player. Due to length, this chapter is unable to address paddle sports (e.g., table tennis, pickle ball) and other racket sports. However, readers should know that there is likely overlap with many of these other sports as regards to injury patterns.

Epidemiology

In order to understand injury patterns in racket sports, it is helpful to review the movement involved in executing a forehand, backhand, or overhead stroke [3]. The power and adjustments of the stroke originate in the lower extremities, with the knees flexed with the hips rotated to load the trunk. This energy is subsequently transferred to the terminal upper extremity by way of the kinetic chain. As the knees extend, the trunk uncoils, and the counterrotation of the hips transfer energy to the shoulder, throughout the arm, and to the racket. The upper arm movements are used to maintain consistency and uniformity in the stroke pattern. Hence, while the power and adaptation of stroke come from the lower extremities, the motion of the upper extremity allows for a more passive transfer of power to the ball [3]. The ability level of the player often corresponds to the ability of the player to utilize the kinetic chain to generate force. Novice players are more likely to rely on the upper extremity to generate force [3]. These differences in technique may explain why we see difference in

K. Adamek
Department of Internal Medicine, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Kylie.Adamek@cuanschutz.edu

B. G. Changstrom (✉)
Department of Internal Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Bradley.changstrom@cuanschutz.edu

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Fig. 64.1 Tennis service. (Reprinted with permission from Keiko Shirai)

Table 64.1 Most common location of injuries in racket sports

	First	Second	Third
Tennis			
Adult recreational	Elbow	Shoulder	Knee
Elite junior	Knee	Ankle	Low back
Professional	Knee	Shoulder	Lumbar/buttock
Badminton	Knee	Ankle	Foot
Squash/racquetball	Ankle	Knee	Elbow

injury patterns (Table 64.1). For this reason, we have outlined this chapter to reflect injuries as they pertain to the professional tennis player, the adult or recreational player, and the junior tennis player. Due to relative lack of available data at this time, racquetball, badminton, and squash are reviewed irrespective of age and ability level.

Professional and Collegiate Tennis

Overall, tennis is a low risk sport, and rates of severe injury in tennis are low [3]. A 2006 review article demonstrated that injury incidence varied between 0.05 and 2.9 injuries per player per year and between 0.04 injuries/1000 h played and 3.0 injuries/1000 h played across all levels of play [4]. In addition, tennis injuries occur in the lower extremity (31–67%), followed by the upper extremity (20–49%), and then by the trunk (3–21%). Most acute injuries are lower extremity injuries [4, 5].

Across all epidemiologic studies in tennis players, there is considerable variation in how data is reported, and there are few prospective studies. In the professional tennis player, there is a large variation in the reported rates of medical withdrawal from tournaments due to variability in data collection. Across a variety of professional-level tournaments, incidence of medical withdrawal ranges from 2.11 to 65.15/1000 match exposures (ME) in women and 4.26 to 52.68/1000 ME in men [6–8]. Acute injuries were about twice as common as chronic injuries in those who reported a medical withdrawal. Lower limb injuries appeared to be the most common cause of medical withdrawal [6–8].

Many professional players elect to play despite injuries, and two recent studies looked at injury rates in professional players. The incidence of injury in the US open tennis players was 48.1/1000 injuries per ME (40.64/1000 ME in women and 55.56/1000 ME in men) [9]. Acute injuries were common and the incidence of acute injury was 27.65/1000 ME. Medical withdrawal was reported at 6.43/1000 ME in this study [9]. McCurdie et al. reported on Wimbledon tennis players using incidence based on the sets played [10]. They reported 20.7/1000 injuries per sets played (23.4/1000 sets played in women and 17.7/1000 sets played in men). Lower extremity injuries were common and made up 47% of injuries in men and 49% of injuries in women. Traumatic injuries represented 48% of injuries in this group; however, 34% of these traumatic injuries had occurred prior to the start of the tournament [10]. Medical illness was commonly reported in a population of the US open tennis players. Incidence was 36.74/1000 match exposures, and ENT and dermatologic conditions were most commonly reported [11].

In NCAA players, incidence was 4.89/1000 injuries per athlete exposure (AE) in men and 4.88/1000 injuries per AE in women. In this study, AE included both competition and

practice time. Lower extremity injuries are common in both men and women collegiate-level players (47% and 52%, respectively) [12].

Adult Recreational Tennis Player

Epidemiologic studies on the adult recreational tennis player are limited to survey studies. One study of US players reported 3.0 injuries per 1000 h of play, reported that most injuries were upper extremity overuse injuries, and reported that risk of injury increased with skill level [13]. A recent study of Dutch adult recreational tennis players reported on acute injuries and found an incidence of 7.87 injuries/1000 h of play [14]. This study also found no difference in prevalence of injury by court surface in the adult recreational tennis player, but did report that acute injuries were more likely in the lower extremity (4.57/1000 h of play) [14]. Rates of elbow injury, specifically lateral epicondylitis or "tennis elbow," are high in the adult recreational player due to excessive wrist extension on the backhand stroke and/or excessive forearm pronation on the forehand and overhead strokes [15]. In a review from Abrams et al., 35–51% of adult recreational players reported a career incidence of elbow pain [5].

Elite Junior Tennis Player

Elite junior tennis players represent a unique subset of players with regard to injury. The adolescent skeleton is not fully formed, and players are susceptible to overtraining [16]. Players with 1 day off or less per week were more likely to suffer an injury compared with those with 2 or more days off per week [17]. Of those players who had reported an injury, they were at 5.4 times greater risk for a subsequent medical withdraw from a tournament [17]. Epidemiologic studies demonstrated an injury incidence that ranges from approximately 2 to 20 per 1000 injuries per hour played [5, 18]. In a study of elite Dutch junior players, the incidence of acute injuries was 1.2/1000 h of tennis play, though overuse injuries predominated [5]. Similar to other populations, acute injuries tend to be located in the lower extremity [5].

Squash and Racquetball

Squash is a high-intensity sport characterized by an indoor playing surface that demands quick and precise movements from its players. It is particularly popular in Australia and the UK, though played by 15 million players participating across 135 nations worldwide [19]. In addition, it is offered at the NCAA collegiate level. Injuries sustained while playing squash account for a relatively low percentage of sports-injury cases. In Australia, a study found that squash only accounted for 2% of all sports injuries leading to adult emergency department presentations, ranking it 12th overall [20]. In a similar study, squash accounted for 1.3% of sports injuries presenting to a subset of sports medicine clinics, ranking it 11th overall [21]. A similar ranking of squash as a cause of sports injuries was observed in a UK population-based study that reported an injury rate of 5 injuries per 1000 squash-playing sessions [22]. The majority of injuries are acute, with the most commonly injured area being the lower extremity [19, 23].

Racquetball shares many characteristics with squash but has very few epidemiologic studies available to review, and no prospective studies exist. Like squash, it is played in an indoor court with the walls acting as part of the court. However, the ball is slightly larger than a squash ball and made of elastic rubber, traveling farther and lending to a much livelier bounce when struck.

While squash and racquetball players have a low frequency of overall injury, the relative rate of severe injury is conversely very high, particularly to the eye. Because the walls of the indoor arena are part of the playing surface, squash and racquetball require a spatial awareness of both the ball and the opponent that is not seen with other racquet sports. Accordingly, squash and racquetball have been established as a major contributor to the incidence of sports-related eye trauma [24].

Multiple studies have ranked squash as the first or second highest-ranked sport associated with sports-related eye injuries [25–28]. While incidence rates vary, one study from Western Australia was used to estimate the rate of eye injury as 17.5/100,000 playing hours [29]. Impacts to the eye from both the racquet and the ball have led to lacerations, corneal abrasions, retinal detachments, lid hemorrhages, and even loss of an eye [30, 31]. Fortunately, the introduction of protective eyewear in both sports has drastically decreased the number of eye injuries sustained by players and encourages continued use [23, 32].

Badminton

Badminton involves striking a feathered shuttlecock with a lightweight racket over an elevated net. Badminton is an Olympic sport and popular in Denmark and many Asian countries, with most epidemiologic studies originating from these areas. Elite players in Hong Kong experienced an incidence rate of 5.04 injuries/1000 player hours, while injuries in tournament-level Japanese players ranged from 0.9 to 5.1 per 1000 player hours [33, 34]. Overuse injuries were approximately three times more common than acute injuries [33]. The most commonly injured areas were the back, shoulder, thigh, and knee [34]. An older study with a mix of

elite and recreational players in Denmark had similar incidence of injuries with rates of 3.0/1000 player hours in men and 2.8/1000 player hours in women [35]. About two third of injuries in this population were due to overuse [35].

Studies of injuries presenting to sports medicine clinics and emergency departments reported higher rates of acute injuries. A study by Kroner et al. looked at injuries among 208 badminton players that presented to the casualty (emergency) department of two hospitals over the course of 1 year [36]. They reported that lower extremity injuries were common (82.9%) and ankle and lower leg injuries made up most of the acute injuries. Ankle injuries accounted for almost 67% of acute injuries, following by the knee with 15.7%, and the foot with 11.0%. In the majority of these cases, injuries were caused by players falling or stumbling while attempting to retrieve the shuttle. Eye injuries made up only 2.3% of injuries [36]. A study from the National Sports Institute in Malaysia reported that most badminton injuries were mild overuse injuries (91.5%) and that most occurred in the lower extremity (63.1%) [37]. Knee (37.1%) and ankle injuries (28.3%) were the most common injuries with patellar tendinopathy representing 42.7% of knee injuries and ankle sprains representing 85.7% of ankle injuries [37]. An earlier study in the UK looked at racket sports injuries presenting to a sports medicine clinic; 70% of injuries were acute and 59% were located in the lower extremity [23].

What Is Unique About Racket Sport Injuries?

Racket sports are played on a wide variety of court surfaces [38]. In 2011, the ITF approved 210 different court surfaces. These are classified into categories including acrylic, artificial clay, artificial grass, asphalt, carpet, clay, concrete, grass, and other (e.g., wood, canvas) [39]. Indoor racket sports such as badminton, squash, and racquetball are also played on a variety of surfaces. Most studies suggest that different playing surface impacts injury patterns and can change the duration of match play in professional players [39]. Despite many of these findings, a recent large study from the Netherlands did not demonstrate any differences in injury patterns by court surface in adult recreational tennis players [14].

Participation in racket sports may reduce cardiovascular disease. A large study of Scottish and English patients demonstrated significant reductions in all-cause mortality and cardiovascular mortality for patients who participated in racket sports compared with sedentary patients with hazard ratios of 0.52 and 0.44, respectively [40].

As individual sports without a time limit, medical issues such as cramping, dehydration, fatigue, and heat exposure are illnesses encountered by racket sports participants. For example, the longest professional tennis match in history was played at the Wimbledon Championships in 2010. This match lasted more than 11 h and was stretched over 3 days of play. Providers should be aware, however, that these illnesses are essentially considered “nontreatable” medical conditions during tennis, racquetball, and squash competitions.

Racket sports have the risk of upper extremity overuse injuries such as tendinopathies and stress fractures. These are almost exclusively located in the dominant arm. Traumatic upper extremity injuries are less common in tennis compared with the squash and racquetball because these sports can involve some incidental contact with the players and with the walled court.

“Tennis leg” is an injury to the plantaris tendon or the medial head of the gastrocnemius [41, 42]. This occurs when a player moves backward to hit an overhead shot and lands on the back leg. Although the plantaris tendon was initially implicated in most of these injuries, recent ultrasound studies have demonstrated that injury to the gastrocnemius is more common [41].

Achilles injuries appear to be more common in badminton than other racket sports. This is likely a reflection of the common use of an *overhead clear* shot in badminton. A study by Krøner et al. reported that 11 of 208 (5.2%) injuries were Achilles tendon ruptures [36]. In addition, a separate study by Høy reported that 13% of injuries were Achilles tendon ruptures [43]. Both of these studies were hospital-based studies and, therefore, the reported injuries were more likely to be severe, and overall risk of Achilles tendon rupture is lower in these populations. However, Fahlström reported on this in both elite- and recreational-level badminton players. Pain located in the Achilles was self-reported in 32% of high-level players over 5 years and in 44% of recreational players over the same time [44, 45].

What Do the Practitioners Need to Know While Covering a Match or Tournament?

Medical coverage of tennis events has been reviewed previously [46]. It is uncommon for physicians to provide medical coverage at most competitions other than professional-level events. Athletic trainers or physical therapists are available at professional and collegiate competitions. Outside of these scenarios, athletes are often expected to provide medical care and make medical withdrawal decisions for themselves. Although professional tournaments will have medical coverage, many professional players will also have access to a centralized medical clinic through his or her national tennis federation that may be providing input during an injury or illness [2]. Communication with these medical teams and the players and coaches is expected of practitioners providing medical coverage.

Generally speaking, these sports allow for medical timeouts of limited duration, and medical treatment is allowed

during breaks in play [47]. However, the rules of tennis state that "play shall be continuous," and medical evaluation and treatment rules are designed to keep within this framework. Players are allowed to have a medical condition evaluated and treated, but this evaluation is often brief and separate treatment time is limited to 3 minutes or less during a medical time out. However, players are allowed to receive medical treatment during expected breaks in play such as changeovers and in between sets. College, professional, and recreational rules are each different as regards the specifics of medical time-outs (Table 64.2). Up-to-date rules should be reviewed by the medical staff prior to any tournament coverage [47–50]. At the collegiate level, there are differences in these rules between divisions and even between men's and women's tennis at the Division 1 level [48]. Finally, medical providers should be aware that some medical conditions are deemed "non-treatable medical conditions." Non-treatable medical conditions are those that cannot be treated within the appropriate time frame, those that had not developed during warm up or

Table 64.2 Medical time-outs (MTO): tennis

Men	Women
NCAA division 1 [48]	
Player may receive one MTO for the entire match including warm-up	Player may receive one MTO for the entire match including warm-up
Player must forfeit the point for receiving an MTO	Once the athletic trainer reaches the player and begins talking/treatment, there is a maximum of 5 min for diagnosis and treatment. The maximum amount of treatment time shall be 3 min
Once the athletic trainer reaches the player and begins talking/treatment, there is a maximum of 3 min for diagnosis and treatment	A player who needs an additional MTO shall be retired
A player who needs an additional MTO shall be retired	A player may receive treatment on any changeover (no limit)
A player may receive treatment on any changeover (no limit)	
NCAA: All other divisions	
Player may receive 1 MTO per medical condition during the warm-up; player may receive 1 MTO per medical condition during the match	
Once the trainer reaches the player and begins talking/treatment, the trainer has a maximum of 5 min for treatment and diagnosis	
The maximum amount of treatment time shall be 3 min	
A player may receive treatment on any changeover (no limit)	
ITF/WTA [47]	
Player may receive a medical evaluation to determine if the player has developed a *treatable medical condition* (TMC)	
The evaluation should be performed within a reasonable length of time, balancing player safety on the one hand and continuous play on the other	
At the discretion of the *physiotherapist/athletic trainer* (PT/ATC), this evaluation may be performed off court with the tournament physician	
If the PT/ATC diagnoses a "non-treatable" medical condition, then no medical treatment will be allowed	
An MTO is allowed when the PT/ATC has determined that medical treatment is required	
The MTO takes place during a change over or set break, unless immediate medical treatment is required	
The MTO is limited to 3 min of treatment at most professional tournaments	
A player is allowed one MTO for each distinct TMC	
Manifestations of heat illness shall be considered as one TMC	
Injuries that manifest as part of a kinetic chain continuum shall be considered as one TMC	
Players may not receive an MTO for muscle cramping	
Junior and adult recreational players in United States (USTA) [50]	
Similar rules to the ITF rules above	
If a qualified medical professional is not available, the player may utilize any person	
After evaluation, treatment time is limited to 3 min	
Total time including evaluation and treatment should not exceed 15 min; if the maximum total time is exceeded, the player shall be penalized under the point penalty system	
A player may receive on-court evaluation or treatment no more than two times	
Bleeding time out [47, 48]	
Treatment time begins when player notifies official or acknowledges that there is bleeding	
Treatment time ends when bleeding has stopped, and the playing area has been cleaned up	
Maximum amount of treatment time is 15 min in NCAA and 5 min in ITF play	
If maximum total time is exceeded and the bleeding has not stopped, the player must retire	
If maximum total time is exceeded and the bleeding has stopped but the playing area is not cleaned up, the referee may move the match or continue cleaning up the court	

Table 64.3 Medical time-outs (MTO): squash, badminton, racquetball

Squash [51]
A player who suffers an *illness* that involves neither an injury nor bleeding must either continue play immediately or concede the game in progress and take the 90-second interval between games to recover. The player must then resume play or concede the match
A referee must determine if a medical *injury* is genuine and if the referee is not satisfied that the injury is genuine, he or she must advise the player to decide whether to resume play immediately, or to concede the game in progress and take the 90-second interval between games and then resume play, or concede the match
If a player suffers an injury that is a *result of the player's own action* (self-inflicted – e.g., collision with a wall or fall), the player is permitted 3 min to recover and, if not then ready to resume play, must concede that game and take the 90-second interval between games for further recovery. The player must then resume play or concede the match
If a player suffers an injury that is a *result of accidental action by both players*, the player is permitted 15 min to recover and may be extended at the discretion of the referee. If the player is then unable to continue, the match is awarded to the opponent
If the player suffers an injury that is *caused solely by the opponent*, the injured player is permitted 15 min to recover if the injury is caused by an accident. If the player is then unable to resume play, the match is awarded to the injured player. If the injury is *caused by the opponent's deliberate or dangerous play or action*, the match is awarded to the injured player if the injury player requires any time for recovery
Whenever bleeding occurs, play must stop, and the player must leave the court and attend to the bleeding promptly. Reasonable time for treatment is allowed. Play may resume only after the bleeding has stopped and, where possible, the wound has been covered. The court must be cleaned and clothing replaced
If blood is again visible during play, no further recovery time is permitted, and the player must concede the game in progress and use the 90-second interval between games for further treatment
Badminton [52]
Injury or illness during a match should be handled carefully and flexibly. The umpire must determine the severity of the problem as quickly as possible. The referee shall be called on the court, if necessary
The referee shall decide on whether a medical official or any other persons are required on court. The medical official should examine the player and advise the player about the severity of the injury or sickness. No treatment causing undue delay shall be administered on the court
If there is bleeding, the game should be delayed until the bleeding stops or the wound is suitably protected
The referee should advise the umpire of the time that may be required for the player to resume play
The umpire shall monitor the elapsed time
The umpire shall ensure that the opposing side is not put at a disadvantage
Racquetball [53]
No time-out shall be charged to a player who is injured during play
An injured player shall not be allowed more than a cumulative total of 15 minutes for an MTO
If the injured player is not able to resume play after a cumulative rest of 15 minutes, the match shall be awarded to the opponent(s)
On any additional injury to the same player, the tournament director or referee, after considering any available medical opinion, shall determine whether the injured player will be allowed to continue
Should any external bleeding occur, the referee must halt play as soon as the rally is over, charge an MTO to the person who is bleeding, and not allow the match to continue until the bleeding has stopped
Muscle cramps and pulls, fatigue, and other ailments that are not caused by direct contact (such as with the ball, racket, wall, or floor) will not be considered an injury

match play, those related to fatigue, or those that require injections or administration of oxygen. Diabetic patients can receive insulin injections if needed [47]. Medical time-outs for squash, badminton, and racquetball are separately reviewed in Table 64.3.

Return to Play

Following traumatic injuries in racket sports, return to play decisions are guided by injury, injury severity, level of play, sport-specific limitations, and recommendations from medical staff. Because substitutions do not exist in these sports, many acute injuries result in medical withdrawal [6, 9], and return to play is not possible during a match unless players comply with the medical time out rules described above.

Return to play for acute lower extremity injuries can be quite variable in racket sports ranging from immediate return in-match for issues such as mild ankle sprains or may require prolonged recovery and possible surgical intervention for issues such as meniscal tears, ACL ruptures, or Achilles tendon ruptures.

Many ophthalmologic injuries may have difficulty returning during match play; however, these are typically uncommon at the elite level due to the eyewear regulations in these higher-risk sports. Casual or recreational players are more likely to forgo protective eyewear and risk these injuries.

Most overuse musculotendinous injuries can return to play as tolerated. Upper extremity stress fractures are uncommon, but play should be withheld until these fractures are healed and appropriate bone health evaluation has been considered.

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Rock Climbing

65

Abigail Y. Wang and Sameer Dixit

Key Points

- Rock climbing is increasing in popularity among participants of all ages.
- Training and safety practices in rock climbing are not universal.
- Proper training in the use of harnesses, ropes, helmets, and crash pads and repetitive inspection of all gear are essential for safe rock climbing.
- The most common rock-climbing injury is a flexor pulley injury.
- Epiphyseal fractures are common in adolescent climbers.

Introduction

The types of rock climbing include bouldering, top rope, lead or sport, "trad" or traditional climbing, and free solo, or climbing without rope (Fig. 65.1). Bouldering involves climbing rocks of shorter height without rope and using crash pads placed on the ground to prevent injuries in the event of a fall. Other climbers may also "spot" the active climber to prevent head injuries. In top rope, the rope is secured to the top of the route prior to climbing, and a partner, or belayer, secures one end of the rope. Lead or sport climbing also has a belayer, but the climber clips the rope into bolts already on the wall as they ascend (Fig. 65.2). Trad climbing is a type of lead climbing, but individuals place their own protective gear into holes rather than using preplaced bolts. Lastly, climbers who free solo climb do not have a belayer or rope to catch them if they fall.

Recently, rock climbing has increased in popularity as both a recreational and competitive activity [1]. Bouldering, top rope, and sport climbing may be done indoors or outdoors, but trad climbing is typically done only outdoors. Safe climbing requires diligence by the climber and the belayer, who holds the other end of the rope. Climbing combines endurance, strength, and agility in a unique way, and climbers may climb actively for many hours in a single day. Theoretically, this can lead to decreased mental focus and, presumably, an increased risk of injury.

Injury Epidemiology

Injury rates in rock climbing are estimated at 4.2 injuries/1000 participation hours, with a career incidence of injury ranging from 1.5 to 4.2 injuries per individual [2]. By comparison, the injury rates are 16 injuries/1000 hours in football and 9.8 injuries/1000 h in basketball [3].

The two primary mechanisms of injury are overuse leading to acute injury and traumatic injuries caused by falls. Most injuries are in the first category, and of these, most involve the upper extremity. The hand and wrist are at especially high risk regardless of the climber's age or sex [3]. By contrast, most lower extremity injuries are caused by traumatic falls (Table 65.1).

The most common injury, A4 or A2 pulley tear, has been associated with certain rock climbing grips. The anatomy is described in Fig. 65.3, and the grading of these injuries is described in Table 65.2. An open crimp, a type of grip in which the proximal interphalangeal joint is flexed and the distal interphalangeal joint is extended, leads to high A2 forces that are likely to rupture in a fall [3]. These tend to spare joints, ligaments, and tendons. A closed-hand crimp, in

A. Y. Wang
Department of Orthopedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: awang61@jhmi.edu

S. Dixit (✉)
Departments of Orthopaedics, Medicine, and Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Lutherville, MD, USA
e-mail: sdixit3@jhmi.edu

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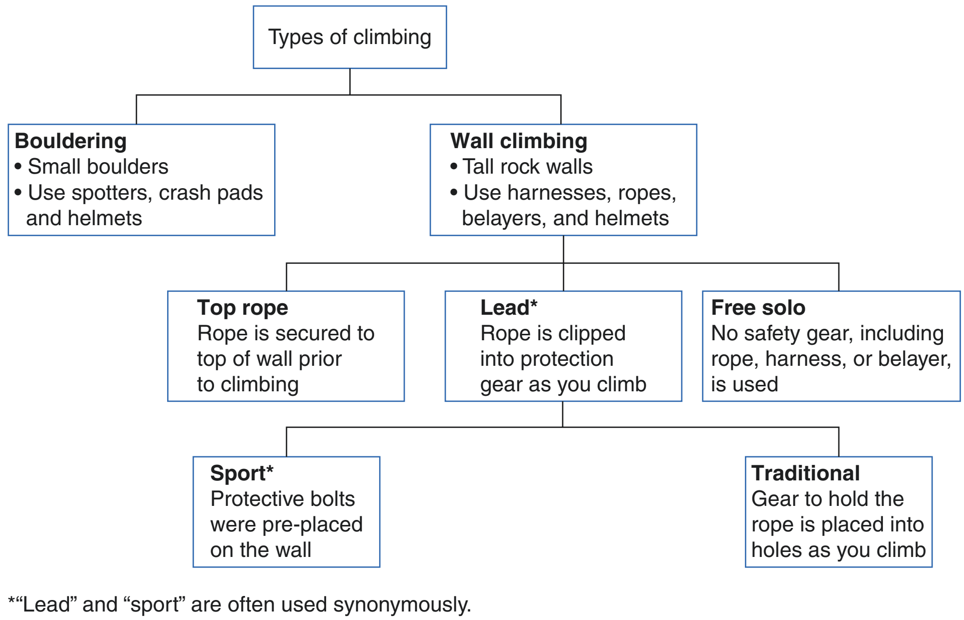

Fig. 65.1 Differences between the common types of climbing

Fig. 65.2 Sport climbing. (Courtesy of Dr. Tom Maino)

Table 65.1 Most common rock climbing injuries

Upper extremity [4]	Lower extremity [3]
A4 or A2 pulley tear	Calcaneal contusions or fractures
Capsulitis	Ankle fractures
Tenosynovitis	Injuries to the talus
Superior labral tear from anterior to posterior (SLAP)	Ankle sprains with ligament injuries

which both interphalangeal joints are flexed, leads to high A4 forces and is more likely to cause A4 pulley tears. Volar plate injuries are also common with a closed-hand crimp. Furthermore, between 1998 and 2012, the most common injury shifted from an A2 pulley tear to an A4 pulley tear, suggesting that sport-wide changes in grip use have affected injury incidence [6]. Other grips that commonly cause inju-

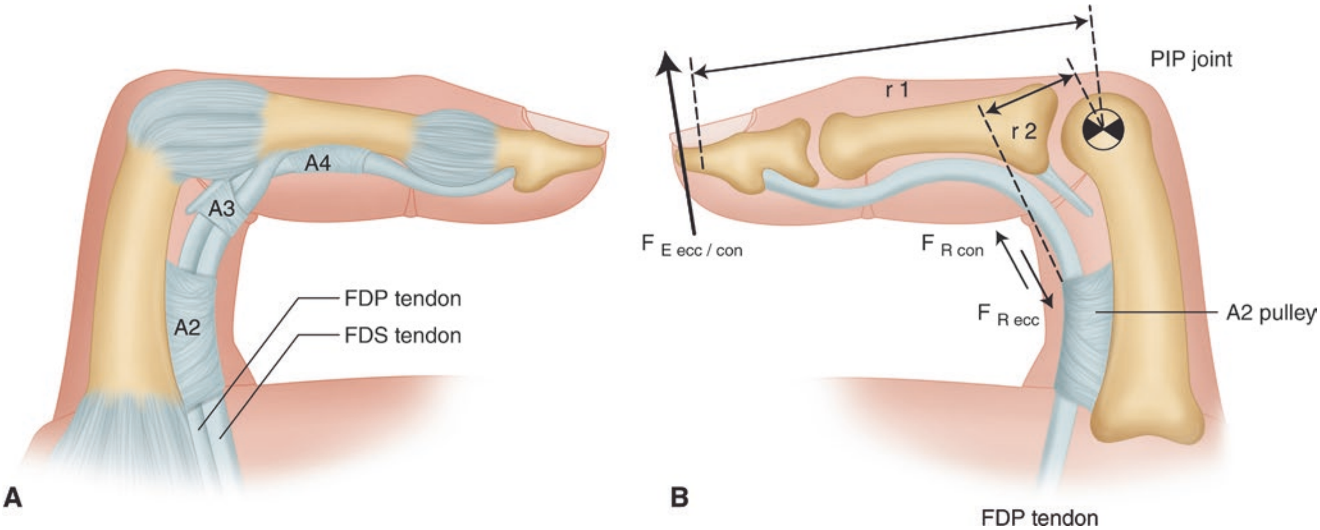

Fig. 65.3 Anatomy of the fingers. The A2, A3, and A4 pulleys are located at the proximal phalanx, PIP joint, and middle phalanx, respectively. (**a**) Proximal to the A2 pulley, the flexor digitorum profundus (FDP) tendon runs dorsal to the flexor digitorum superficialis (FDS) tendon. Underneath the A2 pulley, the FDP tendon runs through the FDS tendon to the palmar side and attaches on the distal phalanx. Volar plates are thickening of the joint capsule palmar to the IP joints that prevent hyperextension (**b**)

Table 65.2 A4 and A2 pulley injury grade [5]

Grade	Description
1	Pulley strain
2	Complete rupture of A4 or partial rupture of A2
3	Complete rupture of A2
4	Multiple pulley ruptures or one pulley rupture with lubrical or collateral ligament damage

ries include finger locks or hand jams, in which parts of or the entire hand is placed in a crack and rotated to provide traction [5]. Table 65.1 shows the most common upper and lower extremity injuries.

Another common climbing-specific injury is climber's elbow. In climber's elbow, the brachialis muscle is torn at the distal muscle-tendon junction [5]. This most commonly occurs when the elbow is both flexed and pronated such as when climbing sideways. Other climbing injuries include strains of the adductors, biceps, abdominal muscles, and spinal muscles; meniscus tears; head injuries from falls; and skin abrasions (Fig. 65.4). Climbers should be encouraged to wear helmets to prevent head injuries from falling rocks.

It is also important to consider injury epidemiology in different climbing types. The incidence of injury in trad climbing is higher than that of lead climbing, top rope climbing, or bouldering [6]. Lead climbing has been shown to involve a higher risk of injury than top rope climbing [7, 8]. The risk of injury is highest in trad followed by lead, top rope, and bouldering [9]. Traumatic injuries in bouldering are more often joint or ligament injuries rather than contusions or lacerations

Fig. 65.4 Skin abrasion as a result of climbing. (Courtesy of Dr. Tom Maino)

as sustained in in rock climbing [2], and they commonly involve the fingers. There is also a higher potential for ankle, knee, and foot injuries in bouldering compared with other types of climbing because of repeated short falls onto mats. In general, outdoor climbing has a higher rate of injury than indoor climbing [6].

Rock climbing injuries typically require prolonged conservative therapy, and some require surgery [9]. It is rare for an injury to lead to acute danger during climbing. Serious injuries are caused predominantly by mistakes and lapses in judgement as opposed to external causes, equipment failure, or ignorance [2]. Loss of focus is a large risk factor for injury in this sport, and climbing at a lower grade is, counterintuitively, associated with a higher rate of injury [10]. The greater energy expenditure in outdoor climbing may account for the higher risk of injury compared with indoor climbing [5]. Similarly, injuries are more likely to occur in the afternoon (and during peak season, from May to September [11]) after individuals have been climbing for several hours [6]. It is important to remind climbers to rest between rigorous climbs because handgrip fatigue can last for 20 min [5].

Adolescents

Adolescent climbers have an injury rate of 4.4 injuries per 1000 climbing hours, which is similar to injury rates in adolescent ice hockey and soccer players [8]. Though not one of the most common injuries overall, finger epiphyseal fractures are common among elite adolescent climbers, especially those aged 15–19 years. Adolescents' bones and ligaments have not fully developed, and epiphyseal fractures can lead to improper bone growth [7]. Therefore, early vigilance for and recognition of these injuries in adolescent climbers is important to avoid long-term consequences. Young climbers are also more likely to reinjure themselves compared with adult climbers (Table 65.3) [2]. Given the high risk for permanent damage, adolescent climbers should be taught the early signs of overuse injuries. They should also be cautioned to avoid using a campus board, a pull up board with small rock climbing holds, or adult training regimens.

Table 65.3 Most common injuries in specific climbing populations [12]

Elite climbers	Adolescent climbers
1. Collateral ligament injury	1. Ligament strain
2. Flexor pulley tear	2. Muscle/tendon strain
3. Flexor pulley strain	3. Tendonitis

Elite Climbers

In the 2005 World Championships in rock climbing, there was a rate of 3.1 injuries per 1000 participation hours, which is similar to the overall injury incidence in the sport [13]. Of these injuries, four were serious and none led to fatalities. Most injuries in elite climbers are localized to the hand or upper extremity. Elite climbers are less likely to have lower-extremity abrasions and lacerations compared with inexperienced climbers (Table 65.3).

Data specific to elite rock climbers are limited. It has been shown that higher climbing intensity, based on average route difficulty multiplied by number of climbing days, is a better estimate of an individual's injury risk than climbing volume or skill level alone [1]. Furthermore, longer climbing experience is not associated with higher risk of injury [14].

Elite climbers are at higher risk of environmentally caused life-threatening injuries [5]. They are more likely to climb in isolated areas with more dangerous environmental factors such as at high altitudes or low temperatures. An elite mode of climbing called free solo climbing, in which no ropes are used, has a high risk of death and injury.

Additional Climbing Types

Ice climbing and mountaineering are subsets of rock climbing. In ice climbing, individuals use ice picks and crampons to climb frozen waterfalls. In mountaineering, the goal is to reach a summit, which may require additional skills such as hiking and ice climbing.

These two climbing types have high rates of injury and fatality in comparison with other climbing modalities (Table 65.4) [6]. Furthermore, a larger portion of these injuries are caused by falls rather than acute injuries following overuse. Injuries unique to these types include frostbite, hypothermia, and altitude sickness.

The American Alpine Club publishes a booklet annually on reported rock climbing and mountaineering injuries [11]. It is not comprehensive, but highlights injury trends. Between July 2015 and July 2016, there were 173 reported accidents, with 111 injured individuals and 37 fatalities. Most accidents occurred while climbing rock rather than

Table 65.4 Most common mountaineering and ice climbing injuries

Most common injury	Mountaineering	Ice climbing
Site	Head or spine	Head
	Trunk	Upper extremity
Type	Polytraumatic	Open wounds
	Multiple fractures	Hematomas

ice or snow. Most injuries were attributable to subjective causes that may be controlled by the climber such as falls, exceeding abilities, or faulty use of equipment. However, there were 31 injuries caused by falling rocks, being stranded, avalanches, or exposure. The most common type of injury was a fracture, followed by lacerations, bruises, and concussions.

What Is Unique About Rock Climbing Injuries?

- Rock climbing safety training is not universal, and safety checks are highly important for injury prevention.
- Many climbers participate in long climbing days that can lead to decreased mental focus and increased risk of injury.
- Some outdoor climbing sites are difficult to access, which affects the speed of emergency care.

What Do Physicians Need to Know?

- Climbing injuries in young and adult climbers may lead to permanent damage if not identified early.
- Familiarity with upper extremity anatomy and function, particularly of the hand, is vital to early recognition of injury.
- Taping of wrists and weight training are the only two consistent injury prevention measures described in the literature; although other measures have been reported, there is conflicting data on their efficacy [15].
- Proper use and maintenance of safety gear can substantially reduce the risk of injury [15].

General Rules About Returning to Climbing After Injury

- When symptoms improve, climbers may be tempted to return to climbing before full healing of their injuries.
- Climbers may consider changing their grip technique to prevent repeated injuries.
- In finger injuries, taping can decrease time to return to climbing [15].

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Rowing

66

Andrew T. Gomez and Ashwin L. Rao

Key Points

- The majority of injuries in rowing are the result of overuse syndromes though many of these overuse injuries can present acutely.
- Low back pain is the most common complaint among rowers.
- A variety of musculoskeletal, dermatologic, neurologic, and vascular pathologies occur that a team physician should be aware of.
- Though rare, the medical team should be prepared for the possibility of traumatic events, cardiovascular events, and cold-water immersion as these occurrences pose logistic challenges.

Introduction

Rowing is increasing in popularity, both on the water and with ergometers for both recreation and competition purposes. Competition occurs at various levels, including in clubs, at the high school, collegiate, elite, masters, and Olympic [1]. Though acute injuries occur, the majority of injuries relate to overuse; in some cases, a sideline physician may be the first to see the initial presentation of an overuse syndrome or may evaluate an athlete with abrupt worsening of a chronic condition [2] (Fig. 66.1).

Sweep rowing and sculling are two major categories of rowing. Sweep rowing involves one oar per rower, and each boat has two, four, or eight rowers. A boat with eight rowers will always have a *coxswain,* while boats with two or four may not. Sculling involves two oars per rower, and each boat has one, two, or four rowers. In both types of rowing, athletes sit facing backward and contact the boat at three points: the feet are fixed, the buttocks rests on a sliding seat, and the hands are placed on the handles of the oar(s). The rowing stroke has four components: the catch, drive, finish or release, and recover [1–4].

Rowing is one the most strenuous sports that requires both aerobic and anaerobic exercise. There are two seasons: the sprint season in the Spring/Summer and the head season during the Fall. Sprint races vary in length from 1 to 2 km depending on competition level, while head races occur over a 4–10 km course as a race against time. Given the differences in the seasons, a crew training regimen will emphasize anaerobic capacity for the sprint season and aerobic capacity for the head season [1, 2].

Injury Epidemiology

The majority of injuries involve the back, the upper extremity and shoulder, and knee. A number of cohorts have documented the various injury locations and types depending on the level of participation, though the classification systems are not standardized. Tables 66.1, 66.2, and 66.3 demonstrate commonly injured locations based on level of competition.

Back Pain

- Low back pain is one of the most common complaints in rowers [1, 9].
- Predisposing factors for back pain in rowers involve excessive hyperflexion and/or twisting of the low back, hamstring strength deficiency relative to quadriceps strength, muscular asymmetries (e.g., erector spinae, hip musculature), and increased respiratory demands [3].

A. T. Gomez
Swedish Providence Medical Group - Mill Creek Sports Medicine, Everett, WA, USA
e-mail: andrew.gomez@swedish.org

A. L. Rao (✉)
Department of Family Medicine, University of Washington, Husky Stadium Sports Medicine Center, Seattle, WA, USA
e-mail: ashwin@uw.edu

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Fig. 66.1 Rowing

Table 66.1 High school and international junior rower injury epidemiology, location of injury [5, 6]

	High school		Junior international	
	Male (%)	Female (%)	Male (%)	Female (%)
Hand/forearm/wrist	22.0	17.4	9.6	13.6
Shoulder/upper arm	–	–	6.2	8.2
Low back	10.2	12.6	34.4	29.9
Thigh/upper leg	11.9	8.4	8.1	8.7
Knee	16.9	18.4	19.6	17.9
Lower leg	10.2	12.6	–	–
Ankle	10.2	8.4	–	–

Table 66.2 National-level rowers injury epidemiology [7]

Injury	
Low back pain	44.1%
Fractures/contusions	14.7%
Hamstring tendinopathy/ischiogluteal bursitis	11.8%
Hip FAI/labrum/chondral injury	5.9%
Thoracic spine/chest wall	5.9%

- Other risk factors include rowing before age 16, use of a hatchet oar blade, weight training, ergometer, ergometer training sessions longer than 30 min, and higher weight and height [10].

Table 66.3 International-level rowers injury epidemiology, location of injury [8]

Location	
Lumbar spine	31.8%
Knee	15.9%
Cervical spine	9.1%
Wrist	6.8%
SIJ/thoracic spine	4.6%

- Rowers with preexisting back pain may be at increased risk of developing back pain during their collegiate rowing career relative to those without preexisting back pain [11].
- Those with back pain should be appropriately evaluated for muscular imbalances as well as strength and flexibility deficiencies, and their technique should be observed [3].
- Modification in technique, stretching, core stability training, and maintenance of correct posture, equipment modification, and decreasing the load per stroke may assist in alleviation of back pain [1, 3].
- Most back pain complaints resolve with relative rest, activity modification, and strength and flexibility training.
- Most athletes with back pain were able to return to full activities in 1–2 weeks, and surgery was rarely required [9].

Discogenic Back Pain

- Repetitive flexion and anterior compressive forces affect the annulus and nucleus pulposus, resulting in the possibility of disk bulging or herniation [12].
- Impairment in the sensorimotor control mechanisms and decreased reflexive action of the multifidus and longissimus muscles may also contribute to injury [12].
- Central nonradicular back pain in a competitive rower may be the result of central disk herniation and should be evaluated as such early in the injury course [13].

Spondylolysis and Spondylolisthesis

- Rowers are at particular risk given repetitive lumbar hyperflexion/hyperextension and rotation in sweep rowers.
- Prevalence of spondylolysis is higher in rowers compared with general population in adolescents and adults [3].
- Predisposing factors include tightness of hip flexors and hamstring, weakness of abdominal and gluteal musculature, lordotic posture, fatigue, high intensity/volume training [3, 12].

Rib Stress Fracture (RSF)

- RSF is the most prevalent stress fracture among rowers, and the incidence has been documented at approximately 9–10% [14, 15].
- The etiology of RSF is debated and likely multifactorial. In general, it is thought to result primarily from the forces of the serratus anterior and external oblique muscles during the rowing stroke [1, 13, 15, 16].
- Risk factors for RSF include excessive training, inadequate nutrition, low bone mineral density, hormonal factors, equipment problems, or recent equipment changes [1, 15, 17].
- The incidence of RSF increased in the early 1990s and is likely related to accelerated training volumes and intensity in addition to the widespread adoption of a broader oar paddle, which increased the load per stroke [16, 18, 19].
- RSF may initially present acutely or insidiously with generalized pain in the thorax, gradually becoming more specific or focal.
- Pain is worse with deep breathing, coughing, sneezing, and position changes. Aggravating movements include the rowing stroke or rolling over in bed [3, 13, 14].
- RSF can occur and affect any rib in any location, but most commonly affects the middle ribs in the anterolateral region in general [15].
- However, in sweep rowing, the majority of RSF occur in the anterolateral/lateral aspect of the rib cage, while in sculling they tend to occur equally along the anterolateral/lateral and posterolateral/lateral aspects of the rib cage. The reasons for this remain unclear [15].
- No association has been established between the injured chest side compared with rowing side among sweep rowers [15].
- Management generally includes complete removal of bending forces from the rib by stopping all rowing on water and with an ergometer.
- Physical therapy and a gradual return to increased activity are recommended.
- Early return to rowing may alter proper rowing mechanics given the presence of pain [3, 9, 13, 14, 17, 20].

Shoulder Injuries

- In general, overuse, improper stroke technique, and muscle imbalances may contribute to acute and chronic shoulder injuries in rowers [3].
- The power phase places significant stress on the shoulder as force is transmitted from the oars to the back [3].
- Younger rowers are at increased risk for shoulder instability relative to their nonrowing peers [13].
- The risk of dislocation in these athletes is greatest at the start of a sprint race [13].

Distal Clavicle Osteolysis (DCO)

- DCO can affect individuals who undergo repetitive motions of the upper extremity, such as rowers and weight lifters, and is commonly misdiagnosed as AC joint injury or sprain, especially when symptoms follow an acute injury.
- Pathophysiology remains debated but likely related to either repetitive microtrauma leading to microfracture in the subchondral bone at the AC joint or synovial invasion of the subchondral bone leading to osteolysis.
- Pain is often insidious but may be acutely precipitated by exacerbating activities; pain is often localized to the anterior shoulder girdle without the sensation of subluxation.
- Examination often yields tenderness at the AC joint.
- Plain radiography should include the Zanca view (AP with 10° cephalic tilt and 30–50% of usual voltage) and display pathologic changes to the distal clavicle and loss of or cystic changes of the subchondral bone at the distal clavicle. Arthritis is not often present, but distal clavicular osteopenia and tapering may be evident.
- MRI may show increased signal intensity with fat suppressed T2-weighted and short-tau inversion recovery

(STIR) images. Bone marrow edema is often present at the distal clavicle and correlates with severity of symptoms.
- Conservative management includes activity modification, physical therapy, and NSAIDs. Intra-articular ACJ corticosteroid injections may be diagnostic and therapeutic.
- Refractory symptoms are managed with surgical intervention [21].

Forearm and Wrist Injuries

- Repetitive rotation of the oar twice per stroke during feathering and squaring predisposes rowers to injuries at the forearm and wrist [13].
- Injuries include intersection syndrome (Oarsman's wrist), Sculler's thumb, exertional compartment syndrome, De Quervain's tenosynovitis, lateral epicondylitis, among others [12].
- Predisposing factors to injury include poor technique, fatigue, and excessive wrist motion during feathering/squaring, tight grip, incorrect oar handle size, poor rigging, wet/cold or rough conditions [1, 3, 12].
- Ensure appropriate oar handle size and rowing "on the square" without feathering may help [1, 3, 12].
- Ensuring the forearm and wrist are warm during rowing may help with prevention, including the use of fleece "pogies," which cover the outside of the hands but permit the hand to contact the handle [4, 12].

Intersection Syndrome

- Intersection syndrome (Oarsman's wrist) involves pain and swelling approximately 4–8 cm proximal to the radial styloid at the dorsal wrist, which is the area where the first extensor compartment tendons cross over the second extensor compartment tendons [3, 12, 13, 22].
- Pain, swelling, and tenderness is present at the dorsal wrist, worse with dorsiflexion or ulnar deviation, and there may be crepitus in more severe cases [1, 4, 13, 22].
- Intersection syndrome can be diagnosed clinically and imaging is not often necessary.
- Intersection syndrome can be distinguished from De Quervain's tenosynovitis, which is also seen in rowers, given the point of maximal tenderness. De Quervain's involves tenderness more distally, at the level of the radial styloid [3, 13].

Sculler's Thumb

- Sculler's thumb is the hypertrophy of the muscle bellies of the extensor pollicis brevis and abductor pollicis longus.
- The hypertrophied muscle may compress the underlying radial extensor tendons, causing swelling over the dorsal aspect of the forearm [3, 12].

Chronic Exertional Compartment Syndrome (CECS)

- Chronic exertional compartment syndrome of the forearm results from repetitive isometric muscle loading of the wrist while gripping.
- Although described as chronic, this syndrome may present acutely with its classic symptoms of pain in the forearm, loss of grip strength, and altered sensation following a period of rowing.
- Symptoms are usually bilateral in rowers.
- Diagnosis of CECS involves monitoring intercompartment pressures before, during, and after exercise with the diagnosis being made with a resting pressure of greater than 15 mmHg, 1-min postexercise pressure of >30 mmHg, or 5-min postexercise pressure >20 mmHg.
- Fasciotomy is the definitive treatment though is rarely needed [12, 23].
- If exertional compartment is sustained, rhabdomyolysis may occur; rhabdomyolysis has also been documented in elite rowers as a result of rigorous exertion as well [24, 25].

Hook of Hamate Fracture

- Hook of hamate fractures are rare but may occur in rowers given their extensive contact with the oar.
- Athletes usually report pain over the hypothenar eminence and hamate hook.
- Pain may be elicited with resisted flexion of the fourth and fifth fingers.
- Plain radiography often provides inadequate visualization and CT is typically required for diagnosis.
- Acute non-displaced fractures can be managed with cast immobilization, while displaced or non-united fractures generally require surgical intervention.
- Complications of hook of hamate fracture include ulnar neuritis, flexor tendon irritation and rupture, ulnar artery thrombosis, and symptomatic nonunion [26].

Triangular Fibrocartilage Complex Injury

- Triangular fibrocartilage complex (TFCC) injuries occur from repetitive load bearing and rotational stresses.
- The TFCC stabilizes the distal radioulnar joint (DRUJ) and also functions as a load absorber of the wrist.
- Ulnar-sided wrist pain in the rower with associated tenderness in the foveal area with occasional clicks and pops may be found on physical examination.
- Plain radiography should include a PA zero view rotation (with the shoulder abducted 90° and elbow flexed 90°), clenched fist pronation PA view, and contralateral views to assess ulnar variance, which may affect treatment decision if ulnocarpal abutment is suspected.
- MRI (occasionally an MR arthrogram) may provide better visualization and aid in diagnosis.
- Unstable DRUJs or stable symptomatic DRUJs may require surgical intervention [26].

Scapholunate Dissociation

- Scapholunate dissociation or instability may be the cause of pain in the dorsoradial aspect of the wrist in a rower as a result of a tear to the scapholunate ligament.
- Athletes may present with a “click” or “pain” in the dorsoradial region of the wrist that may be exacerbated on resisted second finger extension.
- The classic clinical examination for scapholunate instability is the scaphoid shift test, which elicits a pain and/or a click.
- Examination may also yield generalized ligamentous laxity.
- Fluoroscopy or advanced imaging with CT or MR arthrogram may be necessary for diagnosis. Arthroscopy can confirm the diagnosis.
- Surgical intervention is indicated for high-grade scapholunate ligament tears, while lower grade tears may be managed with splinting and PT [27, 28].

Neuropathology in Rowing

- Nerve entrapments occur in rowers and range from digital nerve compression in the fingers, carpal tunnel syndrome, or sciatica in the lower extremity.
- Digital nerve compression may be related to a tight grip with pressure localized to the neurovascular bundle. Equipment modification can alleviate symptoms.
- Carpal tunnel syndrome may also relate to a tight grip combined with repetitive wrist movements during feathering and squaring. Technique modification, loosening the grip, equipment changes, in addition to routine management usually alleviate symptoms.
- Radial and ulnar neuropathies may also occur in rowers given the repetitive motion of the upper extremities.
- Sciatica may occur as a result of an improperly fitted seat that places pressure on the sciatic nerve. Many seats have holes to accommodate the ischial tuberosities, and an improper fit may contribute to symptoms, especially in women given their wide pelvis [1, 29].

Parsonage–Turner Syndrome

- Although the pathophysiology is not entirely understood, a rower who presents with acute onset upper extremity pain lasting for several days to weeks, followed by weakness and muscle atrophy may be suffering from Parsonage–Turner syndrome, also known as acute brachial neuritis.
- Autoimmune, genetic, infectious, and mechanical processes have been implicated the pathophysiology.
- Symptoms vary widely and may include pain, paresthesias, and sensory disturbances.
- Pain is usually localized to the shoulder and often radiates into the arm or neck and aggravated by movement of the shoulder; symptoms are often unilateral, but bilateral pain followed by unilateral motor symptoms has been reported.
- Muscle weakness typically begins days to weeks after the onset of pain and often worsens after the pain subsides.
- EMG findings are often nondiagnostic and may provide more information 4–6 weeks after onset of symptoms.
- Imaging may be useful in ruling out other disorders, including a Pancoast tumor (plain radiography) or nerve root compression with MRI.
- Laboratory studies are generally not helpful in diagnosis.
- Treatment requires a multidisciplinary approach including pharmacologic therapy with oral corticosteroids and opioids or NSAIDs in addition to physical therapy; surgery may be required for cases refractory to conservative therapy [30].

Quadrilateral Space Syndrome (QSS)

- The quadrilateral space (QS) is bound superiorly by the teres minor or scapulohumeral capsule, inferiorly by the teres major and latissimus dorsi, laterally by the surgical neck of the humerus, and medially by the long head of the triceps.

- QSS develops from injury to the axillary nerve or to the posterior circumflex humeral artery (PCHA) as they pass through the quadrilateral space.
- Rowers are at risk given their repetitive shoulder movements.
- Neurogenic QSS, or nQSS, causes non-dermatomal neuropathic pain, numbness, and weakness that radiates down the arm. The etiology is likely the result of fibrous bands or space-occupying lesions.
- Vascular QSS, or vQSS, may result in thrombosis, microembolism, macroembolism, digital or hand ischemia, or signs/symptoms of acute ischemia. The etiology is likely related to repetitive mechanical injury to the PCHA as it passes through the QS.
- Evaluation includes CT angiography, MR angiography, ultrasound, EMG, or digital subtraction angiography (DSA).
- nQSS should be treated conservatively initially with NSAIDs and PT. Definitive treatment involves surgical neurolysis and QS decompression.
- vQSS treatment involves PCHA ligation with or without surgical thromboembolectomy and/or thrombolysis to prevent distal embolization. Anticoagulation should be considered for 3 months [31].

Thoracic Outlet Syndrome (TOS)

- Thoracic outlet syndrome occurs when the neurovascular (brachial plexus, subclavian artery or vein) bundle passing through the thoracic outlet is compressed. Repetitive upper extremity movements may predispose those already at risk to develop TOS.
- The thoracic outlet is the opening created by the first rib, the clavicle, the subclavius muscle, and costoclavicular ligament, and the anterior scalene.
- Etiologies include trauma, repetitive stress, or postural abnormalities.
- Those with an accessory rib or hypertrophied musculature in this region may be particular risk.
- Neurogenic symptoms include pain, paresthesias, weakness; vascular symptoms suggest compression of the subclavian artery or vein.
- Plain films may exclude an accessory rib; MRI/MRA are more useful in locating the neurovascular bundle and/or the presence of stenosis.
- NSAIDs and opioids have been used for pain management, while corticosteroid injections may be diagnostic and therapeutic.
- Surgery may be indicated for athletes with refractory symptoms [32].

Dermatoses in Rowing

- Handles should be cleaned so as not to expose open blisters to infection or spread infection to other athletes [1, 3, 12, 33].
- Repetitive trauma to wet, macerated, or traumatized hands may increase risk of inoculation with the papilloma virus, causing hand warts in crew athletes from boat oar handles [34].
- About 20 of 80 (25%) ofcrew members in one cohort reported they developed warts since beginning varsity crew, highlighting the importance of appropriate hygiene [34].
- Superficial abrasions at the MCP or PIP joints may occur in scullers resulting from impact from the opposing oar handle (known as "Sculler's knuckles") [1, 3, 12].
- "Slide bites" are abrasions that occur on the posterior lower leg resulting from contact between the skin and the track; protective layering with socks or tape prevent this common injury; these lesions should be monitored for infection [1, 3, 12].
- Abrasions from an improperly fitted seat are common and vary from irritation to ulcerations from repetitive chafing; management includes monitoring for infection, changing the seat, using a foam pad, and usage of petrolatum jelly [1, 3, 12].
- "Rower's rump" has been described and may develop from repetitive frictional contact of the buttocks resulting in lichen simplex chronicus. Avoidance of friction, topical corticosteroids, or tar preparations are used for therapy [35].
- Any open lesions while rowing should be monitored, and proper hygiene should be encouraged to prevent the spread of infectious disease through skin breaks.
- Sun protection is always encouraged. In one cohort, 44% of crew athletes never used sunscreen. Application of sunscreen is doubly important given the risk of exposure from sunlight reflected from the water [12, 36].
- Layering and a wind barrier may be necessary during colder months [12].

Trauma

- "Catching a crab" involves inadvertent contact between the water and oar at the incorrect phase of the stroke.
- This contact between the oar and the water typically causes the oar handle to abruptly move toward the rower often resulting in injuries related to impact. In certain instances, rowers can be ejected.

- Contact injuries related to contact with the oar handle may include contusions, concussions, and superficial skin injuries [13].
- An ejected rower may suffer consequences of cold-water exposure or other infectious exposures depending on the circumstances.
- If trauma occurs resulting in concussion, evaluate the athlete with a standardized approach.

Exercise-Associated Collapse (EAC), Syncope, Sudden Cardiac Death

- Rowing performance is associated with both aerobic and anaerobic capacity, resulting in unique hemodynamic alterations during exercise.
- Blood pressures as high as 200 mmHg at the catch phase results from high cardiac output, while working against high systemic vascular resistance [37].
- Despite this, there is no evidence that rowers are at increased risk for sudden cardiac death relative to athletes in other sports.
- Given the large number of competitors at rowing events, there is a possibility of EAC, syncope, or sudden cardiac death.
- Management protocols for EAC, syncope, or sudden cardiac death pose unique logistical challenges [38].
- As AED use is contraindicated on the water, the medical team should have an emergency action plan prepared to rescue collapsed rowers and get them rapidly to shore.
- Furthermore, an athlete that collapses while on the water is at risk of falling overboard, altering boat mechanics, or possibly causing capsize [2].

Cold-Water Immersion

- Flotation devices must be easily available for all rowers.
- Priority for rescue should be given to smaller and leaner individuals in cold-water rescue scenarios.
- After triage, athletes with the most severe hypothermia should be attended to first.
- In potential cold-water rescue situations, heating devices should be available, including heating pads/packs and forced air warming devices.
- Cardiopulmonary bypass, if available, should be initiated for hypothermic patients in asystole or ventricular fibrillation [39].

Coverage of Rowing Events

- Team physicians should familiarize themselves with common musculoskeletal injuries that occur among rowers, especially those that occur at the lower back, shoulder, and upper extremity.
- Dermatopathology is common, and sideline medical personnel should be prepared to care for a variety of skin conditions.
- Though more obscure, neurologic and vascular pathologies occur.
- Emergency action plan (EAP) should be developed for possible traumatic events.
- EAP consensus for traumatic events, EAC, syncope, cold-water immersion, and sudden cardiac arrest in rowing are in their early stages of development. In general, an athlete should be removed from the water if immersed and assessed quickly. Harbor patrol may be required for more significant emergencies.
- Personal flotation devices should be available for all rowers.

Return to Play

- As most injuries in rowing involve overuse, rehabilitation must focus on appropriate form (biomechanics) and rowing mechanics to avoid future injury.
- Only athletes that are completely pain free from rib stress fractures may return to rowing.
- Appropriate attention to skin conditions is required in order to prevent further deterioration, complications of infection, or spreading of infectious organisms to other athletes.
- Although less prevalent, awareness of possible neurologic and vascular injuries expedites appropriate diagnosis, management, and return to play.

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Rugby

67

Darcy Selenke

> **Key Points**
> - Rugby is a contact sport centered around the movement of an oval ball down a field to a goal line.
> - It is a high-velocity game, and no specific protective gear is worn.
> - Most injuries are associated with tackling.
> - Lower limb injuries occur more often than other body parts.
> - Most available injury data comes from Rugby Union at the professional level.
> - Concussions are a common injury.

Introduction

Rugby is a contact sport involving an oval ball that evolved from the Greek and Roman games that involved kicking an air-filled ball. It was developed in England in the 1800s. It is the precursor to Canadian and American Gridiron Football. There are two forms of the game, Rugby Union and Rugby League. They differ in rules concerning how possession is maintained or given up. Rugby Union has scrum, mauls, and rucks, but Rugby League does not. Both games use passing of the ball, tackling and kicking of the football to maintain possession of the ball and score. Scoring requires that the ball cross a goal line at opposing ends of the field of play. There are a few unique play formations in rugby. These include mauls, rucks, and scrums. A maul is when the ball carrier joins with teammates to push the ball down the field, and the opposing team members also join together to oppose the other team (Fig. 67.1). A ruck is when the ball carrier is tackled and places the ball on the ground, and then his teammates defend the ball by pushing away the defensive players. A scrum is when the opposing teams line up pushing against each other to take possession of the ball after a penalty that stopped play.

Players are divided into back row and front row positions. In the back row are a flanker, an eighthman, and two locks. The front row consists of hookers and props. The flanker's job is to tackle the opposition and try to steal the ball. The eighthman is a utilitarian position that can move the ball out of the scrum, carry the ball, and provide extra force in the scrum. Locks are usually the tallest players, provide force from behind to the front line during the scrum, and are targeted to catch the ball during a line out. Hookers are in the middle of the front row during a scrum, locked arm and arm with the props, and try to hook the ball toward the line with their foot or disrupt the other team's attempt at getting the ball in the scrum. Hookers often carry the ball upfield during open play. Props are the force on the front line and provide stability during the scrum. During a line-out, the props support the jumpers that are trying to get high enough to acquire the ball.

Most games are played on grass or turf. There is not specific protective gear worn except optional mouthguards and scrum caps (that protect ears).

Injury Epidemiology

Injury patterns and incidents vary across different studies, gender and levels of rugby play and include head to toe injuries. The most comprehensive injury data at the professional level is the England Professional Rugby Injury Surveillance Project (EPRIS) [1]. The latest published data is from the 2015 to 2016 season. The overall incident of injuries has slowly declined from 100/1000 h played to 62/1000 since 2002. The severity of injury has also decreased. This is thought to be the result of formal concussion recognition programs, introduction of a new

D. Selenke (✉)
Department of Family Medicine, University of Colorado School of Medicine, Denver Health, Winter Park Medical Center, Winter Park, CO, USA
e-mail: Darcy.Selenke@dhha.org

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Fig. 67.1 Maul. (Reprinted with permission from iStock#535116333)

scrum engagement sequence and changes in the line out process of bringing the ball back into play after it has gone out of bounds. During play, the causes of injury observed in the EPRIS study are as follows: tackling (45%), collision (15%), running (9%), ruck (8%), scrum (4%), maul (3%), line out (2%), and unknown (10%) [1].

The most common cause of injury is during tackling at all levels of rugby. The EPRIS findings show that the ball carrier suffers the injury at a slightly higher rate that the tackler [1]. The distribution of injuries during tackling for the ball carrier is concussion (20%), MCL injury (7%), ACL injury (7%), and ankle syndesmosis sprain (4%) [1]. Injuries of the tackler are concussion (47%), ACJ injury (7%), cervical spine stinger/burner (3%), and elbow hyperextension (2%) [1]. Tackles from the side caused the highest injury rate. Increasing tackling technique proficiency has been shown to lower injury rates [2–5].

Concussion rates have increased over the past 20 years, and it is unclear if that is a result of purely a higher rate of injury, improved reporting, or better recognition of concussion. The latter reasons are the most probable. Concussion rates vary across studies from 3% to 17% of all injuries and have become the most common rugby injury. Concussions occur from head to head, head to body, and head to ground mechanisms. In 2015, World Rugby adopted the head injury assessment into law, making it a standard in on field concussion recognition and management [6]. These measures dictate that any player with a suspected head injury sit out at least 10 minutes and receive a side-line assessment. Any player with confirmed concussion signs or symptoms cannot return to play for at least 6 days, and further cognitive assessment such at the SCAT tool needs to be implemented in their return to play treatment plan. Adolescent concussion rates range from 0.2 to 6.9 per 1000 player hours for rugby union and 4.6–14.7 per 1000 player hours for rugby league [7].

In general, just over half of injuries in rugby are closed soft tissue injuries [8]. Injuries are divided among capsular/ligament sprains or tears (20–34%), muscle/tendon strains or tears (20–29%), open wounds (12–27%), bruises/hematomas (10–22%), fractures (4–14%), and lastly concussion (3–25%) [8]. Hamstring injuries are the most commonly seen and highest risk soft tissue injury. Acromioclavicular joint, ankle lateral ligament, ACL, and calf injuries are also quite common. Dislocations of the shoulder and finger occur at lower rates, but require immediate sideline management. Clavicle fractures are the most common type of fracture [8].

Spinal cord injuries are a very small portion of rugby injuries. Most commonly, cervical cord neurapraxia (cervical "stinger") occur with direct axial load to the top of the head. These are self-limited events that do not lead to persistent neurologic symptoms. Acute spinal cord injuries with permanent neurological sequelae and traumatic brain injury was reported as 4.25/100,000 players by a study in South Africa from 2008 to 2011 [9]. In the lumbar region, violent collisions with or without existing disk herniation can lead to paraplegia. The recent changes in rules regarding how a player enters the scrum have decreased serious spinal injuries [9].

Injuries to the torso occur frequently and must be accurately assessed. The rate of thoracic injuries is 8.3/1000 player hours [10]. Rib, clavicle, and scapular fractures can be associated with significant vascular and internal organ injury. Players with shortness of breath, thoracic bony tenderness, signs of internal organ damage, or persistent pain need to be kept out of the game and referred to a higher level of medical assessment and care. These injuries often require x-ray and CT scan evaluation to understand the full breadth of injury [10].

Injury rates are similar forward and back. However, it is a common belief that the seriousness of injuries is higher for forwards. This is thought to be due to higher rates of high-velocity collisions. The specific data on injuries by position is varied and inconclusive [8].

In adolescent rugby, higher weight and higher levels of competition were associated with higher rates of injury [11]. Concussions lead to the most time out of play [11]. The most common sites of injury are head/face, clavicle/shoulder, and knee. This was followed closely by ankle injuries [11].

There is limited data available in terms of women's rugby injuries. Overall women rugby players have a lower injury rate than men. The most common injury in women's rugby is knee followed by ankle injuries [12]. Concussion rates are also lower for women players as opposed to men [12].

Preparation for sideline providers should be the same for both sexes and all age groups. Programs such as Boksmart, a youth injury prevention program in South Africa, have been shown to lower injury rates [2]. There has been no definitive evidence showing a difference in injury rates or patterns when comparing play on turf versus grass [13]. Highest rate of injury occurs in the second and fourth quarters [13].

What Is Unique About Rugby Injuries?

- The high physical velocity and high level of contact of the game lends itself to all types of traumatic athletic mechanisms.
- No protective equipment is required.
- Higher skill/proficiency of tackling leads to lower injury rates.

What Do the Physicians Need to Know While Covering a Rugby Game on the Sideline?

- Be prepared to evaluate and treat the lower extremity, shoulder, and finger injuries, skin wounds, and blunt chest trauma.
- Know how to reduce shoulder and finger dislocations.
- Know how to assess extent of blunt chest trauma.
- Be aware of concussion assessment and return to play protocols.
- Be equipped to clean, dress, and possibly repair skin wounds.

General Rule About Return to Play

- General concussion protocol should be applied.
- Players with joint injuries that require dislocation reduction cannot return to play until further evaluation.
- Players with complete ligamentous ruptures cannot return to play.
- Players with finger and mild soft tissue injuries can be taped or splinted for return to play.
- Athletes with neck stingers may return to play after all neck pain and neurologic symptoms have resolved.
- Any blunt chest trauma resulting in shortness of breath, persistent pain, or bony tenderness requires further evaluation, and the player should not return to play.

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Sailing

68

Jeremy D. Close and Hannah P. Leahy

Key Points

- Highly variable injuries exist, given the type of craft, the position of the crew member, and the weather or environmental conditions.
- Sailors are at risk of both acute injury and chronic overuse injury.
- Low back injuries are common in sailing.
- Heavy weather is the most significant risk factor for acute injury during sailing.
- Bowman are at the highest risk of acute injury and subject to the most physiologic stress such as hyperthermia or dehydration.
- Most injuries are related to trip or fall events and can be in part avoided by proper communication and equipment.

Introduction

Sailing has been done for thousands of years as a means of traveling. It was first widely recognized as a sport in 1851 with the start of America's Cup competition. It was included in Olympic competitions as early as 1896. It has become a very popular sport over the last 20 years due to increases in sponsorship, commercialization, and media attention [1].

It requires great technical skill as well as physical and mental endurance to compete in sailing races. The environmental conditions are a large variable that sailors must face, which can greatly impact their risk of injury.

Table 68.1 Types of sailing crafts

Keelboat (Fig. 68.1)	Dinghy (Fig. 68.2)
>2 crew members	1–2 crew members
Weighted keel provides stability	Body mass of crew provides stability

There are two types of competitive sailing crafts, keelboats and dinghies (Table 68.1). Keelboats are typically larger than dinghies. They tend to be more stable due to the weighted keel (Fig. 68.1). Dinghy racing is done in relatively small boats. This type of boat relies upon a motion called hiking, which is a technique that uses body weight to keep the boat upright (Fig. 68.2). This motion requires a lot of leverage and strength.

Injury Epidemiology

The most common injuries in sailing are sprains, lacerations, contusions, and fractures. The most common mechanism of injury is trip/fall and getting caught in the lines (Table 68.2). Injuries are typically related to sail change or crossing over the centerline while tacking (Table 68.3). Rates of injury have varied in the literature from 0.42 per 1000 h to 2.2 per 1000 h [1, 3]. The location and type of injury varies based on experience of the sailor, boat type, boat position, and weather conditions. Inclement weather has been found to contribute to 23% of injuries [1].

Sailor Experience

Inexperienced or recreational sailors tend to injure their hands or head more frequently, whereas more experienced sailors tend to suffer from chronic strains, sprains, or tendinopathies [1]. In a study of beginner dinghy sailors, not

J. D. Close (✉)
Department of Family and Community Medicine, Sports Medicine, Thomas Jefferson University, Philadelphia, PA, USA
e-mail: Jeremy.close@jefferson.edu

H. P. Leahy
Department of Physical Medicine and Rehabilitation, Wellspan Health, Gettysburg, PA, USA

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Fig. 68.1 A dinghy boat. (Reprinted with permission from iStock – photo ID:479579196)

Fig. 68.2 A keelboat. (Reprinted with permission from iStock – photo ID:475069301)

Table 68.2 Mechanism of injury (by boat type)

Mechanism of injury	Keelboat (%)	Dinghy (%)
Trip/fall	30	19
Caught in lines	22	12
Hit by object	21	21
Winch	8	0
Fall overboard	2	4
N/A	7	6
Other	10	38

Adapted from Nathanson et al. [2]

Table 68.3 Activity associated with acute injury during sailing

Activity	Keelboat (%)	Dinghy (%)
Sail change	18	3
Crossing centerline during tack	17	14
Tailing	10	<1
Grinding	9	0
Walking	8	1
Steering	8	20
Hiking	5	21
Climbing mast	1	<1
Trapeze	0	8
N/A	5	5
Other	18	24

Adapted from Nathanson et al. [2]

Table 68.4 Most common sailing injuries categorized by boat type

Boat	Most common injury	Second most common injury	Third most common injury
Keelboats (e.g., America's Cup, Volvo Ocean Race)	Upper extremities	Spine and neck	Hip and pelvis
Dinghies	Leg contusions	Knee contusions	Leg lacerations

only the rates of injuries were similar between men and women but also the types of injuries sustained [4]. This has not been well studied in a more experienced population of sailors.

Boat Type

The data around injury types by boat size and type has varied depending on resources used to collect data. A self-reported injury study by Nathanson et al. showed dinghy sailing tended to have more lower extremity injuries [2]. But large keelboats in the America's Cup showed to have more upper extremity and neck injuries (Table 68.4) [5].

Boat Position

The various positions on a sailing vessel and location of the boat can put the athlete at risk for different injuries. These injuries can be both musculoskeletal and environmental. Grinders, bowmen, and helmsmen seem to have the highest rates of injury, but this can vary based on types of racing [1, 6]. Bowmen had the greatest risk of hyperthermia and dehydration. The risk of dehydration will change based on upwind or downwind sailing [7]. A review by Nathanson et al. showed that the risk of injury occurred in the middeck, followed by foredeck for keelboats and midship for dinghies [2].

Severity of Injuries

The risk of severe injury is very low with a rate of 0.56 per 1000 days of sailing. Of severe injuries that occur, 25% are fractures, 16% torn tendons or cartilage, 14% concussions, and 8% dislocations. The locations of severe injuries are 24% to the head, 15% to the knee, 10% to the leg, and 9% to the arm [2]. Death related to sailing is often related to falls overboard and capsizing. The fatality rates for all sailing related death are calculated at 1.19 deaths per million sailing person-days, the majority being from drowning and not wearing a personal flotation device [8]. Head and neck injuries can be some of the more severe injuries. They account for 13% of all injuries. These are typically related to boom hits from unaware sailor or helm error. These could be prevented by a proper hatch cover [9].

Other Populations

Paralympic sailing was introduced into the games in 1996 first as a demonstration sport. By 2000, official competitions included two different boats, the single and three-person keelboat, and by 2008, a two-person keelboat competition was added. Rates of injury in sailors with disabilities are similar to those of able-bodied sailors according to limited data [1].

What Is Unique About Sailing Injuries?

- The variability in types of sailing boats and crew member position results in a wide variety of injuries.
- Duration of competition can be a challenge to ensure adequate hydration and nutrition during the competition [6, 10].

- Sailors need to sustain unnatural positions (i.e., hiking).
- Back pain especially is common in dinghy hiking.
- Illnesses are usually due to viruses, hyper or hypothermia, dehydration, or motion sickness.
- Aware of weight restrictions (encourage safe, gradual weight loss programs). Ensure athletes are within ideal body weight range. Watch for female athlete triad.
- High rates of dietary supplement use have been reported (77% of high-level sailing athletes, 38% on daily basis, more common in single crew competitors) [10].
- The risk of sunburn is high due to open water, causing reflection. Appropriate protective equipment is extremely important (not only sunscreen but also hats, long-sleeve shirts, and proper eye protection from UV light) [1].

What Do the Physicians Need to Know While Covering an Event?

- Most life-threatening injury would be head injury resulting in loss of consciousness as they are at risk of drowning.
- Rates of life jacket wear are very low, and use should be encouraged prior to activity.
- Monitor fluid intake and for early signs of heat illness.
- Avoid overdressing when sailing upwind.
- Proper equipment such as nonslip shoes and breathable fabric is recommended.
- Teams should practice and execute good communication techniques to minimize injuries related to unawareness.
- Proper foot wear is important for preventing foot injuries [9].
- Consider boat design when assessing injury risk (reducing below deck temp and increased ventilation).
- Acute injuries occurring on board cannot be evaluated immediately given the physician is usually on shore. Prompt evaluation upon arriving on shore is important given the inherent delay in care.
- May need to coordinate with the coast guard or marine units to facilitate care of sailors. These units should be alerted that the race is happening.
- Establish how to directly communicate with the boat (radio, hand signals).
- Factor in the extra time needed for marine transport as compared with ground transport when deciding upon management of acute injuries.
- For races, especially those lasting longer periods of time, such as offshore or endurance racing, having a record of all sailors with their medical history, medications, and allergies is necessary for expediting medical care or coordinating telemedicine.

General Rule About Return to Play

- Injured athletes that require more frequent monitoring should be limited in long-duration events.
- Recent recurrent loss of consciousness is a contraindication to return to play.
- Concussions are a common injury in sailors, and these athletes must follow the standardized athlete return to play concussion algorithm adapted to a sailing format.
- Evaluate joints for limited mobility that put people at risk for injury.

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Skiing: Alpine

69

J. Herbert Stevenson, Kimberly Sikule, and Elana Bannerman

Key Points

- Physicians taking care of ski-related injuries should be familiar and competent with different levels of trauma.
- Alpine ski injury patterns have changed over time which seems to be most likely due to the changes in the rules, regulations, equipment, and course structure.
- Sprain of the ulnar collateral ligament (UCL) of the first MCPJ (skier's thumb) is the most common upper extremity injury sustained by alpine skiers.
- Knee is the most common lower extremity injury sustained by alpine ski racers accounting for 36% of the injuries.
- Managing medical coverage of an alpine ski event involves coordination of on-site facilities, staff, EMS, and an appropriate trauma center.

Introduction

Alpine skiing is an exciting sport with fast movement and high forces (Fig. 69.1). In super-G and downhill, injuries are mainly related to higher speed and jumps, while in slalom and giant slalom, they are related to high loads created by a combination of turn speed and turn radius [1]. Interestingly, the injury patterns have changed over time and likely correlate with the rules and regulations governing equipment and course structure. This chapter reviews key points for covering mass ski events and treating acute ski injuries.

Common Injuries

- *Head:* Head injuries, including concussion, comprise a significant amount of alpine skiing injuries. Skier education, as well as several high-profile skiing deaths, has led to the increase in helmet use among downhill skiers. Interestingly, while several studies have concluded that the rise of helmets has correlated with the decrease in overall head injuries, the incidence of concussion has not declined [2]. Following suspicion of a concussion, a standard evaluation and treatment protocol should be followed. A thorough examination should be performed to rule out more severe brain injury. Then, the athlete should be restricted from athletics, including skiing, until symptoms have resolved and return to sports protocol is completed. Prior to resuming alpine skiing, the skier should be cautioned that sustaining a "second impact" in close proximity to the first could be catastrophic [3].
- *Shoulder:* Shoulder injuries account for 4–11% of ski injuries [4]. Rotator cuff injuries are the most common, accounting for 24% of shoulder injuries [4]. These can be caused by forced shoulder abduction or excessive external rotation due to falling while still attached to one's ski pole. Unless there is concern for a complete rotator cuff tear, these injuries are generally managed conservatively. Anterior GHJ dislocations/subluxations are the second most common at 22% [4]. These can be sustained through the same mechanism as rotator cuff injuries. They should be reduced in a timely manner to reduce the risk of neurovascular injuries. ACJ sprains (separations) make up 20% of shoulder injuries and are due to a direct medial blow to

J. H. Stevenson (✉)
Department of Family Medicine, Division of Sports Medicine, University of Massachusetts Medical Center, Worcester, MA, USA
e-mail: john.stevenson@umassmemorial.org

K. Sikule
Department of Family Medicine, University of Vermont, Burlington, VT, USA

E. Bannerman
Department of Family Medicine, Primary Care Sports Medicine, Southern New Hampshire Medical Center, Hudson, NH, USA

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Fig. 69.1 Alpine skiing

the lateral shoulder [4]. Clavicle fractures make up 11% of shoulder injuries and occur with a direct impact to the anterior shoulder [4].

- *Hand/wrist:* The most common upper extremity injury sustained by alpine skiers is a UCL sprain of the first MCPJ, appropriately termed "skier's thumb." This injury accounts for 8% of all injuries and occurs when a skier falls on an outstretched hand and the thumb becomes caught in the pole straps [5]. Complete UCL tears should be referred for potential operative management. Partial tears should be immobilized in a thumb spica cast for 4 weeks [5].
- *Knee:* In general, lower extremity injuries are more common in skiers than snow boarders due to their equipment including rigid ski boots. The types of lower extremity injuries in Alpine skiers has changed since the 1970s with changes in design of boots and bindings to prevent fractures and ankle sprains. This in combination with expert skiers/racers increasing the din to prevent binding release has led to an increase in ACL injuries [6].

  The most common lower extremity injury sustained by alpine ski racers is the knee (36%), from which an ACL rupture accounting for 14% of all injuries during the 2006–2008 seasons with higher rates at increased speed [6]. Injury rates expressed as injuries per 1000 ski runs were lowest for the slower events of slalom (4.9/1000) and giant slalom (9.2/1000). The faster speed events of super giant slalom and downhill had the highest rates (11.0/1000 and 17.2/1000, respectively) [1]. The International Ski Federation has made regulations regarding side-cut radius and ski length for Alpine skiing to decrease the incidence of ACL tears which can happen when forces are applied to the knee when it is in valgus and internally rotated. It is unclear at this point if these regulations have helped prevent ACL injuries [7]. Treatment of suspected ACL injuries on the hillside includes immobilization and further evaluation by the medical team.
- *Lower leg:* Another common lower extremity injury is tibia–fibula fracture, especially with hard shell boots, causing force to be applied at the top of the boot. When looking at the National Data Bank for 2011–2012, tibia–fibula fractures make up 25% of lower extremity fractures [8]. From those 10% are proximal fractures, 12% are shaft fractures, and 2.9% are ankle fractures [8]. Suspicion for tibia shaft fractures should be high in a lower energy fall with twisting motion on a fixed foot in a ski binding. Proximal shaft fractures are seen in greater rates at higher forces. Tibia shaft fractures present with inability to bear weight. Patients with tibia fractures should have a good vascular assessment and should be splinted given swelling and risk of acute compartment syndrome. Skier should undergo direct transport from the mountain for open fractures or fractures with neurovascular deficits. If there is significant displacement, then the skier should see an orthopedic surgeon in the next 2 days and can be splinted in a long-leg splint for protected immobilization.
- *Chest wall:* Chest wall injures are involved in 48.5% of severe ski injuries [9]. In Alpine skiing, 31% of chest injuries are due to rib fractures and 23.4% result in lung injuries [9]. Fractures of the ribs are caused by blunt trauma with falls or collision to another skier or objects such as trees. Rib fractures can be diagnosed with plain radiography; however, a negative examination does not exclude the rib fracture. Inspiratory and expiratory chest

Fig. 69.2 Backcountry skiing

radiography may help detect a small pneumothorax. In cases of pneumothorax with other injuries and the need for air evacuation with a helicopter, a chest tube should be placed.

Event Coverage

- Preparation – Covering a large ski event requires a cohesive team of specialized individuals including ski patrol, physicians, and EMS with the ability to directly transport injured athletes to a trauma center. Medical personnel should be trained and competent in the management of acute trauma including airway management and fracture stabilization. If diagnostic capabilities are available expertise in reading plain radiographs and ultrasound imaging, including the FAST (Focused Assessment with Sonography in Trauma) examination, is important.

  An action plan should be created and reviewed with your medical team. This should include the chain of command and the ski-specific triage system for injuries. Each individual's role should be reviewed. For example, it should be the responsibility of the ski patrol to transport the injured skier to the medical tent or ski clinic for advanced medical care. The emergency action plan should also include key logistical information including a course map along with the locations and capabilities each medical station. Medical stations should be placed in logical areas on the course, such as the finish line.

  Depending on the ski area location and facilities available, medical stations may include formal ski clinics. Ski clinics will often have onsite plain radiography and ultrasound for rapid imaging. A tertiary medical center will need to be identified and transportation protocol coordinated. Acute life-threatening trauma often necessitates air transport via helicopter, while less acute injuries may be transported via EMS.
- Environmental exposure – Environmental exposure includes sun, cold, and altitude exposures (Fig. 69.2). Cold exposures are uncommon, as most expert skiers have trained in these conditions for years and use appropriate protective equipment. However, frostbite can be common in spectators and event workers given their prolonged exposure to the elements. Sun exposures, interestingly, are vastly overlooked due to the perception that sunburns will not be an issue in the cold weather. However, the combination of the reflection of UV rays off of the snow and the high altitude can lead to harmful sun injuries. The use of appropriate eye wears including sunglasses or goggles that block UV light along with high SPF sunscreen is essential. Altitude sickness can also be a problem for competitive skiers, as it may take several weeks to acclimatize.
- Triage and transportation – If the physician is an expert skier, he or she should be positioned at the top of the hill, prepared to ski to injured skiers. His or her medical bag should include equipment to manage airways, stop bleeding, and stabilize fractures. Ski patrol should also attend the patient on the hill. They should be prepared to provide spinal stabilization and have an AED. Once the skier is stabilized, ski patrol is responsible for the transport of the patient to the medical clinic or tent. From there, definitive care can be provided, or the patient can be transferred to a hospital via EMS, who should be standing by.

Return to Sports

Return to sport is dependent on the underlying injury. Skiers with minor environmental exposure injuries or minor strains and sprains may return to skiing once symptoms have improved or resolved. Head trauma, including concussions, will require longer recovery dependent on severity. Concussed athletes cannot return to sport until symptom free, have a normal neuro-ocular-cognitive examination, and are cleared by a physician or an approved medical professional with expertise in the care of concussed athletes. Many high-velocity injuries include multiple sites affected and sig-

nificant trauma. These are often season-ending injuries that require extensive recovery time and potential surgical intervention.

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Skiing: Cross-Country

70

Kyle B. Nagle

Key Points

- Cross-country skiing is a low injury risk sport.
- Most cross-country ski injuries are due to overuse.
- Low back pain is more common in cross-country skiers than the general population.
- Traumatic injuries, while rare, do occasionally occur.
- Environmental conditions, particularly cold injury and illness, as well as nutritional and hydration concerns should be prepared for by participants and medical providers to prevent injury.

Introduction

Cross-country skiing, also known as Nordic skiing, incorporates a wide variety of ski techniques and equipment (Fig. 70.1). For the purposes of this chapter, cross-country skiing is considered skiing on groomed trails using lightweight skis without metal edges, boots, and poles for the purpose of racing or fitness. Techniques further on the spectrum toward backcountry and lift service telemark skiing exhibit injury patterns closer to alpine or downhill skiing. Cross-country skiing is generally considered an endurance sport with races often 5–50 km long. Races over 40 km long are often referred to as ski marathons. The duration of the races depends on snow conditions, course factors such as elevation gain or loss, and skier ability and conditioning. Elite level skiers will complete 5-km races in just over 10 min and 50-km races in around 2 h. In recent years, sprint racing has become more common, with skiers competing in multiple heats over a 1- to 2-km course taking 3–4 min to complete.

Within cross-country skiing, there are two techniques, skating (where the skis move outward at an angle to the overall direction of travel in an ice skating type motion) and classic (where the skis are aligned parallel to the direction of travel and use a specific kind of ski wax to gain traction on the snow and thus propulsion). This is similar to swimming which has different strokes (freestyle, back stroke, butterfly, and breast stroke) with separate competitions for each stroke. Within each technique of skiing, there are multiple variations that are used depending on the terrain and speed of the skier. In classical technique, the variations include (from faster to slower conditions) double pole (skis remain parallel and relatively in line with each other, while propulsion is produced purely from the poles hitting the ground at the same time), kick double pole (one leg kick followed by a double pole with both poles hitting the snow at the same time), diagonal stride (contralateral arm and leg moving forward, skis parallel, with a distinct glide phase while balanced on one ski), and herringbone (contralateral arm and leg moving forward together with the skis angled with the tips wider apart than the tails, but no gliding on each ski, used for steep terrain). Skating employs different variations including (from fastest to slowest conditions) free skate (skating without poling), V2 alternate (both poles hitting the snow at the same time, just before the leg pushing to the side in a skating motion, poling only with the leg pushes on one side), V2 (both poles hitting the snow just before each leg push, poling with each leg stride), and V1 (both poles hitting the ground at the same time as one ski, without poling on the other leg stride). Typically races range from approximately 1-km sprints to over 50-km-long marathons. In most cases, propulsive forces are created by both upper and lower body movements, leading to a high aerobic quality to the exercise in addition to the muscular endurance and strength needed to develop high forces over a long period of time.

K. B. Nagle (✉)
Departments of Pediatrics, Orthopedics, and Rehabilitation, Seattle Children's and University of Washington School of Medicine, Seattle, WA, USA
e-mail: kyle.nagle@seattlechildrens.org

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Fig. 70.1 Skiing: cross-country

Cross-country skiing has evolved greatly over the past three decades. New techniques, including skating, and race formats have changed the appearance of the sport. Training developments have also led to changes in how races are skied, including more marathons being raced exclusively using the double pole classic technique without any kick wax. These changes have caused some changes in injury patterns observed in the sport.

Epidemiology

Cross-country skiing is a low-risk sport with high aerobic demands but overall low incidence of injuries [1]. Many injuries are overuse type, though occasionally acute traumatic injuries do occur, usually associated with high-speed falls. Injury rates are 0.1–0.8 injuries per 1000 skier days with similar injury rates between skate and classic technique [2, 3]. A more recent study reported injury rates of 0.51 per 1000 skier days in recreational skiers, while competitive skiers had a lower injury rate of 0.02 to 0.09 per 1000 skier days. Female and male recreational skiers had relatively similar injury rates, 0.65 and 0.40 per 1000 skier days, respectively [4]. Most cross-country ski injuries are overuse injuries. In high-level skiers, there is a prevalence of 8% for overuse knee injuries, 5% for the low back, 1% for the shoulder, and 12% for the anterior thigh. The prevalence of overuse injuries causing moderate to significant decreases in performance or training time is 1% for the knee, low back, and shoulder, but 7% for the anterior thigh [5]. Chronic exertional compartment syndrome of the lower legs in cross-country skiing, particularly with skate technique, also seems to have a relatively high incidence compared with other sports. De Quervain's tenosynovitis and intersection syndrome can also be seen due to the wrist motions during the recovery phase of poling.

When compared with other International Ski Federation (FIS) disciplines—alpine, snowboard, freestyle, jumping, and Nordic combined (cross-country skiing combined with ski jumping)—cross-country had the lowest injury rate (11.4 injuries per 100 athlete seasons) and the lowest rate of severe injuries requiring more than 28 days to return to sports (0.7 injuries per 100 athlete seasons) [6]. This study also reported an injury rate in world cup cross-country skiers of 0.2 and 0.1 injuries per 1000 km skied by men and women, respectively [7]. Just over half of injuries to cross-country skiers occurred while training on snow, and an additional one-third of injuries occurred while racing in world cup or world championship races. About 41% of on-snow injuries were considered slight, requiring no missed time from training or racing. The most common types of injuries in world-cup-level skiers included muscle and tendon injuries (38%), joint and ligament injuries (31%), and contusions (15%). The most common body area injured was the lower back, sacrum, or pelvis, followed by the shoulder and clavicle, and then lower leg and Achilles tendon injuries [6].

Low back pain is more common in cross-country skiers than in the general population [8]. Risk factors for low back pain include increased training volume (10 h per week compared with 6 h per week) [9]. There does not seem to be a sex difference between male and female skiers in developing back pain [2]. Technique (classic or skating) as a risk factor for back pain has an inconsistent role. One study showed back pain to be more common with classic skiing, while other studies report technique not being a factor generally, though specific individuals may find pain to occur more consistently with one technique or the other [2, 8, 9].

Cross-country skiers employ a variety of training methods, ranging from on-snow skiing to roller skiing (skiing with wheels on paved roads and trails) to biking, running, and strength training. Injuries associated with dryland training are similar to athletes engaged in those primary activities. Overuse elbow injuries are not uncommon, particularly with dryland training such as roller skiing. In this activity, ski poles are used on pavement, causing increased ground reaction forces at the upper extremity compared with skiing on snow. Specific risk factors for overuse injuries in cross-country skiers in addition to usual predisposing risk factors include training over 700 h per year (twice the risk of injury compared to less than 700 h per year) and having less than two rest days per week (increasing the risk of overuse injury by five times) [10].

While fractures are infrequently reported in the cross-country ski literature, occasional fractures do occur. Ankle, wrist, and clavicle fractures do occur, usually as the result of falls. Rib fractures have occurred, with one highly publicized case during an Olympic Games resulting in a pneumothorax. With more frequent mass start races where all participants start at the same time, there are also a few reports of traumatic injuries resulting from collisions with other skiers and their equipment. There have been a few reported cases in recent years of collisions between skiers, resulting in ski tips puncturing the chest wall leading to open chest wounds. Also, the sharp tips of ski poles can cause lacerations of the face and hands. Eye protection in the form of sunglasses can help prevent eye injuries from ski pole tips.

Other Considerations

Due to the physical environment most often encountered in cross-country skiing, cold illness and injury should be taken into consideration. Hypothermia can occur as well as frostbite, particularly in longer events with high wind chill factors. Frostbite usually occurs at the distal extremities or areas exposed to wind, such as the nose and cheeks. The International Ski Federation (FIS) has set a lower limit of −20 °C for races, but other conditions such as high wind, snowfall, or even high temperature in some cases can be the cause for modifying or even cancelling events. Nutrition and hydration should also be considered, particularly in longer distance events such as marathons, in a similar manner as running races.

What Is Unique About Cross-Country Ski Injuries?

- Cross-country skiing is a high aerobic demand sport that involves both the upper and lower body, often over long distances.
- Most musculoskeletal injuries to cross-country skiers are due to overuse. Traumatic injuries are rare, but they do occasionally occur.
- Cold injury and illness can occur due to the climates where snow is generally found.

What Do the Physicians Need to Know While Covering a Cross-Country Ski Event?

- Due to the location and often extended courses over which races take place, special consideration should be taken regarding how to cover the entire course for injuries or illnesses.
- Consideration should also be taken to ensure the ability to reach injured athletes and transport the injured athlete to a safe place for further evaluation and treatment as needed.
- Environmental conditions should be considered when planning for the race, including using temperature and wind chill and snowfall rates, to determine timing of the start, delaying the start, shortening the course, or cancelling the race if needed for the safety of the participants.

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Snowboarding

71

Karin VanBaak

Key Points

- Snowboarding is increasing in popularity.
- Injury rates for snowboarding have continued to increase.
- Injury rates for snowboarding are currently higher than for skiing.
- Most snowboarding injuries occur to the upper extremity, particularly the wrist.
- Injury severity and distribution varies based on skill level.
- Recreational snowboarders are most likely to sustain a wrist sprain or fracture.
- Injury profiles for high-profile professional snowboarders look more like skiers with higher rates of lower extremity, specifically knee injuries than recreational athletes.

Introduction

The sport of snowboarding was started in the 1970s and popularized in the 1980s [1, 2]. Long viewed skeptically at traditional alpine resorts, the sport has become more and more popular. During the 2012–2013 season, the United States saw 7.3 million individuals participate in snowboarding, and rates continue to increase [3]. Snowboarding debuted as an Olympic sport in 1998 [1]. Injury patterns vary based on a participant's level of skill, venue of participation, terrain, and equipment (Figs. 71.1 and Fig. 71.2).

K. VanBaak (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Aurora, CO, USA
e-mail: karin.vanbaak@cuanschutz.edu

Injury Epidemiology

Most information that we have on snowboarding injuries come from studies looking at the differences between skiing and snowboarding injuries. Best current data shows that snowboarders are more likely to sustain an acute injury than skiers [3].

Published injury rates for snowboarding vary from 1.16 to 4.2 injuries per 1000 snowboard days. Incidence defined this way is two to three times higher than typical injury rate for skiing [3].

Despite variations in the literature, it appears that upper injuries represent approximately 45% of the total snowboard injury burden and lower extremity injuries represent 23% of all injuries [3].

Echoing this theme, several studies have found male snowboarders with higher rates of injury [4–6], while others have found female snowboarders with higher injury rates [7–9]. When severe snowboard injuries are examined, the majority of these appear to occur in males (86%) [10].

Examining specific injuries, wrist injuries are most common overall (17.7–27.6% of all injuries) and most common upper extremity injury [1, 3, 7]. Head/neck injuries are also common [3, 9].

Comparison Between Skiers and Snowboarders

Because of the varying ways that injury incidence has been defined, it is difficult to compare injury incidence over time. However, it does appear that injury rates for snowboarding

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Fig. 71.1 Snowboarding

Fig. 71.2 Backcountry snowboarding

have continued to increase over the past 10 years, while skiing injury rates have stabilized [3]. As an example, current terrain park injuries are more likely to come from snowboarders (60% of terrain park injuries) than skiers (31% of terrain park injuries) [11].

Taking the literature as a whole, head injury rates appear similar between skiers and snowboarders [3], though some data, including a 7-year prospective study of FIS World Cup athletes, suggests higher concussion rates in snowboarders [12].

Upper extremity injuries, in particular hand/wrist injuries, are more common in snowboarders [3, 5]. One prospective study of recreational athletes showed a 1 in 4 risk of distal radius fracture in snowboarders compared with 1 in 100 for skiers [5].

Lower extremity injuries, particularly knee injuries, are less common in snowboarders [3]. However, ankle injuries appear to be more common in snowboarders [3].

Solid organ and spine injuries are more common in snowboarders than skiers [13].

Mechanism of Injury

About 80–90% of snowboarding injuries are the results of falls [3]. This includes landing from jumps, which, if separated, account for about 25% of all snowboarding injuries [3]. Collisions are less common than in skiing

Table 71.1 Most common snowboarding injury location (body part) categorized by competition level [14]

Performance category	Most common	Second most common	Third most common
Recreational – General location	Upper extremity	Lower extremity	Head/neck/ face
Recreational – Specific injury	Wrist	Head/neck/ face	Shoulder
Elite	Knee	Shoulder/ clavicle	Head/face

Table 71.2 Most common specific snowboarding injuries categorized by competition level [13]

Age category	Most common injury	Second most common injury	Third most common injury
Recreational	Radius fracture	Scaphoid fracture	Other carpal injury
Active professional	Concussion	Radius fracture	Other hand fracture
Retired professional	Finger injury	Clavicle fracture	MCP-UCL injury of thumb

[3] but still associated with increased injury severity [13].

Recreational Snowboarder

Injury rates and distribution vary depending on level of performance (Table 71.1) [3]. A 2015 survey study is a good example of this, showing differences between recreational athletes, active professional athletes, and retired professionals (Table 71.2) [14].

Overall, compared with professional snowboarders, recreational snowboarders are more likely to sustain upper extremity and minor injuries than their professional counterparts [3, 14].

A 2015 survey study found upper extremity injuries to be by far the most common in recreational snowboarders (72% of all injuries), followed by spine and lower extremity injuries (9–11%), and head injuries (5%) [14]. Fractures (48% of injuries) occur more frequently than dislocations (13%) [5].

Individually, hand and wrist injuries tend to be most frequent, specifically distal radius followed by scaphoid fractures [14].

Advanced recreational snowboarders are more likely to sustain serious injuries such as traumatic spinal cord injuries than their beginner counterparts [15].

Active Professional Snowboarder

Injury surveillance data for elite snowboarders is more scientifically rigorous than for recreational athletes [3]. The overall injury rate has been reported at 1.3–4.0 injuries/1000 runs in studies with observation periods of 1 year, with rates increasing to 6.4 injuries/1000 runs in studies with observation period of 6 years [3].

When advanced athletes are separated from the entire snowboarding population, minor injuries such as wrist sprains, wrist fractures, contusions, and lacerations are less common in advanced snowboarders [3]. Professional athletes still suffer majority upper extremity injuries, but the overall proportion is decreased compared with recreational athletes [14].

While some studies have shown higher rates of head/face injuries than lower extremity injuries [16] in professional snowboarders, others have shown a higher rate of lower extremity injuries than head/face injuries [14].

Among professional athletes, the most frequent individual injury in a 2015 survey study was concussion, followed by radius fracture, and other hand injury [14]. When broken down by event, this data showed that snowboard racers were most likely to sustain a hand fracture, freestyle snowboarders most likely to sustain a radius fracture, and snowboard cross athletes more likely to sustain a concussion [14].

Elite snowboarders appear to have more severe injuries than their recreational counterparts. For example, one study showed that injuries resulting in time loss made up 72% of world cup snowboarding injuries [3]. A survey study of world cup snowboarders showed approximately a 37% rate of sustaining a time loss injury [17].

When various world championship level competitions are examined, the highest injury rates appear in the snowboard cross, snowboard slopestyle, and halfpipe events [8, 9, 16, 18, 19]. Injuries predominantly occurred in training.

Injuries in professional snowboarders tend to occur from contact with the ground [19], often attributable to a technical error at take-off [20].

Table 71.3 Risk factors for snowboarding injuries

Risk factor
Beginner level [21]
Participation in competitive event [21]
Suboptimal environmental conditions [21]
Jumping and falling [12]
Visitors to alpine region [4]
Age <16 years [4]
Not wearing a wrist guard [4, 21]
Terrain park [1, 12]
Less snowfall [13]
Not wearing a helmet [1, 22, 23]

Retired Professional Snowboarder

A survey study from 2015 showed that 26% of retired professional snowboarders queried had left their professional career due to injury [14]. The injury distribution in this group was 55% upper extremity injuries and 35% lower extremity injuries [14]. Most frequent specific injuries were finger injuries, followed by clavicle fracture, followed by MCP-UCL injury of the thumb [14].

Unique Injury Considerations

The fracture of the lateral process of the talus is mostly unique to snowboarding and has been colloquially termed the "snowboarder's fracture" [3]. This accounts for about 15% of all snowboarding ankle injuries and 2.3% of all snowboarding injuries [3].

Risk Factors

Risk factors for injuries in snowboarders include beginner level, participation in competitive events, suboptimal environmental conditions [21], jumping and falling [12], visitors to an alpine region, age <16 years, and not wearing a wrist guard [4]. Terrain parks also appear to increase risk and/or severity of snowboard injuries [1, 12]. Snowfall has a negative association with injury severity (Table 71.3) [13].

Wearing a helmet has been controversial as an injury prevention mechanism, with one review of the US National Trauma Data Bank from 2009 to 2010 showing equal incidence of concussion and severe head injury in helmeted and non-helmeted skiers and snowboarders [24]. However, the preponderance of data in snowboarders suggests that helmets do prevent head injuries [1, 22, 23]. Snowboarders in the National Trauma Data Bank cohort were less likely to wear helmets than skiers [24]. Still, there is evidence that personal history of head injury increased helmet use [25].

There is direct and indirect evidence that use of wrist guards does protect against wrist injuries, wrist sprain, and wrist fracture [4, 26].

What Is Unique About Snowboarding Injuries?

- Injury profiles vary widely depending on level of performance.
- The setting of the sport can make injury identification and extraction from field of play difficult.
- The setting of the sport raises the possibility of both minor and catastrophic injury.
- Decisions need to be made ahead of time regarding what injuries will be addressed on site and what need to be transferred for evaluation and management.

What Do the Physicians Need to Know While Covering a Game on the Sideline?

The injury severity seen during snowboarding events varies widely, as described above. A formal snowboarding event or venue should be equipped with an emergency action plan and personnel equipped for treatment or stabilization and evacuation in the case of catastrophic injury, including but not limited to cardiac arrest, severe closed head injury, chest and abdominal trauma, and displaced and/or open fracture. As the most common injuries that would not need emergency referral include minor concussions, sprains, and contusions, a covering medical team should have the personnel and equipment to treat these. Such equipment might include splinting material, ankle/wrist braces, ice, and crutches. A covering physician should have a plan for follow up, as many of these injuries require follow-up evaluation and clearance prior to returning to participation.

General Rule about Return to Play

As with any sport, when considering return to play for snowboarding injuries, the provider needs to take into account the injury type, level of competition, and athlete's understanding of injury risk. One should focus on injury prevention education, particularly based on evidence-based risk factors. It is important to evaluate equipment including helmet, boots, binding, and wrist guards for proper fit and lack of damage prior to using again after an injury.

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Soccer

72

Morteza Khodaee and Seth A. Mathern

Key Points

- Soccer is the most popular sport worldwide.
- Shin guards are the only mandatory personal protective equipment in soccer.
- Lower extremity is the most common anatomical location for injury in soccer.
- Sprains and strains are the most common injuries in soccer.
- Concussion is relatively common in soccer.

Introduction

Soccer (football) is the most popular sport worldwide with an estimated 265 million players in 2006 (Fig. 72.1) [1]. About 24,000 men and 26,000 women participated in National Collegiate Athletic Association (NCAA) soccer in 2013–2014 season. As one of the fastest growing sports in the United States, high school soccer participation increased fourfold among boys and 35-fold among girls from 1973 to 2014 [2]. Shin guards are the only mandatory personal protective equipment in soccer. A small percentage of players may wear mouthguards [3]. After football and wrestling, soccer had the next highest injury rate among US high school sports in 2005/2006 [4]. Sports injuries can be costly and can limit future sports participation [5, 6].

Injury Epidemiology

Injury rate in high school RIO (reporting information online) soccer has been reported as 2.06 per 1000 athletic exposure (AE) [7]. In a study by Roos et al., injury rates in NCAA soccer were 8 and 8.5 per 1000 AE in men and women, respectively [8]. Aoki et al. reported the injury rate as about 22 per 1000 player hours during official professional soccer league matches in Japan [9].

In general, the rate of injury is higher during competition than during the practice [7, 8, 10–12]. An NEISS (National Electronic Injury Surveillance System) study showed an estimated 2.5 million soccer-related injuries that presented to the US emergency departments from 2000 to 2012 [13]. Adolescent soccer players most commonly sustain lower extremity injuries, but more recent concerns have focused on their risk of sports-related concussions (Table 72.1) [14–18].

The most common injuries reported are sprain/strain, contusion, concussion, and fractures (Table 72.2) [7, 9, 10].

The most common mechanism of injury is soccer in player-to-player contact [7, 8, 10–12]. Playing on artificial turf in comparison with natural grass was reported to be associated with lower total injury rate among men's and women's NCAA teams [11, 12]. Midfielders seems to suffer most injuries, followed by forwards, defenders, and goalkeepers [7]. There is continued debate regarding the concussion risk associated with heading the ball (e.g., whether contact with the ball is an important concussion mechanism or whether player–player contact during the act of heading is the more common and more concerning mechanism of concussion), which has intensified concerns among some coaches and parents [19]. Increases in high school soccer participation and the potential long-term effects of injury emphasize the need to understand the epidemiology of high school soccer injuries to direct targeted intervention strategies [20, 21].

M. Khodaee (✉)
Department of Family Medicine and Orthopedics, University of Colorado School of Medicine, Denver, CO, USA
e-mail: Morteza.khodaee@cuanschutz.edu

S. A. Mathern
Department of Family Medicine, University of Colorado School of Medicine, Denver, CO, USA
e-mail: seth.mathern@cuanschtz.edu

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Fig. 72.1 Soccer is popular among children

Table 72.1 Most common body parts injured treated in the US emergency departments, seen at high school and collegiate settings[10]

Body parts	14- to 17-year-olds (2005–2013)		18- to 22-year-olds (2009–2013)	
	NEISS (%)	HS RIO (%)	NEISS (%)	NCAA (%)
Head/face/neck	25.5	21.4	24.4	11.2
Shoulder/arm/elbow	9.4	3.4	7.8	3.5
Hand/wrist	11.9	4.2	9.5	3.4
Trunk	7.3	3.7	6.6	5.3
Thigh/upper leg	1.1	15.3	1.2	27.0
Knee	12.3	16.5	14.5	15.5
Lower leg	5.9	7.4	5.9	8.9
Ankle	18.7	20.6	20.9	15.9
Foot	7.4	6.4	8.1	7.3

Table 72.2 Most common soccer injuries treated in the US emergency departments, seen at high school and collegiate settings [10]

Diagnosis	14- to 17-year-olds (2005–2013)		18- to 22-year-olds (2009–2013)	
	NEISS (%)	HS RIO (%)	NEISS (%)	NCAA (%)
Sprain/strain	33.7	48.3	16.3	21.9
Contusion	17.0	12.3	13.9	16.1
Fracture	18.6	7.5	15.7	2.8
Concussion	7.6	17.9	4.6	5.9
Laceration	4.8	1.1	8.2	1.4
Dislocation	2.6	1.3	4.3	0.5

What Is Unique About Soccer Injuries?

- In professional and national levels, there are only three substitutions during the regulation time, so decision about return to play, particularly after a concussion, might be challenging.
- Players can be removed from play while injured to be examined without making a substitution, but the team will remain one player down until the player is substituted or allowed back into the game.
- Lower extremity is the most common anatomical location for injury.
- Sprain and strain are the most common injuries.

What Do the Physicians Need to Know While Covering a Game on the Sideline?

- Physicians should be familiar with concussion, muscle strain, and ligament sprain management.
- Given the high incidence of ankle sprains, physicians should be familiar with ankle stabilization and taping.
- The clock for soccer does not stop. Thus, the entire time occupied by injury while player is on the field is estimated and added as "stoppage time" or additional time at the end of the half.

General Rule About Return to Play

- Return to play should be guided by patient symptom reporting and ability to tolerate activity prior to returning to play.
- Physicians should be familiar with various injuries, especially concussion and muscle strains (e.g., hamstring strains often have longer return to play times) [22].
- Outside of concussions, there is limited evidence regarding return to play for sprains and strains, and each league may have their own requirements regarding safe return to play.

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Track and Field

73

James Thing and Volker Scheer

Key Points

- Track and field encompasses a wide variety of throwing, jumping, and running sports with different injury profiles and sporting demands.
- Due to the repetitive nature of many of the events, bone stress injury and tendinopathy are frequently seen.
- Track and field events pose an inherent risk to the athlete and officials.
- Thigh injuries, in particular hamstring strains, are the most common injury type seen.
- Male athletes are more commonly injured than their female counterparts.

Introduction

Track and field (athletics) encompasses a variety of Olympic sports including throwing events, jumping events, sprints, and middle-/long-distance running. Track and field events date back to 884 BC and the first ancient Olympics [1]. Each individual sport carries with it an inherent risk of injury, depending upon the nature of the requirements of the event.

Certain events have a low incidence of acute, traumatic injury (e.g., a "spiking" laceration injury in the 10,000 m). Other sports have a much greater incidence of acute trauma (e.g., hamstring or calf strain in the 100 m). Events such as the pole vault have a low overall incidence of injury but a far greater risk of catastrophic trauma (e.g., cervical spine injury or traumatic brain injury) [2].

The risk of acute illness and injury remains high enough to necessitate having a trackside medical team at track and field events, which normally includes a sports medicine physician, paramedics, ambulance technicians/first aiders, and nurses.

Ideally these personnel should be situated to ensure that all areas of the track and field competition and training arena are covered. Busy athletics meetings can often involve simultaneous track and multiple field events requiring effective communication and vigilance on the part of the attending medical team. It is not uncommon for several medical incidents to occur at the same time, requiring careful rationing of medical resources and personnel.

Extraction of injured athletes from the field of play requires detailed planning prior to the event. It is essential for the medical team to be aware of the nature of the different sports as certain events (e.g., discuss, javelin, and hammer pose an inherent risk to both noncompeting officials and athletes). The environment should always be assessed for potential danger prior to the entry of medical personnel onto the field of play.

Injury Epidemiology

The overall incidence of injury in track and field has been reported at an elite level as 134.5 injuries per 1000 registered athletes during competition [3].

When considering all events at an elite competition, the majority of injuries involve the lower limb (~80%) with the hamstring/posterior thigh being the most common, representing one quarter of all injuries [1, 3]. The most common acute injury in track and field is a muscle strain, accounting for 20% of all injuries.

In general, male track and field athletes appear to be more susceptible to acute injury than female athletes. The majority

J. Thing
Sports and Exercise Medicine, Barts Health, London, UK

V. Scheer (✉)
Ultra Sports Science Foundation, Pierre-Bénite, France

Health Science Department, Universidad a Distancia de Madrid (UDIMA), Madrid, Spain
e-mail: v.scheer@ultrasportsscience.org

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of injuries in major championships occur during active competition, with a greater injury risk seen during finals events, when compared with qualifying rounds [3].

When considering the risk of injury in competition for each event category, the highest incidence occurs among individuals competing in combined events such as decathlon and heptathlon (288 injuries per 1000 athletes), followed by middle- and long-distance runners (176 and 188 injuries per 1000 athletes), jump events (139 injuries per 1000 athletes), throw events (100 injuries per 1000 athletes), and sprint events (84 injuries per 1000 athletes) [1].

Track

Sprint

The sprint events include 100 m, 100 m (women) and110m (men) hurdles, 200 m, 400 m, and 400 m hurdles. Team sprint events include the 4×100 m and 4×400 m relays. Sprinters tend to have greater muscle mass, explosive speed requirements, and are therefore more likely to suffer from acute lower limb muscle injuries such as hamstring, quadriceps, and calf sprains [4]. These injuries present with a sudden, “pulling up” from the sprint position with the athlete commonly grasping the affected muscle group and slowing to a stop with an antalgic gait.

Among men, the 100 m (Fig. 73.1) represents the sprint event with the greatest injury risk, with 147 injuries per 1000 athletes during competition. For women, the 100 m hurdles offer the greatest risk with 205 injuries per 1000 athletes [3].

Initial trackside management of an acute muscle injury includes icing and compression of the affected muscle group. The athlete is then assisted off the track with support/using a carry chair, depending on the likely injury severity. Ongoing management will depend on the extent and location of the injury within the musculotendinous unit, with MRI and/or ultrasound imaging used to grade and potentially prognosticate the pathology.

In younger athletes, with an immature skeleton and unfused bony apophyses, the tendon origin is more susceptible to injury than the relatively strong muscle-tendon unit. Avulsion of the bony apophysis is therefore far more common in the teenagers [5].

It is important for the trackside clinician to consider the possibility of an avulsion injury, particularly around the large muscles originating in the pelvis (e.g., rectus femoris origin, proximal hamstring origin, sartorius origin, iliopsoas insertion). If suspected, the athlete should be cautiously assisted from the track via a non-/minimal weight-bearing method and should be transported to hospital for plain radiography to investigate this injury initially [5]. A bony avulsion injury can often be managed conservatively but will generally require a longer recovery period, when compared with muscle-tendon unit injury.

Hamstring injuries are particularly common in adult sprinters and are seen more frequently in males than females [3]. The biceps femoris is the most commonly injured hamstring muscle in sprinters and is involved in three quarter of all hamstring injuries [6]. Significant early re-injury rates (30% in the first year) require a cautious and diligent rehabilitation approach.

Fig. 73.1 Sprinter Usain Bolt during the World Championships in Berlin 2009 where he ran the current 100 m world record in 9.58 s

The sprint hurdles pose a significant threat of injury to athletes. Sprint hurdlers often fall after colliding with the barrier, which frequently results in trauma that can range in severity from minor track abrasions or "burns" as a result of friction of the skin on an artificial track, soft tissue hematomas and lacerations, to upper limb fractures.

Abrasions occurring as a result of a fall on an artificial track surface are not uncommon. Track "burns" can be painful and often result in particulate debris becoming lodged in the wound. Removal may require irrigation with copious volumes of water (see Chap. 40) to extract solid debris.

Middle and Long Distances

The middle-/long-distance events include 800 m, 1500 m, 3000 m steeplechase, 5000 m, 10,000 m, marathon, and 20-km and 50-km race walk. Middle-/long-distance runners tend to be leaner, have greater endurance requirements, and are more likely to suffer from chronic injuries, as a result of a repetitive/overuse mechanism such as bone stress injury and Achilles tendinopathy. Acute exacerbations of these chronic injuries are most likely seen in a trackside environment [4].

For men, the 1500 m represents the highest risk middle-/long-distance event for injury with 308 injuries per 1000 athletes [3]. For women, the highest risk event is the marathon with 214 injuries per 1000 athletes [3].

Bone stress injuries occur as a result of repetitive bone loading when bone resorption exceeds bone formation. These injuries can range from a minor stress reaction, which responds well to load adjustment, to a cortical fracture, potentially requiring surgical intervention depending on anatomical localization (see Chap. 33). Bone stress injury incidence among track and field athletes is 21% in all disciplines [7].

The most common site for bone stress injury in track and field athletes is the tibia (51%) with the majority localized to the posteromedial tibia. Other common sites of bone stress include the metatarsals (21% of all bone stress injury seen), femur (7%), sacrum (5%), sesamoids (5%), fibula (5%), and navicular (3%) [7].

Risk factors for bone stress injury include previous history of stress fracture, an increase in frequency, duration, and intensity of training loads, inadequate recovery, and hard or cambered training surfaces. In addition to this, biomechanical abnormalities of the lower extremities and altered foot mechanics can contribute to the development of bone stress injury [8].

The female athlete triad is frequently seen in track and field athletes. It describes a combination of menstrual irregularities, disordered eating, and reduced bone mineral density (i.e., osteopenia and osteoporosis) and may predispose an athlete to bone stress injury [9, 12].

Spiking injuries can be seen in track events, particularly in fast-paced, close-packed events such as 800 m and 1500 m, and less frequently seen in events where athletes remain in their lanes such as 100 m, 200 m, and 400 m [10]. Lower limb lacerations, resulting from contact with running spikes, are frequently seen and are usually superficial. They may require closure with sutures or steri strips [11].

Field

Throws

The throwing events include the javelin, shot put, discus, and hammer. Throwing athletes tend to have large muscle mass and greater height in order to optimize throwing distance. For men and women, the event with the highest risk of injury is the javelin with 189 (men) and 179 (women) injuries per 1000 athletes [3].

Blisters and palmar calluses are minor ailments seen in throwing athletes, resulting from repeated skin friction with the throwing object such as discus and javelin [4].

Upper limb injury is common in throwing athletes with shoulder pathology frequently seen. Rotator cuff pathology and subacromial pain syndrome (impingement) resulting from the demands of recurrent upper limb loading in an overhead position are also frequently observed [4].

Elbow injury, in particular UCL sprain, is seen in javelin-throwing athletes as a result of recurrent hyperextension of the elbow with valgus stress [4]. This may result in chronic UCL laxity or an acute UCL rupture which presents with sudden onset discomfort over the medial elbow joint, usually on release of the javelin. This requires thorough assessment, early offloading with a brace preventing excessive valgus stress, and investigation to determine the extent of the injury and likely prognosis, and management strategy.

Throwing events may place spectators, officials, and other athletes at risk of serious injury. In particular, caution must be exercised with javelin and hammer throw, with occasional high-profile trauma incidents resulting from errant throws. Landing areas must be clearly marked, and officials must maintain concentration at all times. In addition to this, throwing cages should be used for hammer and discus throw to reduce the risk of nonathlete injury.

Jumps

The jumps events include the high jump, long jump, triple jump, and pole vault. Jumping athletes tend to be lean and require explosive power to optimize jump distance or height.

Achilles and patellar tendinopathy (jumper's knee) are frequently seen in jumping athletes as a result of the repetitive nature of their training and competition. These injuries usually present with gradual onset, progressive discomfort localized to the anterior knee or posterior ankle.

Acute Achilles or patellar tendon rupture should be suspected in jumping athletes with a sudden onset of posterior ankle pain and collapse or anterior knee pain and loss of the extensor mechanism function such as inability to actively straighten the knee (see Chap. 34). If suspected, the athlete should be cautiously assisted from the trackside environment to the medical room for a formal assessment, ideally with point of care ultrasound.

For men and women, the highest risk jump event is pole vault with 276 (men) and 143 (women) injuries per 1000 athletes [10]. Pole vault also represents the track and field event with the single greatest risk of mortality, as a result of landing directly onto the head or neck from a height [4]. Catastrophic injury in pole vaulters is infrequent, with 11 major head injuries, four spinal fractures, one brain stem injury, and one pneumothorax in the USA over a 9-year period with two athlete deaths resulted from direct head trauma [2].

It is imperative for the medical personnel stationed by the vault to be well versed in acute traumatic head and neck injury management, including the use of manual in-line stabilization (MILS) techniques. Jump mat extraction can be complex as the surface is undulating and liable to destabilize once the medical team approaches the athlete. Moulage (practical hands-on) training prior to the event ensures that a plan of action has been established and that the paramedical team is aware of their roles and responsibilities in such a scenario.

What Is Unique About Track and Field Injuries?

- Track and field encompasses a wide variety of different disciplines with different injuries.
- Acute injuries can mostly be observed during competitions.
- Chronic or overuse injuries are often seen in training.
- Injuries are mostly sustained through noncontact as a result of musculoskeletal injuries or ligament sprains.
- Equipment failure is rare but can lead to significant injury.

What Do the Physicians Need to Know While Covering Track and Field Events?

- Knowledge of mechanisms and athletic discipline is important to diagnose and treat injury.
- Acute muscle injuries such as hamstring strains are common in sprinting events.
- Exercise associated collapse (EAC) is one of the most frequent causes of collapse seen mostly at the finish line of longer distance events (e.g., 10,000 m and the marathon). Runners generally recover quickly and fully [13].

General Rule About Return to Play

- Any athlete with suspected severe injury should be held from further competition until further assessment.
- Athletes with minor injuries may be allowed to continue competing, but the injury may have impact on performance.

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Triathlon

74

Brent C. Pottenger and Sameer Dixit

Key Points

- Triathlon is a popular sport for amateurs and professionals worldwide.
- Triathlons involve swimming, cycling, and running predetermined distances.
- Ironman is a type of triathlon that involves the longest event distances.
- Triathlon injuries are specific to each of the activities involved and are influenced by the athlete's performance of all three activities in succession.
- Overuse and running-related injuries are the most common types of injury.
- Physicians covering triathlons should prepare for cardiac and heat-related medical emergencies, although they are rare.

Introduction

Many amateur and professional athletes throughout the world participate in triathlons for recreation, exercise, and competition. The popularity of triathlons may be attributable to the increased visibility of the sport in recent years. The first Ironman competition occurred in Hawaii in 1978, and triathlon became an Olympic event in 2000. Triathlons are endurance events that involve swimming, cycling, and running (in that order) for specified distances (Table 74.1). The four standard types of triathlons are sprint, Olympic, long, and Ironman [1]. Athletes must train, repetitively, to build stamina for triathlons. Most triathlon-related injuries are sustained during training (75%–83%) as opposed to competition (2%–28%) [1] (Fig. 74.1).

Table 74.1 Distances of triathlon activities

	Distance (km)		
Triathlon type	Swimming	Cycling	Running
Sprint	0.75	20	5
Olympic	1.5	40	10
Long	3	80	20
Half ironman	1.9	90	21
Full ironman	3.8	180	42
Ultra	10	421	84

Injury Epidemiology

Knowledge of injury epidemiology helps physicians direct the medical coverage of triathlons and care for patients who are training for and participating in triathlons. Although cardiac and heat-related medical emergencies are rare during triathlons, clinicians must be prepared for them given the high rates of morbidity and mortality associated with delayed recognition and treatment [2, 3].

Running injuries are the most common, followed by cycling and then swimming injuries [1, 2]. Triathletes participating in shorter events, such as the sprint, sustain fewer injuries than those completing longer events [2]. Moreover, triathletes younger than 19 years are more likely to sustain injury than are adults [2].

Overall, injuries to the lower limbs and back are the most common (Table 74.2). Clements et al. found that 72% of knee injuries at triathlons occurred during the running portion [4]. Bertola et al. found that running injuries were most common in the calf for men and in the foot or ankle in women

B. C. Pottenger
Department of Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: bpotten1@jhmi.edu

S. Dixit (✉)
Departments of Orthopaedics, Medicine, and Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Lutherville, MD, USA
e-mail: sdixit3@jhmi.edu

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Fig. 74.1 Swimming part of a triathlon

Table 74.2 Most common sites of injury for triathletes [5, 6, 8, 10, 15–18]

Site	Percent of injuries
Lower limb	36–85
Knee	14–63
Calf	39–46
Ankle/foot	9–35
Back	46–72
Shoulder	19–42

Table 74.3 Most common types of injury for triathletes [7, 15, 18–20]

Type	Percent of injuries
Muscle/tendon	30–55
Contusions/abrasions	51
Ligament/joint	6–29
Miscellaneous/other	23–27
Tendinitis	13–25
Fractures	12

[5]. Common locations of acute triathlon-related injuries are the knee, shoulder/clavicle, and sternum/ribs. These are often sustained during cycling accidents [6].

Triathletes often suffer contusions and abrasions, as well as muscle and tendon injuries. Table 74.3 lists the most common types of injury. Heat stroke and deep lacerations, although less common (both were less than 1% of injuries in one study), have high acuity [2].

Table 74.4 Most common training/competing effects of injuries for triathletes [8, 10, 11]

Effect	Percent of injured triathletes
Interrupt training	78
Stop cycling	26–75
Stop running	42–67
Miss a competition	33
Stop training/competing	20
Stop swimming	17

Injuries can interrupt a triathlete's training and require activity modifications (Table 74.4). When injured triathletes modify their activities, they most often stop running or cycling [1]. Swim training is the least likely to be affected by injury. For the 20% of triathletes who must stop training completely, a running injury is most often the cause (78%), followed by cycling (37%) and swimming (21%) [7, 8].

Overuse injuries are common among triathletes [6]. Table 74.5 provides the rates of overuse and traumatic injuries, including the most common types of traumatic injuries. In an analysis of overuse injuries, Burns et al. found that high preseason running mileage significantly increased the risk of

Table 74.5 Most common mechanisms of injury for triathletes [7, 10, 16, 19]

Mechanism	Percent of injuries
Overuse	41–91
Trauma	15–56
Twist/turn	12
Contact/collision	10
Overstretching	9

injury during triathlon season and that the other two best predictors of injury risk were years of triathlon experience and history of injury [9]. A study of 174 Ironman participants in Norway by Andersen et al. found that overuse injuries are much more common than acute injuries are and that the knee, lower leg, and lower back are the most common sites of overuse injuries [6].

Triathletes must take time off to heal from injuries. Injured triathletes lose approximately 1–2 months of total training time, on average, with a wide variance based on injury type and severity [1]. However, it is unknown how often triathletes continue training or competing while injured. Given the multifaceted nature of the sport, injured triathletes can modify their activities (e.g., by stopping running and spending more time swimming) and thus continue training while injured. The risk of sustaining a running injury is higher among triathletes than among those who only run because triathletes run in tandem with cycling and swimming [1]. The amount of time triathletes take off to recover from injury varies by injury type. One study found that, for running injuries, the mean ± standard deviation time off is 71 ± 174 days, whereas for cycling injuries, it is 21 ± 65 days, and for swimming injuries, it is 13 ± 58 days [10]. This indicates that running injuries require the most recovery time. Also, the amount of time each injured triathlete stops training/competing varies substantially. Triathlon-related injuries affect athlete's lives in other ways, most commonly hindering their daily activities (64%), requiring absence from work (15%), and resulting in permanent impairment or loss of function (4%) [11].

Clinicians treating recreational triathletes can use these data when discussing the risks and benefits of triathlon participation. For some patients, the risk of missing work or sustaining a permanent impairment may outweigh the benefits of participation.

Injury Prevention

To prevent injuries, clinicians can guide triathletes during training. Because overuse and running-related injuries are most common, counseling triathletes to avoid excessive running or increasing mileage too quickly can help prevent these injuries. For example, triathletes could devote more time to cycling and swimming because these activities are low impact and are associated with fewer injuries compared with running.

Nutrition is an important consideration. Given the energy expenditure during training, triathletes may be at risk for a caloric deficit. Hoch et al. found that female triathletes were at risk for components of the "female athlete triad" [12]. Clinicians providing care for triathletes should consider guidance on the importance of increased caloric intake.

What Is Unique About Triathlon Injuries?

- People of all experience levels and athletic abilities participate in triathlons for various reasons, ranging from recreation to professional competition.
- Injuries consist of those that are commonly associated with individual components of the event (running, swimming, and cycling) and those that are associated with performing all three components together.
- Overuse and running-related injuries are the most common types of injury.
- Injury risk increases as the length of the triathlon event increases, and one of the best predictors of injury risk is total running training time.
- Serious acute injuries to anticipate when covering triathlons include lacerations, fractures, heat-related illnesses, and sudden cardiac events.
- Risks and benefits of triathlon participation should be discussed with patients because injuries can result in absence from work and permanent impairment of loss of function.

What Do Physicians Need to Know to Cover Triathlons?

Abrasions and Musculoskeletal Injuries

Knowledge of injury epidemiology helps physicians prepare for covering triathlons. Many triathletes sustain contusions and abrasions, including deep lacerations; therefore, supplies to bandage and care for these injuries are needed. Additionally, participants sustain muscle injuries (including spasms), sprains, and fractures; therefore, clinicians must be prepared to diagnose and provide supportive care for these injuries. Although not well studied, a basic rule for coverage of endurance events is that if an athlete is limping or is unable to maintain proper form, serious consideration should be given to withdrawing the athlete from participation.

Cardiac Events

Planning for cardiac events is an important part of preparing to cover a triathlon. Given the high variability in fitness levels and training regimens between novice and expert partici-

pants, there is the potential for those with unknown underlying cardiac disease to participate in a physiologically challenging event, which could be fatal.

In one study, Harris et al. noted 14 deaths at a rate of 1.5/100,000 (95% confidence interval, 0.9–2.5) during triathlon participation [13]. A total of 13 deaths occurred during swimming, and one occurred from cervical spine trauma during cycling. Of the nine athletes who died during the swimming event and underwent autopsy, seven had cardiovascular abnormalities. The authors speculated that because triathlons begin with chaotic and highly dense mass starts, opportunities for bodily contact, and exposure to cold turbulent water exist, which may exacerbate cardiovascular conditions [13]. This challenging environment must be considered by those providing medical coverage. Determining the optimal way to identify, extract from the water, and triage distressed swimmers is vital [14]. It is recommended to have automatic external defibrillators (AEDs) available, at least at the finish line.

Heat Stroke

Although less common in triathlons than in distance running, heat stroke is a medical emergency that requires immediate attention and appropriate triage. Prevention and treatment of heat stroke are based on the same principles that are described for distance running. Ensuring that adequately supplied hydration stands are available for participants and reminding athletes to hydrate and eat proper nutrition can help prevent heat stroke. Diagnosing heat stroke requires obtaining a rectal temperature (>40 °C), and, when treating heat stroke, access to rapid cooling mechanisms in the medical tent is important.

Preparation for Triathlon Coverage

Medical Equipment

The following medical equipment should be available during triathlons: gauze, medical tape, bandages, a suturing kit, antiseptics (e.g., alcohol wipes, antibacterial ointment), a stethoscope, crutches, braces, ace wraps, towels, rehydration supplies, anti-inflammatory medications, acetaminophen and pain relief medications, muscle pain/spasm topical medications (e.g., methyl salicylate topical), lidocaine patches, a rectal thermometer, cooling equipment (often ice baths), automatic external defibrillators, and point-of-care blood chemistry analyzers.

Emergency Action Plan (EAP)

The EAP should be implemented by an experienced medical director who has complete oversight of the planning and operations of the day of the competition. This person's responsibilities include decisions regarding appropriate equipment, placement of medical tents, management of the medical team, and guidance for rapid transport of emergent cases. The medical team may consist of a group of medical personnel with diverse professional backgrounds, including emergency medical technicians, certified athletic trainers, nurses, paramedics, podiatrists, physical therapists, massage therapists, and physicians. The EAP should include guidance on immediate triage of athletes and plans for transport on race day. Before race day, all medical personnel should be informed about the appropriate protocols and chain of command. Although coverage for emergencies is similar to that described elsewhere in this book, consideration should be given to the challenges of covering swimming events. This includes providing medical personnel with rafts and boats, as appropriate for the event [10].

Return to Participation

When a triathlete sustains an injury, the most important rules guiding return to participation are allowing sufficient rest for healing and using pain as a guide. Most injuries are caused by repetitive training and overuse, which require sufficient rest to heal fully. Many triathletes are driven to perform well in their events and have a strong desire to return to participation; therefore, physicians must guide triathletes to avoid resuming participation too soon. Triathletes could consider increasing their activity by first resuming the two lower-impact activities, swimming and cycling.

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Ultramarathon and Ultra-endurance Sports

75

Volker Scheer and Martin D. Hoffman

Key Points

- Acute traumatic injuries are rare in ultramarathons.
- Traumatic injuries are relatively common in high-speed ultra-endurance sports such as ultra-cycling.
- Clinicians taking care of athletes at ultra-endurance events should be familiar with medical issues such as exercise-associated hyponatremia and rhabdomyolysis.
- Foot blisters are the most common problem among ultra-distance runners.

Table 75.1 Ultra-endurance sports

Ultramarathon foot races
Ultra-distance cycling (road or mountain)
Ultra-swimming
Offshore sailing
Ultra-Nordic skiing
Ultra-paddling
Ultra-sledding
Triathlons
Ultra-adventure races
Other multisport competitions
Fastest known time efforts

Introduction

Ultra-endurance events are defined as sporting events lasting longer than 6 h. Due to the duration, these activities place considerable stress on the body in terms of physiological and psychological demands during training and competition. Adaptation to high training loads and development of nutritional strategies are important to reduce the risk of acute and overuse injuries. A summary of some ultra-endurance events can be seen in Table 75.1. This chapter gives an overview of the most common problems of ultra-endurance events and most specifically of ultramarathon running events. Issues in ultramarathons share some of the same principles as for other ultra-endurance events (e.g., triathlon), which are discussed in more detail elsewhere in this book.

An ultramarathon is any race over the traditional marathon distance of 42.195 km. Participation in ultramarathon races has increased in recent times with an approximate five-fold increase in ultramarathon race finishes over the last 10 years. Ultramarathons are carried out all over the world, many in remote and challenging environmental conditions that place the human body under enormous physiological demands and can compromise provision of medical support (Fig. 75.1) [1–3].

Types of Races

There is significant variability among ultra-endurance races. That is, most ultra-endurance events are unique in terms of distance, environmental challenges, level of aid and medical support provided, and the activity – single activity (e.g., running, sailing, swimming) or a combination of multiple sports (e.g., Ironman triathlon). Among all ultra-endurance sports, foot races and Ironman triathlons may be the most consistent across events.

Ultramarathon races can be divided into continuous and multiple-day staged (discontinuous) events. Typical race dis-

V. Scheer (✉)
Ultra Sports Science Foundation, Pierre-Bénite, France

Health Science Department, Universidad a Distancia de Madrid (UDIMA), Madrid, Spain
e-mail: v.scheer@ultrasportsscience.org

M. D. Hoffman
VA Northern California Health Care System, Mather, CA, USA

Department of Physical Medicine and Rehabilitation, University of California Davis, Sacramento, CA, USA
e-mail: mdhoffman@ucdavis.edu

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Fig. 75.1 Runner during the 230-km Al Andalus Ultimate Trail Run in a remote part of Spain

tances in continuous events include 50 km, 80 km (50 miles), 100 km, and 161 km (100 miles), and multiple-day staged events typically range from 3 to 7 days with distance of 250 km or more. These races can range from being fully supported to nonsupported by the race organizers, with the latter meaning that runners are required to cater and carry their own nutrition and support materials, adding to the weight and stress on the human body. Many races take place in the desert, jungle, mountains, or high-altitude locations, placing additional challenges on the body resulting in various types of injuries and illnesses [2, 3].

Injury Epidemiology

Most medical issues are minor in nature, but they can often have a serious effect on race performance. Serious injuries are rare but need to be recognized early and treated appropriately.

Musculoskeletal (MSK) Problems

Most MSK injuries affect the lower limbs and are typically overuse in nature. They can present acutely during the race and include:

- Acute MSK injuries are less common in ultramarathon running but may be related to running through rough terrain or mountainous areas and may include twisting injuries of the ankle or knee or accidental falls with grazes of the skin or even fractures.
- Often acute exacerbations of chronic overuse injuries can be seen during competition. These include anterior knee pain (patella femoral pain syndrome in up to 31% of runners), lateral knee pain due to iliotibial band syndrome (up to 12%) and lower limb stress reactions (medial tibial stress syndrome in up to 10%), stress fractures, or chronic exertional compartment syndrome (up to 11%). Acute pain in the heel due to Achilles tendinopathy (up to 19%) can also be frequently observed during races [2, 4–6].
- Ultramarathon ankle is a specific injury to ultramarathon participants that may happen in approximately 1.4% of runners. It is due to repetitive plantar flexion and dorsiflexion when running that causes a tenosynovitis of the tendons passing under or adjacent to the extensor retinaculum of the ankle on the anterior aspect. It presents with anterior ankle pain that is vague initially but can become sharper and more localized with continued running. Swelling over the anterior ankle is sometimes observed, and on clinical examination, there may be crepitus and pain on resisted dorsiflexion [2, 4, 5].

Treatment for all acute MSK injuries follows the same basic principle of protection, rest, ice, compression, and elevation (PRICE). Analgesia, taping, physiotherapy, and early rehabilitation may be further strategies to employ that prove beneficial in long-term management. During race, management is generally limited to massage, stretching, and taping.

Trauma

- Major trauma at ultramarathon events is extremely rare, but falls are common and can result in lacerations and fractures. Car accidents involving runners are also possible when race courses cross or include roadways. In remote environments, treatment may be challenging but should be orientated to current trauma life support guidelines.

Skin Problems

The skin is the largest organ in the human body and susceptible to injuries during ultramarathon running, mainly through friction (also see Chap. 40).

- *Foot blisters.* Most common problem in ultramarathon runners (generally between 26% and76% but even up to 100%). Incidence increases with race distance. They are due to shear forces within the epidermis, resulting in separation of skin layers and collection of fluid (Fig. 75.2). The main locations are the toes, heel, and ball of the foot. Causative factors may include poorly fitting shoes, unaccustomed levels of stress, various environmental factors, callous build up, hydration status, and individual factors. Preventative measures include taping, antiperspirants, lubricants, measures to keep the skin dry and shoes free of particulate accumulation, low-friction socks, double layers of socks, toe socks, and good foot care/maintenance including removal of callouses. Painful blisters should be drained under sterile conditions, lancing them in the periphery with a needle assuring adequate hole size and number for continued drainage, taping, or injecting them with iodine. Local skin infection and cellulitis can be a complication in a small number of runners if not treated appropriately [2, 3, 7–12].

Fig. 75.2 Foot blister after a 161-km ultramarathon

- *Chafing.* Superficial skin inflammation due to increased friction on the skin with fabrics or back packs, mostly affecting the nipple, groin, and back area (up to 41.9%). Use of lubricants is a useful preventative measure and may provide some relief during races [2].
- *Subungual hematomas.* Fluid/blood collection under the nail of the feet due to repetitive impact with the running shoe (up to 10%). These can be very painful and the pressure under the nail can be released by piercing it with a needle [2, 9].

Gastrointestinal (GI) Distress

- GI problems are quite common, affecting up to 85% of runners [13, 14]. Symptoms include nausea, vomiting, abdominal cramps, reflux symptoms, and diarrhea. They have been linked to performance decrements, race withdrawal, and decreased nutritional intake. The origin is usually benign, related to decreased blood flow to the intestine during running, nutritional intake, food intolerances, or infection. However, they can also be early signs of more serious pathologies such as exercise-associated hyponatremia, heat illness, cardiac pathologies, and altitude illness which need to be recognized. Typical during race treatment involves supportive care, recognizing that these conditions tend to improve with reduced exercise intensity. Antiemetic medications (e.g., ondansetron or metoclopramide) also have been used successfully to help with nausea and vomiting [3, 13, 14].

Exercise-Associated Hyponatremia (EAH)

- The incidence of EAH can be as high as 31%–51% in ultra-endurance events [15, 16]. Symptomatic EAH is much less common but is potentially life-threatening if not promptly recognized and appropriately managed. The main causative factor is excessive fluid ingestion in the form of either water or sports drinks. Secretion of arginine vasopressin (AVP) due to various non-osmotic stimuli (nausea, vomiting, exercise, thermal stress, and rhabdomyolysis) leads to retention of the excess fluid and development of a dilutional hyponatremia. Early symptoms are nonspecific, such as headache, nausea, and vomiting, progressing to confusion, seizures, cerebral and pulmonary edema, and death if not properly treated. Prompt recognition and confirmatory on-site determination of plasma sodium concentration is ideal, but empiric treatment should not be delayed if point-of-care blood analysis is not available. Treatment is with intravenous or

oral (if possible) hypertonic saline. Further treatment in a medical facility is advisable. Preventative measures include ad libitum fluid intake (drinking to thirst) and educating runners and aid station workers about the potentially fatal consequences of EAH, which has been demonstrated to significantly reduce the incidence of EAH [3, 14–18].

Severe Dehydration

- Mild dehydration is a fairly common phenomenon in ultramarathons, and it has been well demonstrated that endurance athletes can lose 4% or greater body weight during competitions without significant clinical symptomatology or adverse consequences [3]. Thus, the level of dehydration suggested by weight loss of a few percent of body weight is generally not hazardous and rarely requires intravenous (IV) rehydration when oral fluids can be tolerated. Because IV hydration with isotonic or hypotonic fluids can have adverse consequences in an athlete with EAH, and the clinical assessment of dehydration in athletes is challenging, a cautious approach to the field management of presumed dehydration is recommended. IV fluid replacement is best reserved for the severely dehydrated athlete (persistent tachycardia, poor skin turgor, and lightheadedness with standing) who is not recovering with oral fluid replacement or is having ongoing fluid losses with vomiting or diarrhea. When point-of-care analysis of plasma sodium is unavailable to confirm that the athlete is not hyponatremic, caution with IV hydration is warranted, and the athlete should be closely observed, with hypertonic saline readily available to use if neurologic deterioration suggestive of EAH occurs [3, 15].

Exercise-Associated Collapse (EAC)

- This is one of the most frequent causes of collapse mostly at the finish line or at checkpoints when running ceases abruptly. It may affect approximately 1%–4% of ultramarathon finishers. Symptoms are lightheadedness, faintness, dizziness, and collapse due a transient postural hypotension caused by the combination of peripheral vasodilation and blood pooling in the lower legs due to loss of muscle pump activity. Neurological, biochemical, or thermal abnormalities are absent, and treatment involves placing the runner in the supine position with legs elevated to increase central perfusion while assuring the athlete is breathing and has a pulse. Oral rehydration may be helpful once the athlete is alert, but intravenous fluid replacement is rarely necessary unless the runner cannot tolerate oral fluids or is not improving with these simple measures. Runners generally recover quickly and fully, so an athlete who does not quickly recover should be reassessed for other causes of collapse [2, 3, 19, 20].

Muscle Cramping

- Muscle cramping is common during ultramarathon participation (up to 40%) and can have serious effects on performance. Despite this high prevalence, the etiology, risk factors, and prevention strategies for the condition are not well understood. While the traditional hypotheses include heat strain, and fluid or electrolyte imbalance, recent work has demonstrated that muscle cramping in an ultramarathon is unrelated to an altered fluid and sodium balance. Currently, the most compelling theory on the underlying cause of exercise-associated muscle cramping (EAMC) relates to abnormal neuromuscular control as a result of muscle fatigue. It is also recognized that muscles are more vulnerable to cramping when in a shortened position. As such, appropriate management may include stretching, temporarily altering gait to avoid placing the involved muscle in a shorted position, and temporary reduction in exercise intensity. There is also some evolving research suggesting that sodium or select spices may stimulate receptors in the oropharyngeal region, causing a neurally mediated reflex to inhibit the firing of alpha motor neurons of cramping muscles [14, 19].

Acute Kidney Injury (AKI) and Rhabdomyolysis

- Few athletes have clinically significant kidney injuries that require medical attention but up to 50% temporarily fulfil the diagnostic criteria for AKI. It is not unusual that muscle damage, especially associated with downhill running, can increase plasma creatine kinase (CK) concentrations to well over 20,000 IU/L [3]. This extent of rhabdomyolysis is a risk factor for kidney injury, especially in association with reduced perfusion, dehydration, heat stress, underlying renal problems, and ingestion of NSIADs. Runners usually recover with oral rehydration based upon thirst. And at present, the limited research seems to indicate that there is no cumulative damage from these insults to the kidney. It is those athletes with poor urinary output and/or hematuria beyond 24 h post-event who would be wise to seek medical attention [3, 21–24].

Environmental Issues

Many ultramarathons take place in remote and challenging environments, and runners can be exposed to heat, cold, or high altitude which poses additional risks.

- *Heat*: Heat acclimatization is important but may not be fully achieved if not exposed to hot conditions for about 10–14 days. While very rare in prolonged endurance events in which the exercise intensity is relatively low, the potentially life-threatening condition of exertional heat stroke (EHS) should be considered in thermally stressful environments. EHS is the result of heat accumulation when the heat generation through exercise sufficiently exceeds the removal of the heat. It is characterized by a core temperature reading over 40 °C and altered mental state. Early signs are nonspecific with headaches, nausea, vomiting, and confusion that can progress to collapse, seizures, multiorgan failure, coma, and death (3). It is a medical emergency, and immediate cooling is recommended either with cold water immersion or cooling via ice packs to glabrous skin surfaces. Transfer to a medical facility for further care and treatment is recommended [2, 3, 25].
- *Cold*: Prolonged exposure to cool or cold environments, especially when it is wet and/or windy, poses the risk of hypothermia. Early symptoms can be observed when core body temperature drops <35 °C with shivering, fatigue, weakness, and cardiovascular compromise. When core body temperature drops <32 °C, severe hypothermia is present and can lead to cardiac arrhythmias, coma, and death. Stopping heat loss and rewarming the body is the priority of treatment. Provision of oral glucose (e.g., warm sweet drinks) as fuel for intrinsic heat generation is also helpful [3, 26, 27].
- *Altitude illness*: Symptoms of acute mountain sickness can be observed at elevations above 2500 m with headaches, nausea, vomiting, dizziness, and fatigue. Halting further ascent or descent is a key initial intervention. More severe cases may require supplemental oxygen and specific therapy (e.g., dexamethasone and nifedipine) [3].

Vision Problems

- Visual dysfunction during ultramarathons is not very common (<4%) but can impact race performance and safety and may be severe enough that the runner is unable to continue running [7]. When eye pain is present, considerations include foreign bodies, keratoconjunctivitis, or even ulcers. Painless clouding of vision is thought to be due to corneal edema and typically resolved without intervention within a few hours [3, 7, 28, 29].

General Rules About Continuation in the Race

A discussion about whether or not to continue the race is reasonable and is also an important aspect of the medical care in this environment. The medical team should decide when an athlete is unable to continue the race due to medical reasons and needs to be withdrawn from competition. This is to prevent harm coming to the athlete and others. Often it can be helpful to involve friends or family of the athlete in the discussion, but ultimately it is the responsibility of the medical team.

What Is Unique About Ultramarathon and Ultra-Endurance Sports Injuries?

Ultramarathon and ultra-endurance races are extreme forms of exercise that test the limits of human endurance and particular stress the body, resulting in MSK injuries and other medical problems (e.g., EAH, EAC, dehydration, cramping). Adding to the demands on the clinician, many of these races take part in remote and challenging environments with limited resources.

What Do the Physicians Need to Know While Covering These Events

Pre-race medical planning is important to establish the extent of medical coverage needed for each event taking into consideration participant numbers, location of event (remoteness to medical facilities), and environmental issues. Included in the planning should be medical protocols for lost or seriously injured athletes. Medical supplies, equipment, and staffing level and skill mix should be appropriate for the event. Participants should be informed about the level of care they can expect to find at the event. Medical supplies should be stored in a way that is easy to access and identify, but able to withstand environmental conditions and transport. Medical supplies to treat serious medical conditions need to be available (e.g., intravenous setups and fluids, hypertonic saline for EAH, glucose for diabetic athletes, albuterol inhaler for asthmatic athletes, and epinephrine, steroids, or antihistamines for allergic reactions). Supplies to treat minor and common medical problems such as blister kits, antiemetics, analgesics especially for MSK injuries are also important. Athletes should be briefed about common medical conditions and advised to bring their own regular and acute medication to treat common medical problems. Pre-race medical briefings and ongoing athlete education have been associated with reduction in the incidence of EAH [3].

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Volleyball

76

Jill N. Tirabassi

Key Points

- Volleyball is a popular sport for all ages.
- Most injuries are related to jumping and landing.
- Front court positions have a higher injury rate than back court in indoor volleyball.
- More injuries occur in senior level players compared to junior players.
- Lower extremity injuries are the most common injuries in volleyball, with ankle sprain being the most common specific injury.
- ACL rupture, while rare, happens more often in females.

Introduction

Volleyball is one of the most popular sports in the world with players of all ages [1]. Volleyball is considered a noncontact sport with opposing teams separated by the net [1–3]. Indoor (gym) and beach volleyball are the two major types of volleyball. There are typically six players on the hard surface, indoor court in gym volleyball. There are usually two players in beach volleyball, which is played on sand [4, 5]. The goal of the sport is to hit an unreturnable ball to the opponent's side of the court, which usually uses hitting and setting schemes that lead up to strong "spike" [4]. Only the three front players are allowed to attack or block at the net [3]. A strong hit can result in a ball velocity of up to 130 km per hour [4]. Volleyball requires a high level of muscular fitness for optimal performance [6]. The main movements are quick and powerful with sudden direction changes [4].

J. N. Tirabassi (✉)
Department of Family Medicine and Community Health, University of Massachusetts Medical Center, Worcester, MA, USA
e-mail: Jill.Tirabassi@umassmemorial.org

Injury Epidemiology

The incidence rate of injuries in volleyball is lower than other team sports like basketball, hockey, soccer, and handball [1, 2]. However, the injury rate is considered high for a noncontact sport [6, 7]. Games have a higher injury rate than practice [8]. Acute injuries are usually sprains of the fingers and ankles, but they can include strains, fractures, concussions, and facial injuries. There are no differences in injury risk between males and females [9]. There does seem to be a trend toward higher injury rate in senior players compared to junior players [2].

Regardless of age, skill, and gender, there are consistent trends for the most common acute volleyball injuries and their mechanism. The conflict zone, defined as the 50-cm-wide area underneath the net, is a common area for injury [3]. Injuries are the highest at the net, when landing from blocking or an attack (Fig. 76.1) [1, 3, 4, 10]. Landing on a player and contact with the floor account for most of game injuries [8]. Blocking is the most common sport-specific activity that results in injury [11]. Player position does matter regarding injuries. The front row positions (attackers) had the highest incidence of ankle injury [1, 6–10]. Liberos, defensive specialists, had the lowest rate of injury [9].

Most research on volleyball injuries has been performed on indoor volleyball athletes compared to beach volleyball athletes [12]. The rate of injury was similar, and there were no sex differences [5, 12]. However, the injury pattern between indoor volleyball and beach volleyball is different, largely in that there are more defensive injuries in beach volleyball [5]. For example, finger injuries also commonly happen in beach volleyball but more commonly occur from overhead digs, a defensive maneuver [12]. In indoor volleyball, they usually result from blocking [5]. Beach volleyball has about half the injury rate of ankle injuries compared to indoor volleyball. A large part of the reduction is because there are not two person blocks in beach volleyball, so landing on the foot of another teammate does not happen [12]. Acute injuries seen in beach volleyball also include knee and

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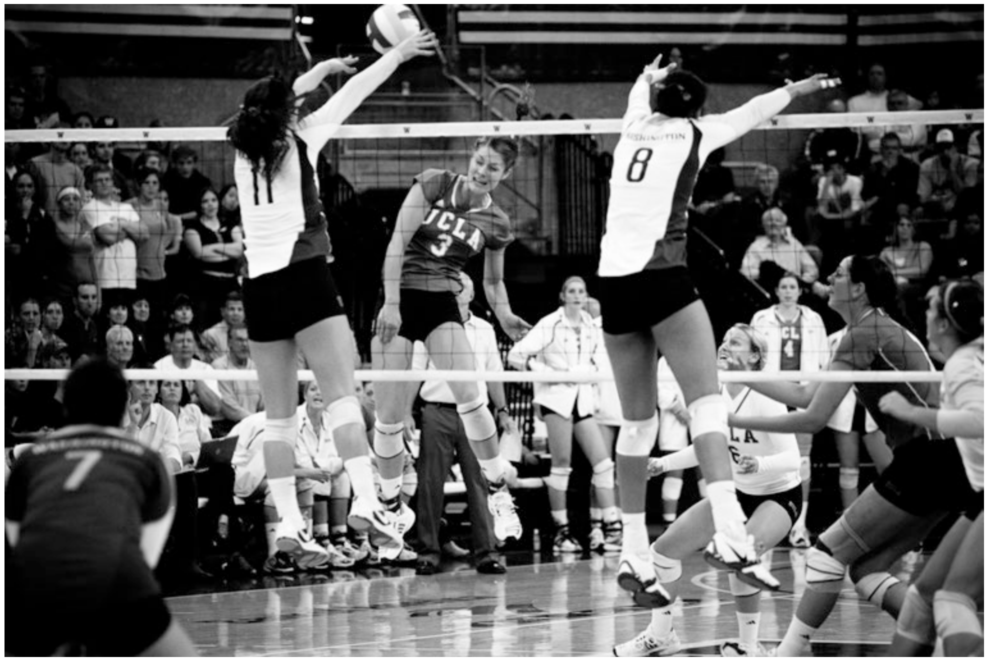

Fig. 76.1 Blocking a spike during a college game. (Courtesy of Ashwin Rao, MD)

low back injuries [12]. Overall, the injury rate in beach volleyball is considered low, and most injuries are considered mild with little time lost from sport [12].

In high school sports, serious injuries resulting in medical disqualification included ankle sprains for both sexes. However, in females, knee sprains and concussions also caused disqualification. Blocking was the most common sport-specific activity causing injury in high school-aged athletes [13]. In world-class/elite volleyball players, no differences were seen in risk of injury between males and females; however, senior players had a higher risk of injury than junior players [2, 9, 10]. In collegiate sports, ankle and knee problems also accounted for the most severe injuries [8]. While elite players may suffer a high rate of injury, they usually are not kept out for more than a week [6]. The majority of injuries are considered minor, meaning the absence from sport was less than 1 week [6, 9, 10]. In one study looking at world-class volleyball players, total injury incidence during game play was 10.7 per 1000 player-hours [9]. Severe injuries (resulting in > 4 weeks of absence from play) were rare at 0.3 per 1000 player-hours, much lower than other major team sports [9].

The most common acute injuries in volleyball are located in the ankle and fingers [9]. Other locations of acute injury include lower back, knee, and shoulder [1]. Serious acute shoulder injuries are rare, they are usually more of an overuse issue [11]. Injuries to the lower extremity represent over half of all game and practice injuries [8].

Ankle ligament sprains are the most common injury in volleyball [1, 8, 9]. The most common cause of ankle injury is from contact with another player, usually from landing on an opponent or teammate's foot [3, 6, 7, 9, 10, 14]. The forced supination that occurs when a player's foot lands on another player's foot under the net results in an inversion injury [2, 15]. The injury rate of ankle sprains has slightly decreased among US collegiate female players from 1988 to 2004, 1.8% in games and 3.0% in practices [8]. The authors of this study did not postulate any reasoning for this decrease but did comment that a center line rule change was implemented in 1998 without much change seen in ankle injuries.

Ankle injuries rarely keep players from the sport for a long period of time. In fact, often players will play through minor injuries [10]. However, due to the volume of ankle injuries, they appear to result in the longest absence from participation [1, 6, 10]. Previous ankle sprain is the highest risk factor for repeat ankle sprain [1, 3, 4]. There is a higher increase in injury rate in the subsequent 6–12 months after injury [14].

Data has shown that wearing ankle braces after ankle sprains can lower the risk of reinjury [1]. In one study, all collegiate female players at one institution wore bilateral double-padded ankle braces at all times, which reduced their ankle injury rate compared to the NCAA injury data over the same time period [16]. Bracing after ankle injury should be used up to 12 months post-injury [14]. Effective ankle injury prevention has been seen with balance training, technical training including changing style of jump, balance board training, and external ankle supports [1, 6, 9, 12, 14].

Finger sprains mainly occur due to contact with the ball [9]. The most common mechanism of injury is while fingers are spread while trying to block the ball. If fingers are lower than the ball, the downward trajectory of the ball puts them at risk for sprain [4, 10]. A less common mechanism for injury is the fingers hitting the net or the overhand dig, com-

monly used by back row players such a liberos [4, 9, 12]. Sprain of the first MCP joint is the most common specific injury of the fingers [4, 15]. Although finger sprains are frequent, they rarely cause loss of time from practice or matches [7]. Usually the player returns quickly while using a splint for bandage [10]. Buddy taping is recommended for collateral ligament injuries, while a thumb spica splint is recommended for the first MCP joint injuries [15].

Finger dislocations and fractures have also been seen [8, 15]. If finger dislocation occurs, it often is a dorsal displacement at the proximal interphalangeal (PIP) joint. Radiographs can provide confirmation of the displacement, which is generally easily reduced. If a displaced or unstable fracture is seen, referral to an orthopedic surgeon is recommended [17].

Other traumatic finger injuries have been reported. Mallet finger can occur if the ball hits the fingertip and forces the distal phalanx into flexion; rupture of the extensor tendon can also occur. The distal interphalangeal (DIP) joint will be flexed and the player will not be able to actively extend the joint. Treatment requires continuous splinting of the DIP joint in extension for 6–8 weeks [17]. Rarer hand injuries can include pisiform fracture [15].

Acute knee injuries are typically the most severe injury seen while covering a volleyball match [10, 15, 17]. Although rarely seen in volleyball, ACL injuries happen more commonly in women [1, 18]. In high-school-aged athletes, knee sprains (including grade III) were only a cause of medical disqualification in females [13]. In collegiate female athletes, injury occurred most frequently to the meniscus, followed by the collateral ligaments and then the ACL [8]. MCL and meniscal tears can be seen with ACL tear [18]. The most common mechanism of ACL injury is hyperextension or torsion of the knee joint during a noncontact event [18, 19]. These can occur during the jumping phase of an attack or while landing a jump on a single leg [8, 18, 20]. There was nearly an equal amount of external valgus motion and internal valgus motion seen as the cause of tear [18].

Preventative efforts should focus on ankle and finger injury prevention given how commonly they occur [9]. Given the severity of ACL injury, some advocate for prevention programs [17]. Several equipment and apparatus have been developed to lessen the change of injury that include ankle braces, knee pads, padded poles, and shock-absorbing playing surfaces like wood [4].

What Is Unique About Volleyball Injuries?

- Although volleyball is considered a noncontact sport, many ankle sprains occur as the result of contact with an opponent or teammate during net play.
- Blocking or overhead defense resulting in ball-to-finger contact can result in finger injuries.
- A player's position has been shown to affect risk of injury.
- Most injuries do not cause significant time away from sport.
- ACL injuries are rare but tend to occur more often in females.

What Do the Physicians Need to Know While Covering a Volleyball Match?

- Knowledge of mechanisms and positions of injury, usually involving the front row players during net play, is important to monitor the most common injuries. A physician covering volleyball matches should be prepared for acute ankle and finger injuries.
- Knowledge of braces, splints, and taping for ankle and finger injuries should be reviewed, and proper equipment should be available. This includes the following:
  - Buddy taping of fingers
  - Thumb spica taping
  - Ankle taping
- While less common, serious knee injuries like ACL tear do occur and so proper knowledge and skill of the knee examination is important.

General Rule About Return to Play

- Taping and bracing is allowed and commonly used to help injured players return to sport, especially with ankle and finger sprains.
- Reinjury of ankle sprains is common, and proper rehabilitation is encouraged for secondary prevention including balance and technical training as well as use of external bracing for up to a year.
- Any player with suspected intra-articular knee injury or instability, like an ACL rupture, should be held from play until further assessment.

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77 Weightlifting

Sheila E. Taylor and Mark E. Lavallee

Key Points

- Weightlifting encompasses different types of activities performed by men and women of all ages.
- Injury types and rates vary depending on the type of activity performed.
- Most acute injuries seen are muscular strains and ligamentous sprains.
- Emergent acute injuries do occur, but less commonly.
- The shoulder is the most commonly affected joint in the upper body, with the knee being the most commonly affected in the lower body.
- Weightlifting in general is safe in pediatric populations when carefully supervised.

Introduction

The importance of exercise to general health and wellness is widely known. One large category of exercise, strength training, has been shown to increase bone density, lean muscle mass, basal metabolic rate, and exercise tolerance [1, 2]. These effects result in an overall decreased osteoporosis risk, decreased insulin resistance, and improved cardiovascular health. As more and more people seek these benefits, many different types of strength training have emerged and are quickly gaining popularity. Over the last decade, there has been a 64.5% increase in persons participating in strength training [3]. As the number of persons participating in strength training increases, so does the number of persons at risk for injury. In order to care for patients who strength train, it is important to first have a general understanding of the different types of strength training. This knowledge will provide the foundation for understanding mechanisms of common injuries and, importantly, how to treat them. Furthermore, this knowledge will allow the sports medicine physician to effectively communicate with the athlete regarding their sport and formulate complex rehabilitation programs and return-to-play progressions after injury.

Types of Strength Training

According to the National Strength and Conditioning Association (NSCA), strength training, weight training, or resistance training "refers to a specialized method of conditioning which involves the progressive use of a wide range of resistive loads and a variety of training modalities designed to enhance health, fitness, and sports performance" [4]. In general, strength training encompasses a variety of activities and goals. There are many different ways to strength train with a variety of strength training equipment. Weightlifting machines, dumbbells, and barbells are the most frequently used strength training equipment. Body weight exercises and resistance bands can also be used to improve strength and sports performance. There are more specific and well-defined types of strength training that will be further explained below.

Powerlifting is a specific type of weightlifting. The goal of powerlifting centers on lifting the maximum weight for one repetition in three specific lifts: the squat, the deadlift, and the bench press (Fig. 77.1). Competitors have three attempts at each lift. The heaviest weight successfully completed for each lift is then added to achieve a final score.

S. E. Taylor (✉)
Christiana Care in Wilmington, University of Delaware, Delaware, USA
e-mail: staylor8@wellspan.org

M. E. Lavallee
York Sports Medicine Fellowship Program, York, PA, USA

Pennsylvania State University College of Medicine, Hershey, PA, USA

USA Weightlifting, Sports Medicine Society, Colorado Springs, CO, USA

International Weightlifting Federation, Budapest, Hungary

York Hospital, York, PA, USA
e-mail: mlavallee@wellspan.org

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Fig. 77.1 Powerlifting: (upper row) squat, (middle row) deadlift, and (lower row) bench press (Courtesy of Dr. Mark Lavallee)

Olympic weightlifting differs from powerlifting in the types of lifts. Olympic weightlifters attempt to perform the snatch and the clean and jerk (Fig. 77.2). The competitor has three attempts at lifting the maximal weight for one repetition to form a combined score.

Bodybuilding is another strength training sport, but it differs drastically from Olympic weightlifting and powerlifting in that the goal of this sport is not strength but rather muscular definition. In bodybuilding, competitors are judged on overall muscle size, muscle symmetry, and muscular development. Bodybuilding often focuses on heavy lifting for muscle growth followed by strict nutrition to attain low body fat levels necessary for extreme muscle definition.

Another type of strength training, CrossFit, has gained immense popularity in the fitness world over the recent years (Chap. 51). CrossFit is branded strength training sport

Fig. 77.2 Olympic weightlifting: (upper row) clean and jerk and (lower row) snatch (Courtesy of Dr. Mark Lavallee)

defined by varied functional movements performed at high intensity. More specifically, these functional movements are gathered from gymnastics, weightlifting, running, and rowing. Each workout of the day (WOD) has a defined set of functional movements with a specific measurable outcome or goal [5]. For example, the goal may be to perform as many repetitions as possible in a set time or to measure the time it takes to complete predetermined set of exercises. CrossFit also has competitions, the CrossFit games, in which competitors gather from all over the world to complete various WODs.

Other sports,, such as track and field, have strength training events like the hammer throw, javelin, shot put, and discus. The goal in each of these events differs slightly, but each requires physical strength to be competitive.

Acute Injuries

In strength training, like in other sporting events, acute injuries occur commonly. Acuity of an injury, by definition, relates to the sudden onset of the injury. This definition, however, gives little information relating to the severity of the injury. Acute injuries in strength training comprise 60–70% of all injuries observed [6]. Acute injuries can be further broken down into emergent and non-emergent injuries. For the covering physician, this distinction is especially important as it dictates further workup and management.

Acute emergent injuries can be further subdivided into categories: musculoskeletal (MSK), neurological, cardiovascular, and pulmonary. The most common acute emergent MSK injuries include fractures, dislocations, and tendon ruptures. In general, persons using free weights sustain a greater proportion of fractures and dislocations. Less common in occurrence are the acute emergent neurologic injuries such as disk herniation, cervical spine injuries, and stroke. Acute emergent cardiovascular events such as myocardial infarctions and arrhythmias and acute emergent pulmonary events such as spontaneous pneumothorax are relatively rare occurrences. In general, acute emergent injuries and events almost always require further workup, management, and treatment at a medical facility. Most acute emergent injuries and events result in significant time away from sport (greater than 5 days) [6].

In contrast to acute emergent injuries, acute non-emergent injuries are most common. Acute non-emergent injuries often do not prevent the athlete from competing or training. If rest is needed, these injuries usually result in only a short pause in training (less than 5 days) [6]. The most common acute non-emergent injuries are muscular strains and ligamentous sprains, accounting for 40–60% of all acute injuries in strength training [3, 7]. One study cites sprain or strain as the most common diagnosis of weight training injuries seen in US emergency departments (46.1%) [3]. The most common mechanism of injury was weights dropping on the person (65.5%) [3]. Furthermore, over 90% of acute injuries occur with free weights (dumbbells and barbells) [3]. Olympic weightlifters tend to injure elbows and knees, while powerlifters and CrossFit athletes more commonly suffer shoulder injuries.

Chronic Injuries

In all strength training sports, the repetitive action of lifting heavy loads poses a risk for chronic tendon issues, arthritis, and overuse injuries. Chronic injuries often result from tendons and muscles undergoing repetitive stress without sufficient recover time. Most chronic injuries result from overuse and account for approximately 30% of all injuries in strength training [6].

Tendinopathies are the most common chronic injuries in strength training and often significantly impact continued training and competition. The knee and shoulder are the most commonly affected joints. Other chronic injuries like arthritis are relatively common given the biomechanics of lifting. Repetitive axial loading like squatting and extension-based lifts like deadlifts put significant stress on the lumbar spine increasing risk for degenerative disk disease and lumbar facet arthropathy.

The repetitive motion and heavy loading of weightlifting can also cause more severe chronic injuries such as stress fractures. Stress fractures in weightlifting most commonly occur in the spine, again due to axial and extension-based loading (e.g., spondylolysis).

Most Common and Unique Injuries in Weightlifting by Region

After a fundamental understanding of types of strength training and injuries is attained, the sports medicine physician must also be familiar with the specific injuries to expect while treating the weightlifting athlete or covering a weightlifting competition. This next section will provide a brief explanation of the most common and unique weightlifting-specific injuries.

Upper Body

The most commonly injured joint in the upper body is the shoulder. Anterior shoulder dislocations can occur in Olympic weightlifting most commonly with the snatch and jerk. In powerlifting, it can occur with the deadlift. The repetitive and overhead nature of weightlifting predisposes to rotator cuff tendinopathy, impingement, and degenerative rotator cuff tears.

First rib fractures have been noted in weightlifting and powerlifting due to the repeated pulling of weights off the ground, located between the scalene muscles and the serratus anterior muscles which diametrically oppose each other and attach on the first rib. This can present as a stress fracture or a chronic occult fracture [8].

Closely associated with shoulder injuries, the clavicle can be affected by weightlifting as well. Distal clavicular osteolysis can be seen in powerlifting due to excessive loads, specifically with bench press. Similarly, the sternoclavicular joint (SCJ) is susceptible to injury in strength sports. In the bench press and front squats, loading forces cause compressive injuries. In the back squat, deadlift, clean, and javelin, distractive forces can cause capsular strain and subluxation. Lastly, direct trauma from front squat and "landing the clean" can cause acute hematoma or chronic inflammation/ thickened tissue over the SCJ. The most common acute emergent SCJ injury from these specific strength training activities is anterior dislocation [6].

Another upper body part at risk of injury with powerlifting is the pectoralis major. Pectoralis major muscle strain and rupture is most commonly seen in powerlifters that bench press. Powerlifters and bodybuilders are at the highest risk due to one-lift maximums and high rate of anabolic steroid and ergogenic aid use . Other sports that risk pectoralis major muscle rupture include boxing, jiu-jitsu, competitive waterskiing, and wind surfing [9].

The elbow is a common site for tendinitis and chronic tendinopathy. Both lateral and medial epicondylitis are commonly seen. Acute elbow dislocations do occur, mostly in Olympic weightlifting during the snatch or jerk. Acute triceps rupture has also been reported. Most case studies cite anabolic steroid use, creatine use, previous injury/surgery, or corticosteroid injection in recent past [6]. Another article cited triceps ruptures seen specifically in master weightlifters over the ages of 55 in the jerk component of the clean and jerk [6]. All cases denied recent steroid use, injection, or fluoroquinolone use.

Hand and finger injuries commonly result from accidents in those using free weights. A commonly cited description of injury is weights being dropped on persons or hands and fingers being pinched with weights. Minor soft tissue injuries like torn calluses are very common in deadlift, snatch, and clean and jerk. This poses risk of infection to lifter and others who will use the barbell later. For the injured lifter, the wound needs to be effectively cleaned and covered. For other lifters, the barbell needs to be effectively disinfected to prevent the spread of blood-borne pathogens.

In addition to hand and finger injuries, wrist injuries commonly occur in strength training. Wrist sprains and tendonitis occur as a result of the repetitive wrist extension needed in the hand positioning and grip of clean. In competitive bench press and clean and jerk, athletes may develop either a ganglion cyst (soft) or a bony exostosis (hard) at the distal radial ulnar joint due to repetitive overloading of the wrist in hyperextension. Stress fractures occur as well due to the repetitive motion, compression, and twisting. Distal radial physeal stress syndrome, avascular necrosis of the capitate, and stress

fracture of the scaphoid all have been described in weightlifting athletes. Intersection syndrome is another injury pattern seen. This is irritation and inflammation of the first and second dorsal compartments where they intersect 6–8 cm proximal to the radiocarpal joint. Again, the etiology of this syndrome is from the repetitive wrist flexion and extension [10]. There is also a rare case of radial stress fracture in a competitive bench presser described in the literature [11].

Lower Body

The knee is the most commonly affected lower body joint in strength sports [3]. In terms of chronic injuries, patellar tendinitis is more common than quadriceps tendinitis in powerlifting and Olympic weightlifting due to the deep loaded knee flexion required for the lifts. Additionally, the repetitive nature of bodybuilding increases the risk of patellar tendinitis. Ligament sprains around the knee are more commonly associated with inappropriate movement of a joint (misplacement of foot in bottom of snatch causing MCL tear). Conversely, tendon ruptures tend to be less associated with inappropriate movement across a joint and more from overloading the tensile strength of the tendon. Many of these cases often involve exposure to ergogenic aids (e.g., creatine), DHEA, anabolic steroids, or fluoroquinolones. Patellar dislocations and acute meniscal tears are rarely reported in strength training, likely due to the slow controlled nature of the lifts. Lastly, a rare but uniquely reported weightlifting injury of the knee is subchondral impaction fracture [12]. Weightlifters are often susceptible to overload contusion injuries to the cartilage and bone due to training with heavy loads. The etiology of subchondral impaction fracture may be an acute incident like a snatch or clean or the cumulative effect of repetitive microtraumas.

Like previously discussed, the lower back is commonly injured both acutely and chronically due to the heavy axial loading and repetitive flexion and extension injuries. Acute disk herniations in competition and training have been described as well as chronic degeneration and stress injuries.

Rehabilitation and Prevention

The topic of strength training injury rehabilitation is a broad topic outside the scope of this chapter and is generally specific to the type of injury sustained. In general, most acute injuries (sprains and strains) resolve with conservative measures (RICE, oral pain medications, gentle stretching, and home exercise program) without lasting symptoms, significant time off from training, or lasting dysfunction or limitations. Chronic injuries often require more prolonged time off with a formal course of injury-directed physical therapy.

Injury prevention is always an important topic in all sports and recreation. Specific to strength training, one of the most important factors in prevention is proper form, coaching, and supervision for novice lifters. Furthermore, having appropriate expectations for age, activity level, and goals prior to strength training can help to prevent some injuries as well. It has been documented that strength training is safe in youth as young as 8 as long as there is adult supervisor [4]. Further research is needed to aid in the development of target gender and age-specific evidence-based injury prevention strategies to decrease injury rates among strength training participants.

Youth/Pediatric Concerns

As a sports medicine physician, one of the primary tenets of patient care is education. It is important to appropriately counsel and guide patients on the risks and benefits of strength training. Specifically with the pediatric population, parents will want to know which strength training activities are safe for their children and adolescents. Strength training has been previously purported to be unsafe and ineffective in children [13]. Clinicians once considered open growth plates in a child as a contraindication to strength training because of potential, perceived risk of injury to the growth plates [14]. However, now it is widely accepted that strength training is generally safe for children and adolescents. Injury surveillance data from ER across the USA showed that the number one predictor of acute weightlifting injury was lack of appropriate adult supervision [3]. Overall, children have lower risk of strength-training-related sprains and strains than adults. That being said, like with any sports activity, there is always a risk of accident or injury. One study cites that the majority of youth strength training injuries are the result of accidents (dropping weights on their fingers and toes, misuse of equipment, and tripping and falling) rather than actual MSK etiologies [14]. Similarly, in another study, children aged 12 and younger were more likely to have hand and foot injuries than persons older than 13 [3]. It is proposed that with less knowledge of how to maintain control of the weights while lifting, the extremities are more susceptible to injury. Additionally, children and adolescents are more apt to engage in dangerous and risky behaviors, putting them at increased risk of injury. Accidents and risky behaviors are potentially preventable with increased supervision and stricter safety guidelines.

Children and adolescents should have carefully devised and supervised strength training programs from a multifaceted perspective of their parents, coaches, and physicians. The NSCA 2009 position statement paper reports that strength training is relatively safe in youth when properly designed and supervised [4]. Strength training can enhance

muscular strength and power, improve cardiovascular risk, improve motor skill, improve sports performance, prevent injuries, improve psychosocial well-being, and promote and develop healthy exercise habits during childhood and adolescence [4].

What Is Unique About Weightlifting Injuries?

- Weightlifting injuries are unique in that there are a wide range of different types of injuries and mechanisms depending on the type of weightlifting.
- Injuries can occur from the lifting activity or by unsafe use of the weight equipment itself (e.g., dumbbells, barbells, and kettlebells).

What Do the Physicians Need to Know While Covering a Weightlifting Event?

- Physicians need to be familiar with the event location, participating support staff, emergency equipment (location of spine board, AED, splinting supplies), and emergency action plans for the event.
- Physicians should position themselves in front of and close to the platforms in order to respond quickly in the event of a dislocation or other catastrophic injury.
- Additional staff may be positioned in the warm-up area.
- Watching the lifts is important in order to identify potential acute injuries, as some athletes may wish to continue competing even in the setting of unsafe injury.

General Rule About Return to Sport or Continue the Competition

Weightlifting competitions often involve multiple attempts at different types of lifts (e.g., three snatches or three deadlifts). Prompt evaluation and diagnosis of injury is important. Taping and certain bracing is allowed if needed. Acute muscle ruptures and dislocations are absolute contraindications to continue competition, even if the athlete wishes to continue. In adult athletes without catastrophic injuries, shared return-to-sport decision-making between the patient and physician often results in the best decision.

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Wrestling

78

Robert Kiningham and Babak Shadgan

Key Points

- High school wrestling injury rate is about 2.5 per 1000 athletic exposures (AE).
- US college wrestling injury rate was 7.25 injuries per AE during the 2005–2006 season.
- Incidence of injuries has been reported as 6–24% during the recent Olympic games.
- Skin inspection of wrestlers occurs prior to matches. Wrestlers with actively contagious lesions are disqualified.

Introduction

Wrestling is a one-on-one combat sport with the goal of pinning the opponent's back to the mat. Points are awarded for gaining control of the opponent and getting their back closer to a pinning position. There are two styles of international wrestling: Greco-Roman and freestyle. The main difference in the two styles is that wrestlers are not allowed to attack an opponent's legs in Greco-Roman wrestling (Fig. 78.1). Women's wrestling is increasing in popularity with collegiate, national, international, and Olympic competitions. The rules for women's wrestling are similar to freestyle. A variant of freestyle wrestling, called folkstyle, is the most common wrestling style in the USA and is the style of choice in US high schools, colleges, and most youth wrestling groups. Folkstyle places more emphasis on controlling the opponent than freestyle or Greco-Roman, and wrestlers spend more time off their feet and on the mat. In freestyle or Greco-Roman, the wrestlers spend most of the match on their feet facing each other in a "neutral" position. Because of differences in scoring and position, there are more "throws" in freestyle and Greco-Roman wrestling than in folkstyle, which leads to more dynamic injuries due to one wrestler forcefully throwing the other wrestler to the mat.

R. Kiningham (✉)
Department of Family Medicine, University of Michigan, Ann Arbor, MI, USA
e-mail: rkiningh@umich.edu

B. Shadgan
Department of Orthopaedics, University of British Columbia, Vancouver, BC, Canada

Medical, Prevention and Anti-Doping Commission, United World Wrestling (UWW), Corsier-sur-Vevey, Switzerland
e-mail: shadgan@mail.ubc.ca

Injury Epidemiology

Musculoskeletal

- High school wrestling injury rates during the 2005–2006 season ranged from 2.33 to 2.50 per 1000 athletic exposures (AE) [1, 2] which ranked second among high school boys sports and was roughly equivalent to boys soccer but considerably less than football.
- US college wrestling injury rate during the 2005–2006 season, per the NCAA Injury Surveillance Program, was 7.25 injuries per AE [2].
- Olympic game injury incidence per wrestler: Athens (2004) 24.2%, Beijing (2008) 9.3%, London (2012) 12%, and Rio (2016) 6.2% [3, 4].
- Injury rate during competition is higher than in practice at all levels [1, 2, 5, 6]. In the NCAA from the 1988–1989 through the 2003–2004 seasons, preseason practices had an injury rate of 8.3 per 1000 AE, compared to in-season practices of 4.7 per 1000 AE [5].
- Injuries most often occur from the standing neutral position and account for the highest percentage of injuries: 39% in high school, 42% in college, and 81% in Olympic wrestling [2].
- Six percent of high school injuries result from illegal moves [2].
- The most common injury sites differ by level of wrestling:
  - Among younger wrestlers, hand, wrist, and finger injuries appear to be the most common [7].

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Fig. 78.1 An Olympic Greco-Roman wrestling match. (Courtesy of Dr. Babak Shadgan)

  - The shoulder is the most common site injured in high school wrestlers, with reports ranging from 18% to 24% of injuries [2, 6].
  - In US college wrestling, the knee is the most common injury site, with 25% of injuries involving the knee, compared to 18% to the shoulder and 17% to the head and face [2].
  - Among Olympic wrestlers, head and face injuries are the most common. Beijing reported 69% of head and face injuries in 2008 and Rio's Olympic games reported 73% in 2016 [3, 4].
- Most wrestling injuries are relatively minor. Only 26% of injuries in high school, 27% in college, and 18% in Olympic wrestling resulted in missing more than 3 weeks of wrestling [7].
- Shoulder dislocation/subluxation is the most common injury requiring more than 3 weeks' recovery among high school wrestlers. Fractures of the hand, shoulder, and elbow are the second most common cause of prolonged recovery [2].
- Knee injuries are more common in college wrestlers, not only in frequency but also in severity. Knee injuries are the most common injury responsible for missing 3 or more weeks of college wrestling and the most common site of injury requiring surgery [2].

Head and Facial Trauma

Head and facial trauma accounted for 9% of injuries in high school wrestling and 17% of injuries in college wrestling during the 2005–2006 season [2]. Head and facial trauma was the most common injury site in the Rio Olympics [4]. Concussion, facial and brow lacerations, auricular hematomas, and epistaxis are included in this group of injuries. The concussion rate for high school wrestling (0.17 per 1000 AE) ranks third behind football and lacrosse and is equal to men's soccer over 11 seasons [8]. The NCAA Injury Surveillance Program for the 2009–2010 to 2013–2014 seasons reported that wrestling had the highest concussion rate of any men's sport at 11 per 10,000 AE. Player contact accounted for 63% of the concussions, while head-to-surface accounted for 22%. The most common injury mechanism-activity was player contact during a takedown attempt (31%) [9]. However, concussions in international and Olympic wrestling are uncommon. There were no concussions reported during the 2016 Rio Olympic games [4].

Acute auricular hematomas from blunt trauma are seen frequently in wrestlers, although the specific incidence has not been reported in high school and college. The incidence of auricular hematoma was 6% in Beijing and 9% in the Rio Olympics. Persistent auricular hematoma results in cartilage death and formation of new abnormal cartilage with fibrosis. This process leads to the development of the characteristic deformity known as a cauliflower ear [10]. Acute hematomas can be treated with aspiration and a pressure dressing. The within-season recurrence rate is very high, and oftentimes a more definitive procedure to prevent recurrence is necessary. A technique by which absorbable mattress sutures are used to close the space between the skin and the cartilage created by the hematoma has proven to be effective in preventing recurrence while allowing the athlete to continue to wrestle [11].

Athletic trainers and physicians who attend wrestling events know that acute epistaxis occurs very frequently among wrestlers. Nasal trauma is the most common mechanism of injury, and bleeding is usually unilateral and anterior.

The dry winter air and artificial heating during wrestling season, as well as the dehydrated state of many wrestlers, also may contribute to the frequency of epistaxis.

Tournament/Match Preparation

The on-site event physician makes the final determination of a wrestler's eligibility for a match. Every wrestler undergoes a skin check prior to wrestling. According to current wrestling rules, 1 h before the weigh-in [12], each competitor must undergo a skin examination carried out by physicians or other health-care professionals appointed by the event organizer. The examiner physician must inspect skin at exposed areas to make sure there is no infectious skin lesion and know the specific criteria for disqualification. More information about international standards and the procedure of the pre-weigh-in medical examination is available at United World Wrestling (UWW) Medical Regulations [13]. The National Federation of State High School Associations (NFHS) Medical Release Form and the NCAA Skin Evaluation and Participation Status Form are publicly available [14, 15]. The skin is also examined for vulnerable lesions or wounds that could potentially bleed or be damaged further during wrestling. Such lesions need to be adequately protected or the wrestler should not be allowed to compete. Nails are inspected to ensure they are cut short and smooth. Wrestlers who pass the examination would be allowed to proceed to weigh-in.

Catastrophic injuries are rare in wrestling, but they can occur, so an emergency medical action plan needs to be established prior to the competition. This plan includes the location of an automatic external defibrillator (AED) and a protocol to retrieve it, C-spine precautions and backboard location and procedures, and a communication system to summon Emergency Medical Services (EMS). A procedure for handling dental injuries and avulsed teeth should also be established.

Tournaments usually include several matches occurring simultaneously on separate mats. The lead physician and head athletic trainer need to formulate a plan to ensure that each mat has an observer familiar with wrestling injuries and rules. The lead physician should be free to oversee the entire event, institute emergency care, and make decisions regarding medical disqualification.

The physician and athletic trainer must be prepared to quickly control minor facial trauma, including epistaxis. Physicians and athletic trainers should have rapid access to nasal cotton plugs, nostril spreader, gauze, athletic tape, and Monsel's solution applicators. Physicians should also be equipped to suture or staple lacerations, particularly during a tournament with multiple matches in a day. Ideally, there should be a designated private "quiet area" to assess injuries after a match and provide wound care, including suturing.

What Is Unique About Wrestling Injuries?

Wrestling is a combat sport between two competitors, so head and facial trauma is common. Physicians need to be skilled with the treatment of minor head and facial traumatic injuries. The most common serious injuries involve joint dislocations (shoulder, elbow, and finger). On-site management of joint dislocations, including evaluation, reduction (if appropriate), stabilization, and transport (if necessary), needs to be reviewed with all members of the medical team prior to a competition. The evaluation of injuries occurs in high-pressure situations and must be done within a time limit (see below). Calming the athlete, while evaluating the severity of the injury in an efficient and timely manner, is part of the "art" of sports medicine that can only be learned by repeated exposure to injured athletes in competitive situations.

What Do Physicians Need to Know While Covering a Wrestling Match?

Physicians who cover wrestling matches need to know the rules regarding pre-match medical disqualification, injury time, concussion time, and blood time during matches. The different levels of wrestling (youth, high school, college, Olympic/international) all have different rules, so it is critical that the physician knows the rules for the event he/she is covering. NCAA and NFHS have communicable disease forms available online to help guide the physician in determining eligibility. International wrestling is now governed by United World Wrestling (UWW), which has up-to-date medical rules on their website [12]. Table 78.1 summarizes the current rules regarding injury, blood, and concussion time during a match.

Wrestling rules change annually, so it is important that the physician stay current and check for updates. It is prudent to

Table 78.1 Rules regarding injury, blood, and concussion time during a match [13, 16, 17]

	NFHS	NCAA	UWW
Injury time	2 min total Third time-out results in medical DQ	90 sec total Third time-out results in medical DQ	Unlimited for assessment. Match terminated at physician's discretion
Blood time	5 min total Multiple match stoppages allowed	Discretion of referee Multiple match stoppages allowed	4 min total for each wrestler. Multiple match stoppages allowed
Concussion assessment time	Concussion time is part of injury time	Unlimited for assessment	Unlimited for assessment

DQ disqualification

check with the lead official prior to match or tournament to make sure that there is agreement about injury time regulations for the competition.

General Rule About Return to Play

Regulations for minimal time to return to play after a skin infection are included in the NCAA and NFHS Communicable Disease Forms. Concussion management of wrestlers does not differ from concussion management for other athletes. Return to play from musculoskeletal injuries requires an assessment of the ability of the wrestler to protect himself and minimize the risk of reinjury. Wearing any rigid joint support requires precompetition assessment and a written permission issued by the medical officer of the competition. All pads and braces will be inspected by the lead official to ensure that they do not present a risk of injury to the opposing wrestler.

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Index

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